ENVIRONMENTAL RISKS IN A DIFFUSIVE SIS MODEL INCORPORATING USE EFFICIENCY OF THE MEDICAL RESOURCE

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Dedicated to Professor Lishang Jiang on the occasion of his 80th birthday

Abstract. To capture the impact of spatial heterogeneity of environment and available resource of the public health system on the persistence and extinction of the infectious disease, a simplified spatial SIS reaction-diffusion model with allocation and use efficiency of the medical resource is proposed. A nonlinear space dependent recovery rate is introduced to model impact of available public health resource on the transmission dynamics of the disease. The basic reproduction numbers associated with the diseases in the spatial setting are defined, and then the low, moderate and high risks of the environment are classified. Our results show that the complicated dynamical behaviors of the system are induced by the variation of the use efficiency of medical resources, which suggests that maintaining appropriate number of public health resources and well management are important to control and prevent the temporal-spatial spreading of the infectious disease. The numerical simulations are presented to illustrate the impact of the use efficiency of medical resources on the control of the spreading of infectious disease.

1. Introduction. Since the most classical and theoretical model, SIR model, was formulated by Kermack and McKendrick in 1927, there are increasing epidemiological models which have led to both quantitative and qualitative predictions in epidemic dynamics [6, 21]. It indicates that compartmental models have been

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playing a key role in the development of mathematical modeling and analysis of infectious diseases. In compartmental models for infectious diseases, the involving population are classified into subgroups associated with different compartments representing different stages of infection. The transmission of the infection is modeled by ordinary differential systems. Except for the demographics of the involving population, the incidence mechanisms and interference strategy including for example quarantine and vaccination of susceptibles will all affect the course of infection of the disease. In general, both the demographical and non-demographical parameters, such as contact rate, transmission probability and the recovery rate are often treated as the constants [2, 4, 23]. For some of the compartmental models, the transmission dynamics can be characterized by the so-called basic reproduction number $R_0$ [2, 6, 21]. In an homogenous environment, the basic reproduction number is usually a constant describing the infection power of the infectious disease in a susceptible population. In general, the infectious disease will be eradicated if $R_0 < 1$, otherwise the infectious disease will become endemic.

For an infectious disease, what the public health agencies and administration care most is also the spatial transmission, i.e., if the endemic can spread to other areas to cause more infection. For the purpose of control and prevention, it is important to also understand the spatial feature and mechanisms of the infection.

There have been many theoretical research on the reaction-diffusion models, such as diffusive virus dynamics model [13], diffusive SEI model [11], diffusive SEI-SI avian-human influenza model [20].

In recent years, spatial diffusion and environmental heterogeneity have been recognized as important factors to affect the heterogeneity of the transmission, spatial variations in the persistence and eradication of infectious diseases such as tuberculosis, measles and flu, especially for vector-borne diseases, such as malaria, dengue fever, lyme diseases and West Nile virus etc.

Just as the case in the compartmental models of ordinary differential systems, the basic reproduction itself is not sufficient to characterize the transmission dynamics [22], for the reaction-diffusion type of spatial transmission model, the common basic reproduction number is not capable of describing the spatial transmission dynamics, especially to reflect the spatial features of the spread in the region considered.

Among the early work using reaction-diffusion model to study the dispersion of the infection, Allen et al [1] proposed an SIS epidemic reaction-diffusion model in a spatially heterogeneous environment with the following governing equations:

\[
\begin{align*}
S_t - d_S \Delta S &= -\frac{\beta(x)SI}{S+I} + \gamma(x)I, & x \in \Omega, \ t > 0, \\
I_t - d_I \Delta I &= \frac{\beta(x)SI}{S+I} - \gamma(x)I, & x \in \Omega, \ t > 0.
\end{align*}
\] (1.1)

For such a spatial transmission model, the basic reproduction number $R_0$ was defined which depends on the epidemic risk related to incidence parameters and population movement. It was proved that for the high-risk domain with $R_0 > 1$, the disease-free equilibrium is always unstable, while for the low-risk domain with $R_0 < 1$, the disease-free equilibrium is stable if and only if the infected individuals have mobility (dispersion rate) above a threshold value.

Subsequently, Peng et al [14, 16] presented a series of works to further understand the impacts of large and small diffusion rates of the susceptible and infected population on the persistence and extinction of the disease in heterogeneous or periodic environment, which showed that different strategies of controlling the diffusion
rates of individuals may lead to very different spatial distributions of the population. Specially, different from the case in [1, 14, 16], which was assumed that the habitat of the populations consists of only the low and high risk areas, Peng [15] concerned a more complicated environment where the low, moderate and high risk areas coexist. The results indicated how population movement and epidemic risk affect the spatial distribution of infectious diseases.

To focus on the new phenomena induced by spatial heterogeneity of environment and advection, Ge et al [7] assumed that the population $N(x, t)$ is constant in space for all time, that is, $N(x, t) \equiv N^*$ for $x \in \Omega$ and $t \geq 0$, then considered the following free boundary problem

$$
\begin{aligned}
& I_t - d_I I_{xx} + \alpha I_x = (\beta(x) - \gamma(x))I - \frac{\beta(x)}{N^*} I^2, \quad g(t) < x < h(t), \ t > 0, \\
& I(g(t), t) = 0, \ h'(t) = -\mu I_x(h(t), t), \quad t > 0, \\
& I(h(t), t) = 0, \ h'(t) = -\mu I_x(h(t), t), \quad t > 0, \\
& g(0) = -h_0, \ h(0) = h_0, \ I(x, 0) = I_0(x), \quad -h_0 \leq x \leq h_0,
\end{aligned}
$$

where $x = g(t)$ and $x = h(t)$ are the unknown left and right boundaries of the infected interval which change with the time $t$. The basic reproduction number $R_F^0(t)$ depending on the epidemic risk in the expanding interval $(h(t), g(t))$ is proposed, and spreading and vanishing of the disease are studied. They proved that spreading happens if $R_F^0(t_0) \geq 1$ for some $t_0 \geq 0$, which means that if the spreading domain is high-risk at some time, the disease will continue to spread till the whole area is infected; while if $R_F^0(0) < 1$, the disease may be eradicated or keep spreading depends on the diffusion rate, the expanding rate and the initial number of the infective individuals. It was also shown that fast diffusion, small expanding rate and small initial infected size are beneficial for controlling and preventing of the infectious disease.

Epidemiologically, the nonlinear recovery rate and the nonlinear incidence rate play a key role in generating the abundant dynamical behaviors of epidemic models. In the earlier compartmental models, the per capita recovery rate of the infected class is usually assumed to be a constant, but in practice, the recovery rate depends on the resources of the health systems, especially the capacity of the hospital settings and the effectiveness and efficiency of the treatment.

Recently, Shan and Zhu [18] introduced the number of hospital beds in an SIR type of compartmental model to study its impact on the dynamics of transmission of infectious diseases. Their analysis and results indicated that the public health resource described by an nonlinear recovery rate can lead to complicated dynamics such as the existence of backward bifurcation, Hopf bifurcations and Bogdanov-Takens bifurcations which can generate multiple limit cycles. The authors even discovered the nilpotent singularities in the model when the recovery rate is modeled by a generalized nonlinear function of the number of infected individuals [19].

In practice, when an infectious disease starts to emerge and spread, usually the contact transmission incidence also depends on the location, and the infected individuals seek treatment in their favorite hospitals or cities with better medical conditions, in particular with less waiting time at a lower cost. The spatial features of the transmission and treatment are complicated in reality due to the complexity of human activities and available medical resources as well as the effective management of the resources.

In this paper, we will consider a simpler or an ideal case that the contact transmission incidence is heterogeneous in the area, but the public health resource are
not evenly distributed, that is, the exit rate or the recovery rate for a given spot of the public health (hospital settings) does not depend on the number of infected individuals seeking for treatment, and at a given position, the recovery rate decreases only due to the crowded higher number of infected individuals waiting for treatment. Therefore, in order to model the impact of available hospital resources on the recovery rate, as in [18, 19], we assume that

\[ \gamma(b, I, x) = \gamma_m(x) + (\gamma_M(x) - \gamma_m(x)) \frac{b(x)}{b(x) + I}, \]  

where \( \gamma_m(x) \) and \( \gamma_M(x) \) represent the minimal per capita recovery rate and the maximal per capita recovery rate at location \( x \), respectively, and \( b(x) \) denotes the available medical resources at location \( x \).

Note that at location \( x \), the medium recovery rate \( \frac{1}{2}(\gamma_m(x) + \gamma_M(x)) \) can be achieved when \( I(x, t) = b(x) \), so the function \( b(x) \) is a measure of available hospital resources at \( x \) in terms of hospital beds per 10000 of populations (HBPR) [18], as well as the medium recovery rate restricted by the medical resource allocation (the number of hospital beds, the number and the ability of medical workers, etc) at \( x \).

Therefore, it is essential to assume that \( b(x) \) to be a function of \( \gamma_m(x) \) and \( \gamma_M(x) \). Since the allocation of resources to prepare the number of hospital beds (settings) is usually decided by the due course of infectious diseases and the decision from the administrative management, and sometimes can be affected by many other factors. In this paper, we will only consider the case when \( b \) remains a constant, the case for example the number of hospital settings suggested from the higher public health policy makers.

To understand the impact of the number of hospital beds on controlling the transmission of infectious disease, we consider the following diffusive problem

\[ \begin{aligned} I_t - d_I \Delta I &= (\beta(x) - \gamma(b, I, x))I - \frac{\beta(x)}{K} I^2, \quad x \in \Omega, \quad t > 0, \\
\partial I / \partial \eta &= 0, \quad x \in \partial \Omega, \quad t > 0, \\
I(x, 0) &= I_0(x) \geq 0, \quad I_0(x) \neq 0, \quad x \in \Omega, \\
\end{aligned} \]  

(1.4)

where \( d_I \) denotes the diffusion coefficient and \( \gamma(b, I, x) \) is defined in (1.3) with constant \( b \), \( \beta(x) \), \( \gamma_m(x) \) and \( \gamma_M(x) \) are positive bounded Hölder continuous functions, which account for spatial dependent rates of disease contact transmission, the minimal recovery rate and the maximal recovery rate, respectively. The homogeneous Neumann boundary condition \( \partial I / \partial \eta = 0 \) means that the infected individuals live in the self-contained environment and no infected individuals move across the boundary \( \partial \Omega \). The initial infected individuals \( I_0(x) \) is continuous over \( \Omega \).

Different from the common compartmental models and reaction-diffusion models with linear recovery rate, the diffusive model (1.4) involves nonlinear recovery rate, therefore the basic reproduction number for (1.4) will be dependent on the minimal per capita recovery rate and the maximal per capita recovery rate. For these reasons, we will redefine the basic reproduction numbers, and use the basic reproduction numbers to characterize the complex dynamics of the temporal and spatial transmission of the disease.

Compared with the model (1.1) considering space-dependent recovery rate \( \gamma(x) \), our model (1.4) involves nonlinear recovery rate \( \gamma(b, I, x) \), which depends on the local medical resource allocation, the use efficiency and the infected cases. Although the two recovery rates all depend on the spacial heterogeneity, the nonlinear recovery rate \( \gamma(b, I, x) \) matches the reality better. In present paper, we will investigate the
influence of the nonlinear recovery rate induced by medical resource allocation and utilization, which is very significant for infectious disease prevention and control. We should point out that the allocation and management of medical resources is a much more complicated issue for public health. Due to the technical difficulties, in this paper we only consider a simplified case by assuming that the hospital resources in terms of HBPR remains a constant.

The rest of this paper is arranged as follows. The next section is devoted to developing the basic reproduction numbers and their properties. The stabilities of the reaction-diffusion system are given in section 3. Numerical simulations are also presented in section 4 to illustrate the impacts of the number of hospital beds, and a brief discussion is also given.

2. Basic reproduction numbers. In this section, as in [1], we define the basic reproduction numbers and investigate their properties and implications for the reaction-diffusion system (1.4). Usually, the basic reproduction number $R_0$ plays a key role in describing infectious diseases transmission and in the investigations of the dynamical behaviors of epidemic model. From the biological point of view, the basic reproduction number has a clear epidemiological interpretation, that is, $R_0$ is the expected number of secondary infections due to a single individual over the course of its infection. Therefore $R_0$ remains a useful measure of the likelihood and severity of disease outbreaks. If the epidemic model is ODE or PDE system with constant coefficients, we know that the basic reproduction number $R_0$ can be obtained by the next generation matrix method. However, when the transmission contact rate and exit rate are spatial heterogeneity, as in [1], they introduced the new basic reproduction number by variational method. In model (1.4), we consider the nonlinear recovery rate that relate to the maximal and minimal recovery rates.

Let us introduce the basic reproduction numbers $R_0^*$ and $R_{0*}$ by

$$R_0^* = R_0(\Omega, \beta(x), \gamma_m(x), d_I) = \sup_{\phi \in H^1(\Omega), \phi \neq 0} \frac{\int_\Omega \beta(x) \phi^2 dx}{\int_\Omega (d_I \|\nabla \phi\|^2 + \gamma_m(x) \phi^2) dx},$$

$$R_{0*} = R_0(\Omega, \beta(x), \gamma_M(x), d_I) = \sup_{\phi \in H^1(\Omega), \phi \neq 0} \frac{\int_\Omega \beta(x) \phi^2 dx}{\int_\Omega (d_I \|\nabla \phi\|^2 + \gamma_M(x) \phi^2) dx}.$$

Specially, if the contact transmission incidence and the recovery rate are homogeneous, that is, $\beta(x) \equiv \beta^*$ and $\gamma_m(x) \equiv \gamma^*$ (or $\gamma_M(x) \equiv \gamma^*$), (where $\beta^*$ and $\gamma^*$ are constants, ) then $R_0^* = \frac{\beta^*}{\gamma^*}$ (or $R_{0*} = \frac{\beta^*}{\gamma^*}$), which is consistent with the usual basic reproduction number $R_0$ for the corresponding non-spatial model.

The following results were given in [9] (Lemma 2.3):

**Lemma 2.1.** $1 - R_0^*$ has the same sign as $\lambda_1^*$, where $\lambda_1^*$ is the principal eigenvalue of the reaction-diffusion problem

\[
\begin{align*}
-d_I \Delta \psi &= \beta(x) \psi - \gamma_m(x) \psi + \lambda_1^* \psi, & x \in \Omega, \\
\frac{\partial \psi}{\partial \eta} &= 0, & x \in \partial \Omega.
\end{align*}
\]  

(2.1)

**Lemma 2.2.** $1 - R_{0*}$ has the same sign as $\lambda_{1*}$, where $\lambda_{1*}$ is the principal eigenvalue of the reaction-diffusion problem

\[
\begin{align*}
-d_I \Delta \psi &= \beta(x) \psi - \gamma_M(x) \psi + \lambda_{1*} \psi, & x \in \Omega, \\
\frac{\partial \psi}{\partial \eta} &= 0, & x \in \partial \Omega.
\end{align*}
\]  

(2.2)

With the above defined reproduction numbers, we have

\[
\begin{align*}
\frac{\partial \psi}{\partial \eta} &= 0, & x \in \partial \Omega.
\end{align*}
\]
Theorem 2.3 ([1]). The following assertions hold.

(a) \( R^*_0 \) and \( R_0^* \) are positive and monotone decreasing functions with respect to \( d_I \);

(b) \( R^*_0 \to \max_{x \in \Omega} \frac{\beta(x)}{\gamma_m(x)} \) and \( R_0^* \to \max_{x \in \Omega} \frac{\beta(x)}{\gamma_M(x)} \) as \( d_I \to 0 \);

(c) \( R^*_0 \to \int_\Omega \frac{\beta dx}{\gamma_m dx} \) and \( R_0^* \to \int_\Omega \frac{\beta dx}{\gamma_M dx} \) as \( d_I \to \infty \);

(d) 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 \( d_I > d_I^* \). If \( \int_\Omega \beta(x) dx > \int_\Omega \gamma_m(x) dx \), then \( d_I^* = \infty \), that is, \( R^*_0 > 1 \) for all \( d_I > 0 \); if \( \beta(x) < \gamma_m(x) \) in \( \Omega \), then \( d_I^* = 0 \), that is, \( R_0^* < 1 \) for all \( d_I > 0 \); the similar result holds for \( R_0^* \);

(e) If \( \beta(x) \equiv \beta^* \) and \( \gamma_m(x) \equiv \gamma^* \) (or \( \gamma_M(x) \equiv \gamma^* \)), then \( R_0^* = \frac{\beta^*}{\gamma^*} \) (or \( R_0^* = \frac{\beta^*}{\gamma^*} \)).

Similarly as stated in [1], we say the location \( x \) is low-risk if \( \beta(x) \leq \gamma_m(x) \) and it is high-risk if \( \beta(x) > \gamma_M(x) \). If \( \gamma_m(x) < \beta(x) \leq \gamma_M(x) \), we then say that the location \( x \) is moderate-risk; accordingly, the environment (or the region \( \Omega \)) is low-risk (high-risk, moderate-risk) if \( \int_\Omega \beta(x) dx \leq \int_\Omega \gamma_m(x) dx \) \( (\int_\Omega \beta(x) dx > \int_\Omega \gamma_m(x) dx , \int_\Omega \gamma_m(x) dx < \int_\Omega \beta(x) dx \leq \int_\Omega \gamma_M(x) dx) \).

Theorem 2.3 implies that the basic reproduction numbers depend on the maximal local risk if the diffusion rate is small, while if the diffusion rate is big, they depend on the risk of the whole region.

3. Stability. In this section, we will establish the dynamical behaviors in term of the thresholds \( R^*_0 \) and \( R_0^* \). First, by the maximum principle, \( I(x, t) \) is positive for \( x \in \Omega \) and \( t \in (0, T_{\text{max}}) \), where \( T_{\text{max}} \) is the maximal existence time for solutions to (1.4). Again by the maximum principle, \( I(x, t) \) is bounded on \( \Omega \times (0, T_{\text{max}}) \). Hence, it follows from the standard theory for parabolic systems [12] that \( T_{\text{max}} = \infty \) and that a unique classical solution \( I \) to problem (1.4) exists for all time.

We start with the existence, uniqueness of the equilibria to problem (1.4). We are only interested in nonnegative steady-state solutions to (1.4), i.e., nonnegative solutions to the elliptic problem

\[
\begin{aligned}
-d_I \Delta I_s &= (\beta(x) - \gamma(b, I_s, x)) I_s - \frac{\beta(x)}{N^*} I_s^2, & x \in \Omega, \\
\frac{\partial I_s}{\partial n} &= 0, & x \in \partial \Omega.
\end{aligned}
\] (3.1)

The problem (3.1) always admits a trivial solution, that is, \( I_s^E \equiv 0 \) in \( \Omega \), it is called the disease-free equilibrium (DFE) as in [1].

When \( R_0^* = \max_{x \in \Omega} \frac{\beta(x)}{\gamma_M(x)} (\beta(x), \gamma_M(x), \gamma_m(x)) > 1 \), owing to Lemma 2.2, the eigenvalue problem

\[
\begin{aligned}
-d_I \Delta \psi &= \beta(x) \psi - \gamma_M(x) \psi + \lambda_1 \psi, & x \in \Omega, \\
\frac{\partial \psi}{\partial n} &= 0, & x \in \partial \Omega
\end{aligned}
\] (3.2)

admits a unique positive eigenfunction \( \psi_*(x) \) with \( ||\psi_*||_{L^\infty(\Omega)} = 1 \) and the principal eigenvalue \( \lambda_1 < 0 \).

It is not difficult to see that \( \bar{T}(x) = N^* \) is an upper solution to problem (3.1). Let \( \bar{I}(x) = \varepsilon \psi(x) \), where \( \varepsilon \) will be determined in the following. Direct computations yield that

\[
-d_I \Delta \bar{I} = (\beta(x) - \gamma_M(x)) \bar{I} + \lambda_1 \bar{I} \leq (\beta(x) - \gamma(b, \bar{I}, x)) \bar{I} - \frac{\beta(x)}{N^*} \bar{I}^2
\]
provided that $0 < \varepsilon \leq \min\{N^*, \frac{-\lambda_1 N^*}{\max_{x \in \Omega} \beta(x)}\}$. Therefore $I(x)$ is a lower solution, and $\overline{I}(x)$ and $\underline{I}(x)$ are the ordered upper and lower solutions of the elliptic problem (3.1), respectively. We then have at least one positive solution of the elliptic problem, we denote it by $I^*_s(x)$, it is called the endemic equilibrium (EE).

Furthermore, if $b > \max_{x \in \Omega} \frac{(\gamma_M(x) - \gamma_m(x))N^*}{\beta(x)}$, we can claim that the positive solution $I^*(x)$ of (3.1) is unique. In fact, we denote

$$f(I) = \frac{\beta(x) - \gamma_m(x) - (\gamma_M(x) - \gamma_m(x))}{b + I} \frac{b}{N^*},$$

direct calculation shows that

$$f'(I) = \frac{\beta(x) - \gamma_m(x) - (\gamma_M(x) - \gamma_m(x))}{(b + I)^2} \frac{b}{N^*} - \frac{\beta(x)I}{N^*} < 0.$$

By Proposition 3.3 in [5], the uniqueness of the solution can be guaranteed.

If $R_0^* \leq 1$, we claim that the nonnegative equilibrium is only DFE. In fact, if there is a nontrivial nonnegative equilibrium $I^*_s$, the solution must be positive everywhere by strong maximum principle and it satisfies

$$-d_1 \Delta I^*_s = (\beta(x) - \gamma(b, I^*_s, x))I^*_s - \frac{\beta(x)}{N^*} (I^*_s)^2 < (\beta(x) - \gamma_m(x))I^*_s,$$

multiplying the above inequality by $I^*_s$ and integrating it on $\Omega$ yield

$$\int_{\Omega} d_1 |\nabla I^*_s|^2 dx < \int_{\Omega} (\beta(x) - \gamma_m(x))I^*_s dx,$$

it together with the definition of $R_0^*$ gives

$$R_0^* = \sup_{\phi \in H^1(\Omega), \phi \neq 0} \left\{ \frac{\int_{\Omega} \beta(x)\phi^2 dx}{\int_{\Omega} (d_1|\nabla \phi|^2 + \gamma_m(x)\phi^2) dx} \right\} \geq \frac{\int_{\Omega} \beta(x)I^*_s dx(I^*_s)^2 dx}{\int_{\Omega} (d_1|\nabla I^*_s|^2 + \gamma_m(x)(I^*_s)^2) dx} > 1,$$

which leads a contradiction to the assumption $R_0^* \leq 1$.

To give a summary, we have the following results about the existence of steady-state solution to problem (1.4).

**Theorem 3.1.** When $R_0^* \leq 1$, problem (1.4) admits a unique disease-free equilibrium $I^*_0$. When $R_0^* > 1$, problem (1.4) has an endemic equilibrium $I^*_s$, furthermore, the endemic equilibrium is unique if $b > \max_{x \in \Omega} \frac{(\gamma_M(x) - \gamma_m(x))N^*}{\beta(x)}$.

Now we consider the local stability of the DFE. Linearizing (1.4) around the DFE, we arrive at $U_t - d_1 \Delta U = U(\beta(x) - \gamma_M(x))$, where $U(x,t) = I(x,t) - 0$. Suppose that $U = \phi(x)e^{\lambda t}$, then we have

$$\begin{cases} -d_1 \Delta \phi = (\beta(x) - \gamma_M(x))\phi - \lambda \phi, & x \in \Omega, \\ \frac{\partial \phi}{\partial n} = 0, & x \in \partial \Omega. \end{cases}$$

(3.3)

If $R_0^* \leq 1$, it follows from Lemma 2.1 that there exist positive eigenfunction $\psi(x)$ and $\lambda_1^* \geq 0$ such that

$$\begin{cases} -d_1 \Delta \psi = (\beta(x) - \gamma_m(x))\psi + \lambda_1^* \psi, & x \in \Omega, \\ \frac{\partial \psi}{\partial n} = 0, & x \in \partial \Omega. \end{cases}$$

and using again the multiply-multiply-subtract-integrate trick yields

$$\int_{\Omega} (\gamma_m(x) - \gamma_M(x))\phi(x)\psi(x) dx = (\lambda + \lambda_1^*) \int_{\Omega} \phi(x)\psi(x) dx,$$
which implies \( \lambda < -\lambda^*_1 \leq 0 \). Hence, the disease-free equilibrium is locally asymptotically stable.

Next, we consider the global stability of the DFE. We will show that the disease always becomes extinct if \( R_0^* \leq 1 \). In fact, since \( R_0^* \leq 1 \), it follows from Lemma 2.1 that the eigenvalue problem (2.1) admits a positive solution \( \psi(x) \) and \( \lambda^*_1 \geq 0 \). We now define

\[
\mathcal{I} = M(1 + t)^{-1}\psi(x),
\]

then it satisfies, for \( x \in \Omega \) and \( t > 0 \),

\[
\mathcal{I}_t - d_1 \Delta \mathcal{I} = (\beta(x) - \gamma_m(x) + \lambda^*_1)\mathcal{I} - M(1 + t)^{-2}\psi(x)
\geq (\beta(x) - \gamma(b, \mathcal{I}, x))\mathcal{I}_t - \frac{\beta(x)}{N^*}(\mathcal{I})^2
\]

if \( M \geq \max_{x \in \Omega} N^*/(\beta(x)\psi(x)) \). Furthermore, if \( M \) is chosen sufficiently large such that \( I(x,0) \leq \mathcal{I}(x,0) \) for \( x \in \Omega \), then \( \mathcal{I} \) is an upper solution of problem (1.4) and the comparison principle shows that \( I(x,t) \leq \mathcal{I}(x,t) \) for \( x \in \Omega \) and \( t > 0 \). Since \( \mathcal{I}(x,t) \to 0 \) as \( t \to \infty \) for \( x \in \overline{\Omega} \), we then have \( I(x,t) \to 0 \) as \( t \to \infty \) for \( x \in \overline{\Omega} \).

Finally, we consider the global asymptotic stability of the EE by the upper and lower solutions method. As Theorem 4.4 in Chapter 5 in [17], we investigate the asymptotic behavior of the time-dependent solution \( I(x,t) \) to (1.4) in relation to its corresponding steady-state solution \( I_s(x) \) satisfying (3.1).

In the case \( R_0^* > 1 \), it was shown before that the pair \( \tilde{I} = M \) and \( \hat{I} = \varepsilon \psi \) are the upper and lower solutions of (3.1) for any constants \( M \geq N^* \) and \( 0 < \varepsilon \leq \min\{N^*, \frac{-\lambda^*_1}{\max_{N^*} N^*/\beta(x)}\} \).

Every pair of upper and lower solutions of (3.1) is also a pair of upper and lower solutions of (1.4) whenever \( \tilde{I}(x) \leq I_0(x) \leq \hat{I}(x) \) in \( \overline{\Omega} \). Furthermore, for any \( I_0(x) \geq 0 \) and \( I_0(x) \neq 0 \), then \( I(x,t) > 0 \) in \( \overline{\Omega} \). In particular, we can choose \( t_1 > 0 \) and \( \varepsilon > 0 \) sufficiently small such that \( \tilde{I} = M \) and \( \hat{I} = \varepsilon \psi \) are also the upper and lower solutions of (3.1) in \( \overline{\Omega} \times [t_1, \infty) \).

Observe that the function on the right side of the equation in (1.4) is smooth for \( I \geq 0 \), we then have the Lipschitz condition

\[
|||\beta(x) - \gamma(b, I_1, x)|||_1 - \left|\frac{\beta(x)}{N^*}I^2_s - \frac{\beta(x)}{N^*}I^2_s\right| \leq K(||I_1 - I_2||)
\]

for \( \tilde{I} \leq I_1, I_2 \leq \hat{I} \), where \( K = K(N^*, \beta(x), \gamma_m(x), \gamma_M(x), \tilde{I}, \hat{I}) \). Now we consider the equivalent elliptic problem

\[
\begin{align*}
-d_1 \Delta I_s + K I_s &= K I_s + (\beta(x) - \gamma(b, I_s, x))I_s - \frac{\beta(x)}{N^*}I^2_s, & x \in \Omega, \\
\frac{\partial I_s}{\partial n} &= 0, & x \in \partial \Omega
\end{align*}
\]

with initial value \( \tilde{I}^{(0)} = \varepsilon \psi \), and \( \hat{I}^{(0)} = M \), respectively, where \( K \) is the above Lipschitz constant. By the iteration process

\[
\begin{align*}
-d_1 \Delta \tilde{I}^{(k)} + K \tilde{I}^{(k)} &= K \tilde{I}^{(k-1)} + \left[(\beta(x) - \gamma(b, \tilde{I}^{(k-1)}, x)) - \frac{\beta(x)}{N^*}I^{(k-1)}_s\right]I^{(k-1)}_s, \\
-d_1 \Delta \hat{I}^{(k)} + K \hat{I}^{(k)} &= K \hat{I}^{(k-1)} + \left[(\beta(x) - \gamma(b, \hat{I}^{(k-1)}, x)) - \frac{\beta(x)}{N^*}I^{(k-1)}_s\right]I^{(k-1)}_s,
\end{align*}
\]

for \( x \in \Omega \). It is easy to see that the iteration sequences \( \{\tilde{I}^{(m)}_s\}, \{\hat{I}^{(m)}_s\} \) possess the monotone property

\[
\tilde{I}^{(0)}_s \leq \tilde{I}^{(m)}_s \leq \tilde{I}^{(m+1)}_s \leq \hat{I}^{(m+1)}_s \leq \hat{I}^{(m)}_s \leq \tilde{I}^{(0)}_s, \quad x \in \overline{\Omega}
\]

and the limits

\[
\tilde{I}_s = \lim_{m \to \infty} \tilde{I}^{(m)}_s, \quad \hat{I}_s = \lim_{m \to \infty} \hat{I}^{(m)}_s
\]

(3.6)
exist and satisfy the equations
\begin{equation}
\begin{aligned}
-\partial_t \Delta \tilde{I}_s &= [\beta(x) - \gamma(\tilde{I}_s, x)] - \frac{\beta(x)}{N_s} \tilde{I}_s, \quad x \in \Omega, \\
-\partial_t \Delta L_s &= [\beta(x) - \gamma(L_s, x)] - \frac{\beta(x)}{N_s} L_s, \quad x \in \Omega.
\end{aligned}
\end{equation}

(3.7)

The pair \( \tilde{I}_s \) and \( L_s \) are the maximal and minimal solution to (3.1) in \( (\tilde{I}, \tilde{I}) \). Moreover, since that \( I_0(x) \in (\tilde{I}, \tilde{I}) \), the time-dependent solution \( I(x, t) \) to (1.4) satisfies
\[ L_s(x) \leq \liminf_{t \to \infty} I(x, t) \leq \limsup_{t \to \infty} I(x, t) \leq \tilde{I}_s(x), \quad x \in \Omega. \]

If \( b > \max_{x \in \Omega} \frac{(\gamma_M(x) - \gamma_m(x))N^*}{\beta(x)} \), the positive solution to (3.1) is unique, and we then have \( \tilde{I}_s = L_s := I^*_s \) and \( \lim_{t \to \infty} I(x, t) = I^*_s(x) \) uniformly in \( \Omega \).

Based on the above discussions, we have the following results about stability.

**Theorem 3.2.** When \( R_0 \leq 1 \), the unique disease-free equilibrium \( I^*_0 \) is globally asymptotically stable; When \( R_{0s} > 1 \) and \( b > \max_{x \in \Omega} \frac{(\gamma_M(x) - \gamma_m(x))N^*}{\beta(x)} \), the unique endemic equilibrium \( I^*_s \) is globally asymptotically stable.

When \( R_{0s} < 1 < R^*_0 \), the dynamical behaviors of the system (1.4) are complicated, even for the corresponding ODE problem
\begin{equation}
\begin{aligned}
I' &= (\beta - \gamma_m - (\gamma_M - \gamma_m) \frac{b}{b^*}) I - \frac{\beta}{N} I^2, \quad t > 0, \\
I(0) &= I_0 > 0,
\end{aligned}
\end{equation}

the positive equilibria should be the positive roots of the quadratic equation
\[ G(I) = \beta I^2 + [b \beta - (\beta - \gamma_m)N^*] I + bN^*(\gamma_M - \beta). \]

(3.9)

If \( R_{0s} < 1 < R^*_0 \), that is, \( \gamma_m < \beta < \gamma_M \) (moderate-risk), the equation \( G(I) = 0 \) may have two roots if \( \Delta > 0 \), where
\[ \Delta := [b \beta - (\beta - \gamma_m)N^*]^2 - 4bN^*(\gamma_M - \beta). \]

Therefore if \( 0 < b < b^* \) with \( b^* \) satisfies \( b^* \beta + 2\sqrt{b^* \beta N^* (\gamma_M - \beta)} = (\beta - \gamma_m)N^* \), then \( G(I) = 0 \) have two positive roots \( I_1^* \) and \( I_2^* \), so system (3.8) admits two endemic equilibria if and only if \( \gamma_m < \beta < \gamma_M \) and \( 0 < b < b^* \). While if \( \gamma_m < \beta < \gamma_M \) and \( b = b^* \), system (3.8) has one endemic equilibrium of multiplicity 2. But if \( \gamma_m < \beta < \gamma_M \) and \( b > b^* \), system (3.8) has no positive equilibrium.

As discussed in [18], if \( \gamma_m < \beta < \gamma_M \) and \( b > b^* \), the DFE is globally asymptotically stable since it is unique nonnegative steady-state solution. If \( \gamma_m < \beta < \gamma_M \) and \( b < b^* \), Theorem 3.4 in [18] showed that the equilibrium with low endemicity \( I_1^* \) is a hyperbolic saddle, and the equilibrium with high endemicity \( I_2^* \) is an anti-saddle. The system undergoes saddle-node bifurcation if \( b = b^* \).

4. Numerical illustration and discussion. In this section, we will carry out numerical simulations to illustrate our analytical results obtained above. Let us first consider the diffusive model (1.4) and fix some coefficients and initial functions. Assume that
\[ d_I = 1, \quad N^* = 1000, \quad b = 80, \quad I(0) = 200 - 30 \cos(6x), \]
\[ \Omega = (0, \pi), \quad \gamma_m(x) = 10 + 2 \cos(2x), \quad \gamma_M(x) = 12 + 2 \cos(2x), \]
then the asymptotic behaviors of the solution to problem (1.4) will be determined by the coefficients \( \beta(x) \).
Example 4.1. Fix big $\beta = 13.2 + 3 \sin(2x)$, since that

$$R_{0*} \geq \frac{\int_0^\pi \beta(x)dx}{\int_0^\pi \gamma_M(x)dx} = 1.1 > 1,$$

the environment is high-risk, and Theorem 3.2 shows that the endemic equilibrium exists and is stable. It is easy to see from Figure 1 (Left) that the solution $I(x,t)$ stabilizes to a positive equilibrium.

Example 4.2. For small $\beta = 8.2 + 2 \cos(2x)$, since that $\lambda_1^* = 1.8 > 0$ and then $R_{0*} < 1$ by Lemma 2.1, the environment is then low-risk. Theorem 3.2 shows that the solution decays to zero and the disease-free equilibrium is globally asymptotically stable, see Figure 1 (Right).

Next we consider the corresponding ODE problem (3.8) for the moderate-risk case, and assume that

$$N^* = 1000, \; \gamma_m = 10, \; \gamma_M = 12, \; \beta = 10.5,$$

direct calculation shows that $b^* \simeq 3.41885$.

Example 4.3. Fix big coefficient $b = 4$, since that $b > b^*$, the nonnegative steady-state solution to (3.8) is trivial and the DFE is globally asymptotically stable, it is shown in Figure 2 (Left) that the solutions with six different initial values always decay to zero. For small coefficient $b = 2$, there exists two equilibria: the one with low endemicity $I_1^* (\simeq 7.4942)$ is unstable, and the other with high endemicity $I_2^* (\simeq 38.125)$ is a locally asymptotically stable, any solution with the small initial value goes to zero and the solution with big initial value goes to the equilibrium with high endemicity $I_2^*$, see Figure 2 (Right).

The number of hospital beds denoted by the parameter $b$ (HBPR) is an important index to measure the medical resource available for controlling the endemic of infectious diseases [3, 24, 26]. Based on the work by Shan and Zhu [18], we establish and study a diffusive SIS model (1.4) and analyze the impact of the number of hospital beds on the spatial transmission of infectious diseases, and we consider a simpler case by assuming that the distribution of the hospital resources (hospital beds) remain a constant.
If $b = 0$, the system (1.4) will be simplified as

\begin{equation}
\begin{cases}
I_t - d_t \Delta I = (\beta(x) - \gamma_m(x))I - \frac{\beta(x)}{N^2} I^2, & x \in \Omega, \ t > 0, \\
\frac{\partial I}{\partial n} = 0, & x \in \partial \Omega, \ t > 0, \\
I(x, 0) = I_0(x) \geq 0, \ I_0(x) \neq 0, & x \in \overline{\Omega}.
\end{cases}
\end{equation}

(4.1)

We then have $R_{0*} = R_0^* (\equiv R_0)$, it is well-known ([8]) that the dynamics of the system (4.1) are completely determined by the basic reproduction number $R_0$, that is, the disease will be eliminated if $R_0 < 1$, otherwise the unique endemic equilibrium exists and is always stable.

If $b \neq 0$, our results show that when $R_{0*} > 1$, the environment is **high-risk**, that is, the average of the disease contact transmission is greater than that of the maximum recovery rate, increasing the number of hospital beds can only reduce limited number of the infectious, but cannot eliminate the disease. In this case, we have to reduce the contact rate $\beta(x)$, and therefore the best strategy to control the transmission of the disease is isolating and quarantining of people in the high-risk habitat, which has been proved to be effective. The recent typical example is the control of the severe acute respiratory syndrome (SARS), which brook out in southern China in 2003 and caused an eventual 8,096 cases and 774 deaths [25]. Isolation and quarantine remain the most effective means to prevent the spread of SARS since there is no cure or protective vaccine for it that is safe for use in humans to date [10].

When $R_0^* \leq 1$, the average of the disease contact transmission is smaller comparatively, and the minimum recovery rate is enough. The disease is always vanishing no matter what the number of the hospital beds. We then say the environment is **low-risk** if $R_0^* \leq 1$.

When $R_{0*} < 1 < R_0^*$, the environment is **moderate-risk**, that is, the average of the disease contact transmission is between those of the maximal and minimal recovery rate, increasing the number of the hospital beds can eliminate the disease, small number of the hospital beds induces complicated issues. Therefore, preparedness of the minimum number of hospital beds in case of an emerging infectious
disease is a critical issue. Our results can help the public health agencies or administration arrange the appropriate number of hospital beds so as to optimize the allocation of public health resources.

In reality, the allocation and management of medical resources is a complicated process which is of paramount importance for decision making of public health. As one of the future work, we will generalize the simplified model to consider the spatial dependent allocation of medical resources in controlling the spatial endemic infectious disease. It is also an interesting topic to model and simulate certain infectious disease by making use of the clinical or historical data to estimate the least minimum of hospital beds for control of the spread of the disease. We leave this also for the future work.

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