A DIFFUSIVE SIS EPIDEMIC MODEL INCORPORATING THE MEDIA COVERAGE IMPACT IN THE HETEROGENEOUS ENVIRONMENT

JING GE*
School of Mathematical Science, Huaiyin Normal University
Huaian 223300, China
and
School of Mathematical Science, Yangzhou University
Yangzhou 225002, China

LING LIN
Department of Mathematics and Statistics, York University
Toronto, ON M3J 1P3, Canada

LAI ZHANG
Department of Mathematics and Mathematical Statistics
Umeå University, SE-90187, Umeå, Sweden

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Abstract. To explore the impact of media coverage and spatial heterogeneity of environment on the prevention and control of infectious diseases, a spatial-temporal SIS reaction-diffusion model with the nonlinear contact transmission rate is proposed. The nonlinear contact transmission rate is spatially dependent and introduced to describe the impact of media coverage on the transmission dynamics of disease. The basic reproduction number associated with the disease in the heterogeneous environment is established. Our results show that the degree of mass media attention plays an important role in preventing the spreading of infectious diseases. Numerical simulations further confirm our analytical findings.

1. Introduction. The history of human is that of struggling with infectious diseases, and the fighting will never stop. Although science and technology have been advanced substantially and medical facilities have greatly improved, infectious diseases remain a main threat to human’s health according to WHO recent claimant [28], and are responsible for a quarter of all deaths in the world annually. Since the pioneering work of Kermack and McKendrick in the early 20th century, a great number of epidemic models built on differential equations have been developed for understanding the dynamics of infectious diseases and the associated transmission mechanisms. Due to the rapid progress of science and technology, media coverage plays a critical role in preventing disease outbreak [29]. For example, the outbreak

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* Corresponding author.
of Zika virus in Latin America came into the global public view through mass media in the spring of 2016. Attributed to the intensive and timely media coverage of the virus including daily number of infected cases, as well as the media education of preventive measures for the public, the local outbreak did not evolve into a global outbreak. Generally speaking, it will be greatly helpful for preventing the spreading of diseases if the public is informed in time and educated enough knowledge.

In the past few years, several spatially-independent systems were developed to investigate the impact of media coverage on the spreading and control of infectious diseases [7, 14, 15, 20, 21, 22, 24, 25]. It was found that media coverage can greatly delay the epidemic peak and decrease severity of outbreak [17]. Although not always effective during the entire outbreak, media coverage can play a critical role in the early stages [29], where Xiao, Tang and Wu formulated and proposed the epidemic model with the 2009 A/H1N1 influenza outbreak data in Shanxi province of China. The proposed model was then converted into a switching system, and through a self-adaptive media impact switching on and off, we can better control the disease transmission. Recently, Yan and co-workers [30] extended a class epidemic model of SEIR type to stress the impact of media coverage, which leads to an epidemic model of SEIRM type. Based on the number of new hospital notifications and the accumulated number of hospital notifications for the Shanxi province, they quantitatively analyzed the effect of the parameters related to media coverage on disease transmission during the entire outbreak. Cui et al. [7] developed an SIS model with media coverage being described by a nonlinear contact transmission function.

There are two apparent constraints to the epidemic models that are built on ordinary differential equations. One is that parameters such as the contact transmission rate and the recovery rate are often considered as constants [4, 5], which ignore environmental heterogeneity. The other is the absence of individual propagation within the environment, which can be described under the framework of reaction diffusion equations [2, 12, 13, 16, 23]. While it has been commonly accepted that individual propagation and environmental heterogeneity are of paramount importance in the determination of persistence and eradication of infectious diseases [3, 10, 11], such as SARS and dengue fever, a challenge of studying an epidemic model with explicit consideration of these two factors is to establish the basic reproduction number, which is a key to describe the disease transmission mechanism, especially to reflect the spatial feature of the spreading in the region considered.

Allen et al. [3] proposed the following SIS epidemic model to explicitly account for the environmental heterogeneity and individual propagation

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

where \(S(t, x)\) and \(I(t, x)\) are the densities of susceptible and infected individuals at location \(x\) and time \(t\), respectively. \(d_I\) and \(d_S\), respectively, represent the diffusion rates of the susceptible and infected individuals. \(\beta(x)\) and \(\gamma(x)\) are the spatially dependent contact and recovery rates, which are positive bounded H"older continuous functions. They derived the basic reproduction number \(R_0\) which is dependent on the diffusion rate \(d_I\) and population movement, and further characterized a high-risk and low-risk domain. They found that in high-risk domain the disease-free equilibrium is always unstable if \(R_0\) is greater than unity, while in low-risk domain, the disease-free equilibrium is stable if and only if infected individuals diffuse above
a threshold value (in this case, $R_0$ is less than unity). Recently, Peng and Yi [18] considered a similar model in a heterogeneous environment where the low, moderate and high risk domain coexist. They proved the global stability of endemic equilibrium under the condition of identical diffusive rates (i.e., $d_S = d_I$), also see [19] and references therein for more details).

Very recently, there have been some studies on SIS type PDE model in the heterogeneous environment [1, 8, 27]. The authors of [1] investigated a two-strain pathogen reaction-diffusion model with a bilinear disease transmission rates. They studied the long-time behavior of the solution and provided the conditions of competitive exclusion and coexistence between the two strains. In [8], Cui and Lou considered effects of the diffusion and advection for an SIS epidemic model and introduced the basic reproduction number $R_0$ for advection rate and mobility of the infected individuals. They found that if the domain is low-risk, there may exist a critical value for the advection rate, under which the disease-free equilibrium changes its stability at least twice as $d_I$ varies from zero to infinity, while the disease-free equilibrium is unstable for any $d_I$ when the advection rate is bigger than the critical value. Different from problem (1.1), the diffusive SIS model with the mass action infection was studied by Wu and Zou in [27], they overcame the difficulties due to the unboundedness of the nonlinear term and the nonlocal term in the equilibrium problem and explored the asymptotic profiles of the endemic steady-state for small and large diffusion rates.

Epidemiologically, the nonlinear contact transmission rate plays 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 nature world, the contact transmission rate depends on multiple factors, such as temperature, climate and the impact of the intensity of mass media coverage. Furthermore, in some area when there is an infectious disease starting to emerge or re-emerge and spread, the contact transmission must be changeable due to the complexity of human activities.

To grasp the impact of the media coverage and heterogenous environment on preventing and controlling the transmission of infectious diseases, we consider the following reaction-diffusion problem

\[
\begin{aligned}
&S_t - d_S \Delta S = -\beta(x, m, I) \frac{SI}{S+I} + \gamma(x) I + \Lambda(x), & t > 0, & x \in \Omega, \\
&I_t - d_I \Delta I = \beta(x, m, I) \frac{SI}{S+I} - \gamma(x) I, & t > 0, & x \in \Omega, \\
&S(t, x) = I(t, x) = 0, & t > 0, & x \in \partial \Omega, \\
&S(0, x) = S_0(x) > 0, I(0, x) = I_0(x) \geq 0, & I_0(x) \neq 0, & x \in \Omega, 
\end{aligned}
\]

where $\Lambda(x)$ is positive and continuous on $\Omega$, which resembles the recruitment through birth and immigration into the susceptible class at location $x$. The contact rate $\beta(x, m, I)$ is extended from model (1.1) by incorporating the influence of infected individuals and media coverage. To model the impact of media coverage, a media impact factor function is considered, which is of exponential function, such as $\beta(x, m, I) = e^{-mI}$, or other nonlinear functions, such as $\beta(x, m, I) = \beta_1 - mI$ and $\beta(x, m, I) = \frac{mSI}{S+I}$, where the positive constant $m$ is an index reflecting the impact of media coverage. In the present paper, we consider a general nonlinear contact function and specify the concrete form when numerical simulation is performed. We assume that $\beta(x, m, I)$ is a positive continuous function and further assume that $\beta(x, m, I)$ is monotonically decreasing with respect to $m$ and $I$, respectively.
The null Dirichlet boundary condition \( S(t, x) = I(t, x) = 0 \) means that there is no infection outside of the environment and on the boundary.

Different from the classical reaction-diffusion epidemic models including model (1.1), our epidemic model (1.2) involves nonlinear contact transmission rate and the non-constant recovery rate in heterogeneous environment. Thus the basic reproduction number for (1.2) can be dependent on all of the considered factors. For these reasons, we will redefine the basic reproduction number, and use it to explore the temporal and spatial transmission mechanisms of infectious diseases.

The rest of this paper is arranged as follows. In Section 2, we introduce the basic reproduction number and analytically study its properties. In Section 3, we investigate the existence and stability of the disease-free equilibrium. Section 4 is devoted to the global stability of the unique endemic equilibrium when the diffusion coefficients are identical. Numerical simulations are presented in Section 5 to graphically illustrate the impact of the media coverage. The paper ends with a discussion section.

2. The basic reproduction number. In this section, we will introduce the basic reproduction number \( R_0^D(\Omega) \) and analyze its properties and implications for the reaction-diffusion system (1.2). Epidemiologically, \( R_0 \) is defined as the expected number of secondary infections caused by an infective individual upon entering a totally susceptible population. It characterizes the infection risk of a disease, and further provides information for controlling disease transmission. For epidemic models described by spatially-independent systems, the basic reproduction number can be obtained by the next generation matrix method [9, 26]. However this method fails when the rates of contact and recovery are environmentally dependent, which is the case of our epidemic model. To handle this problem, we use the variational method as stated in [3].

Let us introduce the basic reproduction number \( R_0^D(\Omega) \) for (1.2) by
\[
R_0^D(\Omega) := R_0^D(\Omega, \beta(x, m, I), \gamma(x), d_I) = \sup_{\phi \in H^1_0(\Omega), \phi \neq 0} \left\{ \frac{\int_{\Omega} \beta(x, m, 0) \phi^2 dx}{\int_{\Omega} (d_I |\nabla \phi|^2 + \gamma(x) \phi^2) dx} \right\}.
\]
When the contact and recovery rates are constant, that is, \( \beta(x, m, 0) \equiv \beta^* \) and \( \gamma(x) \equiv \gamma^* \), \( R_0^D = \frac{\beta^*}{\gamma^*} \) (where \( \lambda(\Omega) \) is the principal eigenvalue of the operator \(-\Delta\) with homogeneous Dirichlet boundary condition), which is consistent with the one obtained by the spectral radius of the next generation matrix [9].

For convenience, we introduce some notations. For a closed linear operator \( A : D(A) \subset L^2(\Omega) \to L^2(\Omega) \), where \( D(A) \) is the domain of \( A \), the spectral radius \( \rho(A) \) of \( A \) is defined by
\[
\rho(A) = \sup \{ \text{Re}(\lambda) : \lambda \in \sigma_p(A) \},
\]
where \( \sigma_p(A) \) denotes the point spectrum of \( A \).

For any \( w \in C(\Omega, \mathbb{R}) \), it is well-known that \( \rho(d_I \Delta + w) = \lambda^* \) and \( \lambda^* \) is the principal eigenvalue of the operator \( d_I \Delta + w \) under the Dirichlet boundary condition on \( \partial \Omega \). Moreover, the corresponding eigenfunction \( \phi(x) \) is strictly positive in \( \Omega \), and
\[
-\lambda^* = \min \left\{ \int_{\Omega} (d_I |\nabla \phi(x)|^2 - w(x) \phi^2(x)) dx : \phi \in H^1_0(\Omega), \int_{\Omega} \phi^2(x) dx = 1 \right\}.
\]
With the above definitions of \( \lambda^* \) and \( R_0^D(\Omega) \), we have the following result (also see Lemma 2.3 in [3] for details):

\[\text{Ref. to the next page}\]
Lemma 2.1. \( \text{sign}(R_0^D - 1) = \text{sign}(\lambda^*) \), where \( \lambda^* \) is the principal eigenvalue of the reaction-diffusion problem

\[
\begin{cases}
-d_I \Delta \psi = (\beta(x,m,0) - \gamma(x))\psi - \lambda^* \psi, & x \in \Omega, \\
\psi(x) = 0, & x \in \partial \Omega.
\end{cases}
\] (2.1)

From the expression of \( R_0^D(\Omega) \), we have the following properties, which are similar to Theorem 2 in [3].

**Theorem 2.2.** The following assertions hold.

(a) \( R_0^D(\Omega, \beta(x,m,0), \gamma(x), d_I) \) is a positive and monotonically increasing function with respect to \( \beta(x,m,0) \), and decreasing with respect to \( \gamma(x) \) and \( d_I \), respectively; that is,

\[
R_0^D(\Omega, \beta_1(x,m,0), \gamma_2(x), d_{I,2}) \leq R_0^D(\Omega, \beta_2(x,m,0), \gamma_1(x), d_{I,1})
\]

provided that \( \beta_1(x,m,0) \leq \beta_2(x,m,0) \), \( \gamma_1(x) \leq \gamma_2(x) \) in \( \overline{\Omega} \) and \( d_{I,1} \leq d_{I,2} \).

(b) \( R_0^D(\cdot, \beta(x,m,0), \gamma(x), d_I) \) is a strictly increasing function for fixed \( \beta(x,m,0) \), \( \gamma(x) \), \( d_I \), in the sense that

\[
R_0^D(\Omega_1, \beta(x,m,0)|_{\Omega_1}, \gamma(x)|_{\Omega_1}, d_I) \leq R_0^D(\Omega_2, \beta(x,m,0), \gamma(x), d_I)
\]

provided that \( \Omega_1 \subseteq \Omega_2 \subseteq \mathbb{R}^n \), with the strictly inequality if \( \Omega_2 \setminus \Omega_1 \) is a nonempty open set.

(c) \( R_0^D(\Omega, \beta(x,m,0), \gamma(x), d_I) \) is a strictly decreasing function with respect to the parameter \( m \), in the sense that

\[
R_0^D(\Omega, \beta(x,m_2,0), \gamma(x), d_I) \leq R_0^D(\Omega, \beta(x,m_1,0), \gamma(x), d_I)
\]

provided that \( 0 < m_1 < m_2 \).

(d) If \( \beta(x,m,0) \equiv \beta^* \) and \( \gamma(x) \equiv \gamma^* \), then \( R_0^D(\Omega) = \frac{\beta^*}{d_I \lambda(\Omega)} \), where \( \lambda(\Omega) \) is the principal eigenvalue of the operator \( -\Delta \) with homogeneous Dirichlet boundary condition.

**Proof.** The monotonicity in (a) and (c) directly follows by the expression of \( R_0^D(\Omega) \). The proof of the monotonicity in (b) is similar to that of Corollary 2.3 in [6].

It remains to verify (d). Denote \( \lambda(\Omega) \) as the principal eigenvalue of the operator \( -\Delta \) with homogeneous Dirichlet boundary condition. That is, there exists a function \( \phi(x) > 0 \) satisfying

\[
\begin{cases}
-\Delta \phi = \lambda(\Omega) \phi, & x \in \Omega, \\
\phi = 0, & x \in \partial \Omega.
\end{cases}
\]

Then we have

\[
\int_{\Omega} d_I |\nabla \phi|^2 dx = -d_I \int_{\Omega} \phi \Delta \phi dx = d_I \int_{\Omega} \lambda(\Omega) \phi^2 dx
\]
due to the Dirichlet boundary condition. Therefore, from the expression of \( R_0^D(\Omega) \) we deduce \( R_0^D(\Omega) = \frac{\beta^*}{d_I \lambda(\Omega)} \).

**Remark 2.1.** From the above property in (c), we know that the \( R_0^D(\Omega) \) is decreasing with \( m \). Epidemiologically, if we strengthen the intensity of the mass media reports, the epidemic risk will become smaller, then the epidemic will be controlled rapidly and effectively.
3. The stability of disease-free equilibrium. In this section, we aim to establish the existence, uniqueness, stability of disease-free equilibrium in terms of the threshold \( R_0^D (\Omega) \). First, if the initial values \( S_0(x) \) and \( I_0(x) \) are nonnegative and nontrivial, we can obtain that both \( S(t,x) \) and \( I(t,x) \) are positive and bounded for \( x \in \Omega \) and \( t > 0 \) by the maximum principle for parabolic systems whenever the solution exists. Therefore, it follows from the standard theory for semi-linear parabolic systems that \( (S(t,x), I(t,x)) \) is also a classical solution for all \( t > 0 \) [6].

It is easy to see that the disease-free equilibrium is a solution \((S_0(x), 0)\), which is independent of time. Moreover, \( S_0(x) > 0 \) for \( x \in \Omega \) and it satisfies

\[
\begin{cases}
-d_s \Delta S_0 = \Lambda(x), & x \in \Omega, \\
S_0(x) = 0, & x \in \partial \Omega.
\end{cases}
\]

(3.1)

In what follows, we establish the global stability for the disease-free equilibrium.

**Theorem 3.1.** Suppose \( R_0^D (\Omega) < 1 \), the disease-free equilibrium \((S_0(x), 0)\) is globally asymptotically stable, in the sense that all its nonnegative solutions to problem (1.2) converge to the disease-free equilibrium \((S_0(x), 0)\) as \( t \to +\infty \). It is unstable if \( R_0^D (\Omega) > 1 \).

**Proof.** Firstly, from the second equation in (1.2), we can derive

\[
I_t \leq d_t \Delta I + (\beta(x, m, I) - \gamma(x))I, \quad x \in \Omega, \quad t > 0
\]

because \( I(t,x) \) is nonnegative. Subsequently, let \( \bar{I} \) be any solution satisfying

\[
\bar{I}_t \geq d_t \Delta \bar{I} + (\beta(x, m, \bar{I}) - \gamma(x))\bar{I}, \quad x \in \Omega, \quad t > 0,
\]

with \( \bar{I}(0, x) \geq I(0, x) \) and \( \bar{I}(t, x) = 0 \) for \( x \in \partial \Omega \) and \( t > 0 \). We conclude \( 0 \leq I(t,x) \leq \bar{I}(t,x) \) for \( x \in \Omega \) and \( t > 0 \) by the comparison principle. The condition \( R_0^D (\Omega) < 1 \) implies that \( \lambda^* = \rho(d_t \Delta + \beta(x, m, 0) - \gamma) < 0 \), then there exists a corresponding eigenfunction \( \phi(x) > 0 \) satisfying

\[
\begin{cases}
-d_t \Delta \phi = [\beta(x, m, 0) - \gamma(x)]\phi - \lambda^* \phi, & x \in \Omega, \\
\phi = 0, & x \in \partial \Omega.
\end{cases}
\]

Let \( \bar{T}(t,x) = Me^{\lambda^* t} \phi(x) \), then we can choose \( M \) sufficiently large such that \( M\phi(x) \geq I_0(x) \). We will show \( \bar{T}(t,x) \) is the upper-solution of \( I(t,x) \). In fact,

\[
\begin{align*}
\bar{T}_t - [d_t \Delta \bar{T} + (\beta(x, m, \bar{T}) - \gamma(x))\bar{T}] &= Me^{\lambda^* t}[(\beta(x, m, 0) - \beta(x, m, \bar{T}) - \gamma(x))\phi] \\
&= Me^{\lambda^* t}[(\beta(x, m, 0) - \beta(x, m, \bar{T}))\phi] \geq 0
\end{align*}
\]

(3.2)

due to the monotonicity of \( \beta(x, m, I) \). From the fact that

\[
\lim_{t \to +\infty} \bar{T}(t,x) = \lim_{t \to +\infty} Me^{\lambda^* t} \phi(x) = 0 \quad \text{uniformly for } x \in \Omega,
\]

we derive \( \lim_{t \to +\infty} I(t,x) = 0 \) uniformly for \( x \in \Omega \).

It remains to verify that \( S(t, \cdot) \) tends to \( S_0 \) as \( t \to \infty \). In fact, due to \( -d_s \Delta S_0 = \Lambda \), we can rewrite the first equation in (1.2) as

\[
\frac{\partial(S - S_0)}{\partial t} = d_s \Delta (S - S_0) + [\gamma - \frac{\beta S}{S + I}]I, \quad x \in \Omega, \quad t > 0.
\]

(3.3)

Then the boundedness of \( \beta(x, m, I) \) and \( \gamma(x) \) yields that

\[
\left\| \gamma - \frac{\beta S(t, \cdot)}{S(t, \cdot) + I(t, \cdot)} \right\|_{L^\infty(\Omega)} \leq M_1 e^{\lambda^* t}, \quad t > 0,
\]

(3.4)
where $M_1$ is a positive constant. Applying the variation-of-constant formula to (3.3) together with the Dirichlet boundary condition, we deduce that
\[
\|S(t, \cdot) - S_0\|_{L^\infty(\Omega)} \leq Ce^{\lambda t} (\|S(0, \cdot) - S_0(0, \cdot)\|_{L^\infty(\Omega)} + M_1 \int_0^t e^{-(\lambda - \lambda^*) s} ds) \to 0 \text{ as } t \to \infty,
\]
where $\lambda = \rho(d_S\Delta) < 0$.

Next, if $R^D_0(\Omega) > 1$, it is well-known that there exist a $\lambda_0 > 0$ and $\psi > 0$ satisfying
\[
\begin{align*}
&\left\{ \begin{array}{ll}
d_t \Delta \psi + [\beta(x, m, 0) - \gamma] \psi = \lambda_0 \psi, & x \in \Omega, \\
\psi = 0, & x \in \partial \Omega,
\end{array} \right.
\end{align*}
\]
where $\lambda_0$ is a principal eigenvalue, and $\psi > 0$ is the corresponding eigenfunction. Thus the disease-free equilibrium $(S_0(x), 0)$ is unstable via standard linearization and spectral analysis. The proof is completed. \qed

4. Existence and stability of the endemic equilibrium. We will be interested primarily in the existence and stability of endemic equilibrium solutions to problem (1.2), which is more important in the spreading of infectious diseases.

For the notations convenience, we denote the endemic equilibrium of problem (1.2) by $(S^*(x), I^*(x))$, which satisfies the following elliptic problem
\[
\begin{align*}
&\left\{ \begin{array}{ll}
0 = d_S \Delta S - \beta(x, m, I) S I \frac{S}{S + I} + \gamma(x) I + \Lambda(x), & x \in \Omega, \\
0 = d_I \Delta I + \beta(x, m, I) S I \frac{S}{S + I} - \gamma(x) I, & x \in \Omega, \\
S(x) = I(x) = 0, & x \in \partial \Omega.
\end{array} \right. 
\end{align*}
\]

It follows from the strong maximum principle that $S^*(x)$ and $I^*(x)$ are strictly positive in $\Omega$. Adding the two equations in (4.1), we have the equivalent problem
\[
\begin{align*}
&\left\{ \begin{array}{ll}
0 = \Delta(d_S S + d_I I) + \Lambda(x), & x \in \Omega, \\
0 = d_I \Delta I + \beta(x, m, I) S I \frac{S}{S + I} - \gamma(x) I, & x \in \Omega, \\
S(x) = I(x) = 0, & x \in \partial \Omega.
\end{array} \right. 
\end{align*}
\]

We can deduce that $S + \frac{d_I}{d_S} I = S_0$ from the first equation in (4.2), where $S_0$ is unique positive function satisfying (3.1). Therefore we can write $S$ as
\[
S = \frac{d_S S_0 - d_I I}{d_S}.
\]

We obtain the following equality by substituting (4.3) into the second equation in (4.2)
\[
d_I \Delta I + \left[\beta - \gamma - \beta \frac{d_S I}{d_S S_0 + (d_S - d_I) I}\right] I = 0.
\]

Now we give the existence of the endemic equilibrium.

**Theorem 4.1.** Suppose that $R^D_0(\Omega) > 1$. Then problem (4.2) admits a unique nonnegative solution $(S^*(x), I^*(x))$ such that $S^*(x), I^*(x) \in C^2(\Omega)$ and $I^*(x) > 0$. Moreover, $S^*(x) > 0$, and $0 < I^*(x) < \frac{d_S}{d_I} S_0(x)$ for $x \in \Omega$.

**Proof.** Owing to (4.3), we know that $(S^*(x), I^*(x))$ is a positive solution to problem (4.2) if and only if $I^*(x)$ is a positive solution to problem (4.4) with $I^*(x) < \frac{d_S}{d_I} S_0(x)$ and $S^*(x) = \frac{d_S S_0(x) - d_I I^*(x)}{d_S}$ for $x \in \Omega$.

In what follows, we consider the boundary value problem
\[
G(I) := d_I \Delta I + f(x, m, I) I = 0 \text{ for } x \in \Omega \text{ and } I(x) = 0 \text{ for } x \in \partial \Omega,
\]
where
\[ f(x, m, I) := \beta(x, m, I) \left( 1 - \frac{d_S}{d_S S_0 + (d_S - d_I)I} \right) - \gamma(x), \quad x \in \Omega, ~ I \in \left[ 0, \frac{d_S}{d_I} S_0(x) \right]. \]

In order to obtain the existence of the positive solution to (4.2), we will construct a lower-solution \( \underline{I} \) and upper-solution \( \overline{I} \) to problem (1.2) such that \( 0 < \underline{I} \leq \overline{I} \leq \frac{d_S}{d_I} S_0 \). Since \( R_0^D(\Omega) > 1 \), we derive that
\[ \lambda^* = \rho(d_I \Delta + \beta(x, m, 0) - \gamma(x)) > 0 \]
is the principal eigenvalue of the operator \( (d_I \Delta + \beta(x, m, 0) - \gamma(x)) \) and the corresponding eigenfunction \( \phi(x) \) is strictly positive for all \( x \in \Omega \).

Setting \( \underline{I} = \varepsilon \phi \) and \( \overline{I} = \frac{d_S}{d_I} S_0 \), we will show that \( \underline{I} \) and \( \overline{I} \) are lower and upper-solution to problem (4.5) if \( \varepsilon > 0 \) is sufficiently small. Recall that \( d_I \Delta \phi + \beta(x, m, 0) \phi - \gamma(x) \phi = \lambda^* \phi \).

Direct calculations yield that
\[
G(\underline{I}) = d_I \Delta \varepsilon \phi + \varepsilon \phi f(x, \varepsilon \phi) \\
= \varepsilon [d_I \Delta \phi + \beta(x, m, \varepsilon \phi)(1 - \frac{d_S}{d_S S_0 + (d_S - d_I)\varepsilon \phi}) \phi - \gamma \phi] \\
= \varepsilon [\lambda^* + \beta(x, m, \varepsilon \phi) - \beta(x, m, 0) - \beta(x, m, \varepsilon \phi) \varepsilon^{-1} (d_S S_0 + (d_S - d_I)\varepsilon \phi)].
\]

If \( \varepsilon \) is selected sufficiently small, together with the monotonicity of \( \beta(x, m, I) \) we easily obtain \( G(\underline{I}) > 0 \) due to \( \lambda^* > 0 \), namely, \( \underline{I} \) is a lower-solution to problem (4.5).

On the other hand, since
\[
G(\overline{I}) = d_I \Delta \overline{I} + \overline{I} f(x, \overline{I}) \\
= d_I \Delta \overline{I} + \overline{I} \beta(x, m, \overline{I})(1 - \frac{d_S}{d_S S_0 + (d_S - d_I)\overline{I}}) - \gamma(\overline{I}) \\
= -\Lambda(x) - \gamma(\overline{I}) < 0
\]
for \( x \in \Omega \) and \( \overline{I}(x) = 0 \) on \( \partial \Omega \), it follows that \( \overline{I} \) is an upper-solution to (4.5). Moreover, if \( \varepsilon \) is selected sufficiently small, we have \( \underline{I} \leq \overline{I} \leq \frac{d_S}{d_I} S_0(x) \) on \( \Omega \). We conclude that there exists an \( I^*(x) \in [\underline{I}, \overline{I}] \) satisfying problem (4.5). Therefore, we derive \( 0 < I^*(x) \leq \frac{d_S}{d_I} S_0(x) \).

In the following, we will prove \( I^*(x) < \frac{d_S}{d_I} S_0(x) \) in \( \Omega \), it suffices to prove that \( S^*(x) > 0 \) in \( \Omega \). In fact, thanks to the boundedness of \( \beta(x, m, I) \), we can obtain \( -d_S \Delta S^* \geq -CS^* \) from the first equation in problem (4.1), where \( C \) is the positive constant. Therefore, we derive \( S^*(x) > 0 \) in \( \Omega \) by applying the strong maximum principle.

Noticing that \( f(x, m, I) \) is strictly decreasing with respect to \( I \) for \( I \in [0, \frac{d_S}{d_I} S_0(x)] \), we can show the uniqueness by applying the multiply-multiply-subtract-integrate trick as stated in [3].

We have obtained the existence and uniqueness of endemic equilibrium when \( R_0^D(\Omega) > 1 \) and that both \( S^* \) and \( I^* \) are positive in \( \Omega \). In what follows, we will investigate the stability of the endemic equilibrium. If the diffusion coefficients \( d_S \) and \( d_I \) are not equal, it is hard to analyze its stability, even for the local stability. With identical diffusion rates, namely, \( d_S = d_I \), we can obtain that the endemic equilibrium is globally asymptotically stable for problem (1.2).

Theorem 4.2. Suppose that \( R_0^D(\Omega) > 1 \) and \( d_S = d_I \). Then the endemic equilibrium \((S^*(x), I^*(x))\) of problem (1.2) is globally asymptotically stable.

Proof. To show the global stability, we only need to prove that all positive solutions to (1.2) converge to a unique endemic equilibrium \((S^*(x), I^*(x))\) as \( t \to \infty \).

First, let \( d = d_S = d_I \) and \( N(t, x) = S(t, x) + I(t, x) \). We can get the following equivalent system of (1.2)
\[
\begin{aligned}
N_t &= d \Delta N + \Lambda(x), & \quad & t > 0, x \in \Omega, \\
I_t &= d \Delta I + \beta(x, m, I) \frac{(N - I)}{N} - \gamma(x) I, & \quad & t > 0, x \in \Omega, \\
N(t, x) &= I(t, x) = 0, & \quad & t > 0, x \in \partial \Omega, \\
N(0, x) &= S_0(x) + I_0(x), & \quad & I(0, x) = I_0(x), \quad x \in \Omega.
\end{aligned}
\]

\[ N(t, x) = S(t, x) + I(t, x), \quad x \in \Omega. \]
It is obvious that the system (4.8) is quasi-increasing. By applying the strong maximum principle, we can easily deduce that any nonnegative solution $(S(t, x), I(t, x))$ to system (1.2) is strictly positive for $x \in \Omega$ and $t > 0$.

Next, we investigate the global asymptotic stability of the endemic equilibrium by the method of upper and lower solutions. Since it satisfies $N^*_t = d\Delta N^* + \lambda(x)$, $N^*(x) = S_0(x)$, we have

$$\frac{\partial (N - N^*)}{\partial t} = d\Delta (N - N^*).$$

Integrating above equality from 0 to $t$, we have

$$N(t, x) - N^*(x) = d \int_0^t \Delta (N(s, x) - N^*(x)) ds.$$

Using the Dirichlet boundary value condition and the classical semigroup theory, we deduce that

$$\|N(t, \cdot) - N^*(\cdot)\|_{L^\infty(\Omega)} \leq Ce^{-\lambda t}\|N^*\|_{L^\infty(\Omega)} \to 0 \text{ as } t \to +\infty,$$

where $\lambda = \rho(d\Delta)$ and $C$ are positive constants. Recalling $R_0^D(\Omega) > 1$ gives that $\lambda > 0$, it follows from the boundedness of $L^\infty$ norm that $N(t, x)$ converges to $N^*(x)$ uniformly in $\overline{\Omega}$.

Thus for any small $\varepsilon > 0$, there exists a constant $T^* > 0$ such that

$$N^*(x) - \varepsilon < N(t, x) < N^*(x) + \varepsilon, \quad x \in \overline{\Omega}, \quad t \geq T^*.$$

Let $\overline{I}_\varepsilon(t, x)$ be the solution to the parabolic problem

$$\begin{cases}
\left\{ \begin{array}{l}
\partial \overline{I}_\varepsilon + \beta(x, m, T_\varepsilon) \frac{(N^*_t + \varepsilon - I_\varepsilon)T_\varepsilon}{N^*_t + \varepsilon} - \gamma(x) \overline{I}_\varepsilon, \\
\overline{I}_\varepsilon(0, x) = 0,
\end{array} \right. & t > 0, \quad x \in \Omega, \\
\overline{I}_\varepsilon(t, x) = 0, & t > 0, \quad x \in \partial \Omega,
\end{cases}$$

where $\varepsilon > 0$ such that

$$I^*_t(x) - \varepsilon < N(t, x) < I^*_t(x) + \varepsilon, \quad x \in \overline{\Omega}, \quad t \geq T^*.$$

Let $\overline{I}_\varepsilon(t, x)$ be the solution to the parabolic problem

$$\begin{cases}
\left\{ \begin{array}{l}
\partial \overline{I}_\varepsilon + \beta(x, m, I_\varepsilon^*) \frac{(N^*_t + \varepsilon - I_\varepsilon^*)I_\varepsilon^*}{N^*_t + \varepsilon} - \gamma(x) \overline{I}_\varepsilon, \\
\overline{I}_\varepsilon(0, x) = 0,
\end{array} \right. & t > 0, \quad x \in \Omega, \\
\overline{I}_\varepsilon(t, x) = I(T^*, x), & x \in \partial \Omega.
\end{cases}$$

Similarly, let $\underline{I}_\varepsilon(t, x)$ be the solution to the parabolic problem

$$\begin{cases}
\left\{ \begin{array}{l}
\partial \underline{I}_\varepsilon + \beta(x, m, I_\varepsilon) \frac{(N^*_t - \varepsilon - I_\varepsilon)I_\varepsilon}{N^*_t - \varepsilon} - \gamma(x) \underline{I}_\varepsilon, \\
\underline{I}_\varepsilon(0, x) = 0,
\end{array} \right. & t > 0, \quad x \in \Omega, \\
\underline{I}_\varepsilon(t, x) = I(\varepsilon), & t > 0, \quad x \in \partial \Omega,
\end{cases}$$

and then

$$\lim_{t \to \infty} \underline{I}_\varepsilon(t, x) = I_\varepsilon(x) \quad \text{uniformly for } x \in \overline{\Omega}, \quad \text{where } I_\varepsilon(x) \text{ is a unique positive solution to the corresponding elliptic problem, that is,}

$$\begin{cases}
\partial I_\varepsilon - \beta(x, m, I_\varepsilon) \frac{(N^*_t - \varepsilon - I_\varepsilon)I_\varepsilon}{N^*_t - \varepsilon} - \gamma(x) I_\varepsilon, \\
I_\varepsilon(0, x) = I(T^*, x),
\end{cases}$$

By the comparison principle, we conclude that

$$I^*_t(t - T^*, x) \leq I(t, x) \leq \overline{I}_\varepsilon(t - T^*, x), \quad x \in \Omega, \quad t \geq T^*.$$

Therefore, we have

$$I_\varepsilon^*(x) \leq \liminf_{t \to \infty} I(t, x) \leq \limsup_{t \to \infty} I(t, x) \leq I_\varepsilon^*(x).$$

On the other hand, letting $\varepsilon \to 0$ yields

$$\lim_{\varepsilon \to 0} I_\varepsilon^*(x) = I^*(x),$$

where $I^*(x)$ is the unique solution to problem (4.2). It immediately follows that

$$\lim_{t \to \infty} I(t, x) = I^*(x), \quad x \in \overline{\Omega},$$
which together with \( \lim_{t \to \infty} N(t, x) = N^*(x) \) and \( N(t, x) = S(t, x) + I(t, x) \) gives \( \lim_{t \to \infty} S(t, x) = S^*(x) \) for \( x \in \Omega \). The proof is completed. \( \Box \)

5. **Numerical simulations.** In this section, we will perform numerical simulation to verify our theoretical results on the impact of media coverage. To this aim, we assume that

\[
d_I = 1, \quad d_S = 1, \quad I_0(x) = 1 + 1.5 \sin x + \sin \left( 6x - \frac{\pi}{2} \right),
\]

\[
\Omega = (0, \pi), \quad \gamma(x) = 0.01 + 0.005 \cos(4x), \quad \beta(x, m, I) = 100 \times e^{-m(I+4)},
\]

\[
S_0(x) = 4 + 10 \sin x - 4 \sin \left( 6x - \frac{\pi}{2} \right), \quad \Lambda(x) = 6 + 4 \cos(4x).
\]

Then we can see how the asymptotic behavior of epidemic model (1.2) is affected by the parameter \( m \).

**Example 5.1.** Set \( m = 1 \) and we have

\[
R_D^0(\Omega) := \sup_{\phi \in H^1_0(\Omega), \phi \neq 0} \left\{ \frac{\int_{\Omega} \beta(x, 1, 0) \phi^2 dx}{\int_{\Omega} 100 \times e^{-\sin^2 x} \phi^2 dx} \right\}
\]

\[
\geq \frac{100 \cos^2 x + (0.01 + 0.005 \cos(4x)) \sin^2 x}{100 e^{-\frac{\pi}{2}}} > 1.
\]

Theorem 4.2 shows that the endemic equilibrium exists and is stable. From Figure 1, one can see that the solution \((S(x, t), I(x, t))\) stabilizes to an endemic equilibrium. It means that the disease can persist if the intensity of media coverage is weaker. We can control the spread of the disease by strengthening the intensity of media coverage.

**Example 5.2.** Set \( m = 5 \) and we have

\[
R_D^0(\Omega) := \sup_{\phi \in H^1_0(\Omega), \phi \neq 0} \left\{ \frac{\int_{\Omega} \beta(x, 5, 0) \phi^2 dx}{\int_{\Omega} (\gamma(x) \phi^2 + \gamma(x) \phi^2) dx} \right\}
\]

\[
< \frac{\int_{\Omega} 100 \beta(x, 5, 0) \phi^2 dx}{\int_{\Omega} \gamma(x) \phi^2 dx} < \frac{20000}{2500} < 1.
\]

According to Theorem 3.1, the infected individuals \( I(x, t) \) goes to extinction quickly and the susceptible individuals \( S(x, t) \) converges to a positive steady-state, see Figure 2, which implies that the disease-free equilibrium is asymptotically stable. It also shows that increasing \( m \) (increase the intensity of media coverage) can help prevent spread of infectious disease. This theoretical results can remind the public health agencies or administration to take preventative action and educate individuals.
Figure 2. For $m = 5$, the infected individuals $I(x, t)$ with the given initial condition decays to zero quickly (left); the susceptible individuals $S(x, t)$ stabilizes to a positive steady-state (right).

6. Discussion. In this paper, we have formulated and investigated a deterministic SIS model describing the impact of media coverage on the transmission dynamics of infectious diseases. The impact of media coverage is characterized by the nonlinear function $\beta(x, m, I)$. We have introduced the basic reproduction number $R^D_0$ and explored its properties. It is well-known ([9]) that the dynamics of problem (1.2) are completely determined by the basic reproduction number $R^D_0$. The disease will become extinct if $R^D_0$ is less than unity, and persist otherwise. Then we investigated the asymptotic behaviors of problem (1.2) and found that the disease-free equilibrium is globally asymptotically stable when $R^D_0 < 1$ (Theorem 3.1). For $R^D_0 > 1$, we only verify that the endemic equilibrium is globally stable in the special case $d_S = d_I$ (Theorem 4.2). We conjecture that the endemic equilibrium is globally stable in the general situation (i.e., $d_S \neq d_I$), which deserves further study in the future.

With the increasing trend of globalization and the development of information technology, the mass media has a direct and rapid influence on everyday life, and changes human behaviors. In the last decade, epidemic models incorporating the impact of media coverage have causing considerable attention and investigations. Behavior changing can induce great effect in the transmission of a disease. Our model utilizes a nonlinear contact transmission rate to describe the media impact, which is similar to that proposed in [7], but there are essential differences between these two models. They considered a classical ordinary differential SIS epidemic model in homogeneous environment, while spatial diffusion term and heterogeneity are included in ours to accommodate high biological realism.

Compared to model (1.1) which also considered spatially dependent contact rate $\beta(x)$, our nonlinear contact transmission rate $\beta(x, m, I)$ further depends on the impact of media coverage. Psychologically, people will try to avoid infection contact once warned by media coverage. Consequently, the rate of contact with infection will decrease, preventing the spreading of infectious diseases. We explored the influence of the nonlinear transmission contact rate induced by media coverage and found that media coverage has an important role in the transmission and control of infectious diseases.

Our numerical simulations are consistent with our analytical findings. We observed that the intensity of media coverage can affect the persistence or extinction of the disease. That is, when the parameter $m$ is small, the infected and susceptible individuals stabilize to a positive equilibrium (see Fig.1). If the parameter $m$ is big, the infected individuals decays to zero quickly and susceptible individuals stabilizes to a positive steady-state (see Fig.2). Usually when media coverage is strengthened, populations will take effective strategy to avoid the contact with others by staying home or reducing social activities. For
the severe infectious disease, the mass media calls for the government to take measures, such as isolation and quarantine, which have proved to be effective. A typical example is the control of the severe acute respiratory syndrome (SARS), which first appeared in Guangdong province, China in November, 2002. In the following year the SARS spread rapidly throughout Asia and certain other parts of the world. The media coverage played an important role in preventing the global spreading of SARS, since it informed people of preventative measures, and people changed their behaviours accordingly such as avoiding exposure to the public.

Although, the media coverage cannot eliminate the disease eventually, it helps the public to get timely information of the ongoing situation and knowledge about how to protect themselves. These informative messages will influence human behaviour and affect the course of the epidemic, which is in line with our model predictions.

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E-mail address: gejingyu@163.com
E-mail address: linglin@my.yorku.ca
E-mail address: lai.zhang@umu.se