Research Article

Partial Differential Equations of an Epidemic Model with Spatial Diffusion

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The aim of this paper is to study the dynamics of a reaction-diffusion SIR epidemic model with specific nonlinear incidence rate. The global existence, positivity, and boundedness of solutions for a reaction-diffusion system with homogeneous Neumann boundary conditions are proved. The local stability of the disease-free equilibrium and endemic equilibrium is obtained via characteristic equations. By means of Lyapunov functional, the global stability of both equilibria is investigated. More precisely, our results show that the disease-free equilibrium is globally asymptotically stable if the basic reproduction number is less than or equal to unity, which leads to the eradication of disease from the population. When the basic reproduction number is greater than unity, then disease-free equilibrium becomes unstable and the endemic equilibrium is globally asymptotically stable; in this case the disease persists in the population. Numerical simulations are presented to illustrate our theoretical results.

1. Introduction

In this paper, we consider the following SIR epidemic model with a specific nonlinear incidence rate described by

\[
\begin{align*}
\frac{dS}{dt} &= \Lambda - \mu S - \frac{\beta SI}{1 + \alpha_1 S + \alpha_2 I + \alpha_3 SI}, \\
\frac{dI}{dt} &= \frac{\beta SI}{1 + \alpha_1 S + \alpha_2 I + \alpha_3 SI} - (\mu + \bar{d} + r) I, \\
\frac{dR}{dt} &= r I - \mu R,
\end{align*}
\]

where \( S, I, \) and \( R \) are susceptible, infectious, and recovered classes, respectively. \( \Lambda \) is the recruitment rate of the population, \( \mu \) is the natural death rate of the population, \( \bar{d} \) is the death rate due to disease, \( r \) is the recovery rate of the infective individuals, \( \beta \) is the infection coefficient, and \( \beta SI/(1 + \alpha_1 S + \alpha_2 I + \alpha_3 SI) \) is the incidence rate, where \( \alpha_1, \alpha_2, \alpha_3 \geq 0 \) are constants. It is very important to note that this incidence rate becomes the bilinear incidence rate if \( \alpha_1 = \alpha_2 = \alpha_3 = 0 \), the saturated incidence rate if \( \alpha_1 = \alpha_3 = 0 \) or \( \alpha_2 = \alpha_3 = 0 \),

the Beddington-DeAngelis functional response introduced in [1, 2] and used in [3] when \( \alpha_3 = 0 \), and Crowley-Martin functional response presented in [4–6] if \( \alpha_3 = \alpha_1 \alpha_2 \). Moreover, the function \( \beta S/(1 + \alpha_1 S + \alpha_2 I + \alpha_3 SI) \) satisfies the hypotheses (H1), (H2), and (H3) of general incidence rate presented by Hattaf et al. in [7]. From the biological point of view, the transmission rate of infectious diseases remains unknown in detail and may be different from one disease to another. In the classical epidemic models, this rate was assumed to be linear with respect to the numbers of susceptible and infected individuals. This assumption is based on the law of mass action which is more appropriate for communicable diseases such as influenza but not for sexually transmitted diseases such as HIV/AIDS. For one reason, the transmission rate in system (1) is assumed to be nonlinear and has the form \( \beta I/(1 + \alpha_1 S + \alpha_2 I + \alpha_3 SI) \) that measures the saturation effect which represents that the number of individual contacts reaches a certain maximum value due to social or spatial distribution of the population. For more details on the choice of the nonlinearity of the incidence rate, we refer the reader to the book of Capasso [8].
On the other hand, the spatial content of the environment has been ignored in the model (1). However, due to the large mobility of people within a country or even worldwide, spatially uniform models are not sufficient to give a realistic picture of disease diffusion. For this reason, the spatial effects cannot be neglected in studying the spread of epidemics. Therefore, we consider the following SIR epidemic model with specific nonlinear incidence rate and spatial diffusion:

\[
\frac{\partial S}{\partial t} = d_S \Delta S + \Lambda - \mu S(x,t)
\]

\[
- \frac{\beta S(x,t)I(x,t)}{1 + \alpha_1 S(x,t) + \alpha_2 I(x,t) + \alpha_3 S(x,t) I(x,t)},
\]

\[
\frac{\partial I}{\partial t} = d_I \Delta I + \frac{\beta S(x,t)I(x,t)}{1 + \alpha_1 S(x,t) + \alpha_2 I(x,t) + \alpha_3 S(x,t) I(x,t)} - (\mu + d + r) I(x,t),
\]

\[
\frac{\partial R}{\partial t} = d_R \Delta R + r I(x,t) - \mu R(x,t),
\]

(2)

where \(S(x,t), I(x,t),\) and \(R(x,t)\) represent the numbers of susceptible, infected, and removed individuals at location \(x\) and time \(t\), respectively. The positive constants \(d_S, d_I,\) and \(d_R\) denote the corresponding diffusion rates for these three classes of individuals.

The aim of this work is to investigate the global dynamics of the reaction-diffusion system (2). Note that \(R\) does not appear in the first two equations; this allows us to study the system

\[
\frac{\partial S}{\partial t} = d_S \Delta S + \Lambda - \mu S(x,t)
\]

\[
- \frac{\beta S(x,t)I(x,t)}{1 + \alpha_1 S(x,t) + \alpha_2 I(x,t) + \alpha_3 S(x,t) I(x,t)},
\]

\[
\frac{\partial I}{\partial t} = d_I \Delta I + \frac{\beta S(x,t)I(x,t)}{1 + \alpha_1 S(x,t) + \alpha_2 I(x,t) + \alpha_3 S(x,t) I(x,t)} - (\mu + d + r) I(x,t),
\]

with homogeneous Neumann boundary conditions

\[
\frac{\partial S}{\partial v} = \frac{\partial I}{\partial v} = 0, \quad \text{on } \partial \Omega \times (0, +\infty),
\]

(4)

and initial conditions

\[
S(x,0) = \phi_1(x) \geq 0, \quad I(x,0) = \phi_2(x) \geq 0, \quad x \in \Omega.
\]

Here, \(\Omega\) is a bounded domain in \(\mathbb{R}^n\) with smooth boundary \(\partial \Omega\). \(\partial S/\partial v\) and \(\partial I/\partial v\) are, respectively, the normal derivatives of \(S\) and \(I\) on \(\partial \Omega\).

The rest of paper is organized as follows. The next section deals with the global existence, positivity, and boundedness of solutions of problem (3)–(5). In Section 3, we discuss the stability analysis of equilibria. In Section 4, we present the numerical simulation to illustrate our result. Finally, the conclusion of our paper is in Section 5.

2. Global Existence, Positivity, and Boundedness of Solutions

In this section, we establish the global existence, positivity, and boundedness of solutions of problem (3)–(5) because this model describes the population. Hence, the population should remain nonnegative and bounded.

**Proposition 1.** For any given initial data satisfying the condition (5), there exists a unique solution of problem (3)–(5) defined on \([0, +\infty)\) and this solution remains nonnegative and bounded for all \(t \geq 0\).

**Proof.** System (3)–(5) can be written abstractly in the Banach space \(X = C(\Gamma) \times C(\Gamma)\) of the form

\[
u'(t) = F(u(t)), \quad t > 0,
\]

\[
u(0) = u_0 \in X,
\]

where \(u = (S, I), u_0 = (\phi_1, \phi_2), F(u) = \text{col}(d_S \Delta S, d_I \Delta I),\) and

\[
F(u(t)) = \left(\begin{array}{c}
\Lambda - \mu S - \frac{\beta SI}{1 + \alpha_1 S + \alpha_2 I + \alpha_3 SI} \\
\frac{\beta SI}{1 + \alpha_1 S + \alpha_2 I + \alpha_3 SI} - (\mu + d + r) I
\end{array}\right).
\]

(7)

It is clear that \(F\) is locally Lipschitz in \(X\). From [9], we deduce that system (6) admits a unique local solution on \([0, T_{\text{max}})\), where \(T_{\text{max}}\) is the maximal existence time for solution of system (6).

In addition, system (3) can be written in the form

\[
\frac{\partial S}{\partial t} = d_S \Delta S = F_1(S, I),
\]

\[
\frac{\partial I}{\partial t} = d_I \Delta I = F_2(S, I).
\]

It is easy to see that the functions \(F_1(S, I)\) and \(F_2(S, I)\) are continuously differentiable satisfying \(F_1(0, I) = \Lambda \geq 0\) and \(F_2(S, 0) = 0 \geq 0\) for all \(S, I \geq 0\). Since initial data of system (3) are nonnegative, we deduce the positivity of the local solution (see the book of Smoller [10]).

Now, we show the boundedness of solution. From (3)–(5) we have

\[
\frac{\partial S}{\partial t} - d_S \Delta S \leq \Lambda - \mu S,
\]

\[
\frac{\partial S}{\partial v} = 0,
\]

(9)

where

\[
S(x,0) = \phi_1(x) \leq \|\phi_1\|_\infty = \max_{x \in \Omega} \phi_1(x).
\]

By the comparison principle [11], we have \(S(x,t) \leq S_1(t),\)
where \( S_1(t) = \phi_1(x) e^{-\mu t} + (\Lambda / \mu) (1 - e^{-\mu t}) \) is the solution of the problem
\[
\begin{align*}
\frac{dS_1}{dt} &= -\mu S_1, \\
S_1(0) &= \|\phi_1\|_\infty.
\end{align*}
\] (10)
Since \( S_1(t) \leq \max \{\Lambda / \mu, \|\phi_1\|_\infty\} \) for \( t \in [0, \infty) \), we have that
\[
S(x,t) \leq \max \left\{ \frac{\Lambda}{\mu}, \|\phi_1\|_\infty \right\}, \quad \forall (x,t) \in \overline{\Omega} \times [0, T_{\text{max}}). 
\] (11)

From Theorem 2 given by Alikakos in [12], to establish the \( L^\infty \) uniform boundedness of \( I(x,t) \), it is sufficient to show the \( L^1 \) uniform boundedness of \( I(x,t) \).

Since \( \partial S / \partial \nu = \partial I / \partial \nu = 0 \) and \( (\partial / \partial t)(S+I) - \Delta (d_S S + d_I I) \leq -\mu (S+I) \), we get
\[
\frac{\partial}{\partial t}\left( \int_\Omega (S+I) \, dx \right) \leq \text{mes} (\Omega) \Lambda - \mu \left( \int_\Omega (S+I) \, dx \right).
\] (12)
Hence,
\[
\int_\Omega (S+I) \, dx \leq \text{mes} (\Omega) \max \left\{ \frac{\Lambda}{\mu}, \|\phi_1\|_\infty \right\}, \quad \forall (x,t) \in \overline{\Omega} \times [0, T_{\text{max}}). 
\] (13)
which implies that \( \sup_{t \geq 0} \int_\Omega I(x,t) \, dx \leq K := \text{mes}(\Omega) \max \{\Lambda / \mu, \|\phi_1\|_\infty\} \). Using [12, Theorem 3.1], we deduce that there exists a positive constant \( K^* \) that depends on \( K \) and on \( \|\phi_1\|_\infty \) such that
\[
\sup_{t \geq 0} \|I(\cdot,t)\|_\infty \leq K^*. 
\] (14)

From the above, we have proved that \( S(x,t) \) and \( I(x,t) \) are \( L^\infty \) bounded on \( \overline{\Omega} \times [0, T_{\text{max}}) \). Therefore, it follows from the standard theory for semilinear parabolic systems (see [13]) that \( T_{\text{max}} = +\infty \). This completes the proof of the proposition. \( \square \)

3. Qualitative Analysis of the Spatial Model

Using the results presented by Hattaf et al. in [7], it is easy to get that the basic reproduction number of disease in the absence of spatial dependence is given by
\[
R_0 = \frac{\beta \Lambda}{(\mu + a_1 \Lambda)(\mu + d + r)},
\] (15)
which describes the average number of secondary infections produced by a single infectious individual during the entire infectious period.

It is not hard to show that the system (3) is always a disease-free equilibrium of the form \( E_f(\Lambda / \mu, 0) \). Further, if \( R_0 > 1 \), the system (3) has an endemic stationary state \( E^*(S^*, I^*) \) where
\[
\begin{align*}
S^* &= \frac{2(a + a_2 \Lambda)}{\beta - a_1 a + a_2 \mu - a_3 \Lambda + \sqrt{\delta}}, \\
I^* &= \frac{\Lambda - \mu S^*}{a},
\end{align*}
\] (16)
with \( a = \mu + d + r \) and \( \delta = (\beta - a_1 a + a_2 \mu - a_3 \Lambda)^2 + 4a_2 \mu (a + a_2 \Lambda) \).

The objective of this section is to discuss the local and global stability of the equilibria.

3.1. Local Stability of the Equilibria. First, we linearize the dynamical system (3) around arbitrary spatially homogeneous fixed point \( E(S^*, I^*) \) for small space- and time-dependent fluctuations and expand them in Fourier space. For this, let
\[
S(\vec{x},t) \sim e^{\Lambda t} e^{j \vec{k} \cdot \vec{x}},
\] (17)
\[
I(\vec{x},t) \sim e^{\Lambda t} e^{j \vec{k} \cdot \vec{x}},
\] (18)
where \( \vec{x} = (x, y) \) and \( \vec{k} := (k_x, k_y) := (k^2, \vec{k}) \) are the wavenumber vector and frequency, respectively. Then we can obtain the corresponding characteristic equation as follows:
\[
\det (J - k^2 D - \lambda I_2) = 0,
\] (19)
where \( I_2 \) is the identity matrix, \( D = \text{diag}(d_S, d_I) \) is the diffusion matrix, and \( J \) is the Jacobian matrix of (3) without diffusion \( (d_S = d_I = 0) \) at \( E \) which is given by
\[
J = \frac{-\mu}{\beta S(1+a_2)} \begin{pmatrix}
\beta T (1 + a_2 I) & -\beta S (1 + a_2 S) \\
\beta S (1 + a_2 S) & \beta T (1 + a_2 I) \\
\end{pmatrix}.
\] (20)
The characterization of the local stability of disease-free equilibrium \( E_f \) is given by the following result.

Theorem 2. The disease-free equilibrium \( E_f \) is locally asymptotically stable if \( R_0 < 1 \) and it is unstable if \( R_0 > 1 \).

Proof. Evaluating (18) at \( E_f \), we have
\[
\left( \mu + k^2 d_S + \lambda \right) (a (R_0 - 1) - k^2 d_I - \lambda) = 0.
\] (21)
Clearly, the roots of (20) are \( \lambda_1(k) = (\mu + k^2 d_S) < 0 \) and \( \lambda_2(k) = a (R_0 - 1) - k^2 d_I \). Note that \( \lambda_1(k) \) is negative if \( R_0 < 1 \) for all \( k \). Hence \( E_f \) is locally asymptotically stable if \( R_0 < 1 \). If \( R_0 > 1 \), \( \lambda_2(0) \) is positive. So \( E_f \) is unstable. \( \square \)

Next, we focus on the local stability of the endemic equilibrium \( E^* \).

Theorem 3. The endemic equilibrium \( E^* \) is locally asymptotically stable if \( R_0 > 1 \).

Proof. Evaluating (18) at \( E^* \), we have
\[
\lambda^2 + a_1(k) \lambda + a_2(k) = 0,
\] (22)
where
\[
a_1(k) = \mu + k^2 (d_S + d_I)
\] (23)
\[
a_2(k) = \frac{I^* (1 + a_2 I^*)}{(1 + a_1 S^* + a_2 I^* + a_3 S^* I^*) S^*} + \frac{a_2 I^* + a_3 S^* I^*}{1 + a_1 S^* + a_2 I^* + a_3 S^* I^*},
\] (24)
\[
a_2(k) = \left( \mu + \frac{\beta I^* (1 + \alpha_2 I^*)}{(1 + \alpha_1 S^* + \alpha_2 I^* + \alpha_3 S^* I^*)^2} + k^2 d_S \right) \\
\times \left( \frac{\beta S^* (\alpha_1 I^* + \alpha_3 S^* I^*)}{(1 + \alpha_1 S^* + \alpha_2 I^* + \alpha_3 S^* I^*)^2} \right) \\
+ \frac{\beta^2 S^* I^* (1 + \alpha_1 I^*) (1 + \alpha_1 S^*)}{(1 + \alpha_1 S^* + \alpha_2 I^* + \alpha_3 S^* I^*)^2}.
\]

(22)

We have \( a_1 > 0 \) and \( a_2 > 0 \); then \( E^* \) is locally asymptotically stable.

3.2. Global Stability of the Equilibria. The purpose of this subsection is to determine the global stability for reaction-diffusion equations (3)–(5) by constructing Lyapunov functionals. These Lyapunov functionals are obtained from those for ordinary differential equations (1) by applying the method of Hattaf and Yousfi presented in [14].

The system (1) is particular case of the model proposed by Hattaf et al. [7] with \( f(S, I) = \frac{\beta S}{1 + \alpha_2 S + \alpha_3 I} \). To study the global stability of \( E_f \), for (1), the authors Hattaf et al. [7] proposed the following Lyapunov functional:

\[
V_1 = S - S_0 - \int_{S_0}^S f (S_0, 0) f (X, 0) \, dX + I,
\]

(23)

where \( S_0 = \Lambda / \mu \).

From [14], we construct the Lyapunov functional for system (3)–(5) at \( E_f \) as follows:

\[
W_1 = \int_{\Omega} V_1 (S(x, t), I(x, t)) \, dx.
\]

(24)

Calculating the time derivative of \( W_1 \) along the solution of system (3)–(5), we have

\[
\frac{dW_1}{dt} = \int_{\Omega} \left\{ - \frac{\mu (S - S_0)^2}{S (1 + \alpha_1 S_0)} \right. \\
+ a I \left( \frac{1 + \alpha_1 S + \alpha_2 I + \alpha_3 S I}{1 + \alpha_1 S + \alpha_2 I + \alpha_3 S I} R_0 - 1 \right) \left. \right\} \, dx \\
- \frac{d_S S_0}{1 + \alpha_1 S_0} \int_{\Omega} \frac{|V S|^2}{S^2} \, dx \\
\leq \int_{\Omega} \left\{ - \frac{\mu (S - S_0)^2}{S (1 + \alpha_1 S_0)} + a I (R_0 - 1) \right\} \, dx \\
- \frac{d_S S_0}{1 + \alpha_1 S_0} \int_{\Omega} \frac{|V S|^2}{S^2} \, dx.
\]

(25)

Since \( R_0 \leq 1 \), we have \( dW_1/dt \leq 0 \). Thus, the disease-free equilibrium \( E_f \) is stable, and \( dW_1/dt = 0 \) if and only if \( S = S_0 \) and \( I(R_0 - 1) = 0 \). We discuss two cases as follows.

By the above discussion, we deduce that the largest compact invariant set in \( \Gamma = \{ (S, I) \mid (dW_1)/dt = 0 \} \) is just the single-ton \( E_f \). From LaSalle invariance principle [15], we conclude that \( E_f \) is globally asymptotically stable.

Using same technique, we construct a Lyapunov functional \( W_2 \) for system (3)–(5) at \( E^* \) from the Lyapunov functional \( V_2 \) defined by Hattaf et al. in [7]. It is easy to show that \( V_2 \) verifies the condition (15) given in [14]. Hence, it follows from [14, Proposition 2.1] that \( W_2 \) is a Lyapunov functional for the reaction-diffusion system (3)–(5) at \( E^* \) when \( R_0 > 1 \). We summarize the above in the following result.

**Theorem 4.** (i) If \( R_0 \leq 1 \), the disease-free equilibrium \( E_f \) of (3)–(5) is globally asymptotically stable for all diffusion coefficients.

(ii) If \( R_0 > 1 \), the endemic equilibrium \( E^* \) of (3)–(5) is globally asymptotically stable for all diffusion coefficients.

4. Numerical Simulations

In this section, we present the numerical simulations to illustrate our theoretical results. To simplify, we consider system (3) under Neumann boundary conditions

\[
\frac{\partial S}{\partial \nu} = \frac{\partial I}{\partial \nu} = 0, \quad t > 0, \quad x = 0, 1,
\]

(27)
and initial conditions
\[
S(x,0) = \begin{cases} 
1.1x, & 0 \leq x < 0.5, \\
1.1(1-x), & 0.5 \leq x \leq 1,
\end{cases}
\]
\[
I(x,0) = \begin{cases} 
0.5x, & 0 \leq x < 0.5, \\
0.5(1-x), & 0.5 \leq x \leq 1.
\end{cases}
\]  

In Figure 1, we show that the maximum value of the numbers of susceptibles and infectious individuals is concentrated at the middle of the interval [0, 1] and these numbers decrease linearly to zero at the boundaries \(x = 0\) and \(x = 1\).

Now, we choose the following data set of system (3): \(d_S = 0.1, d_I = 0.5, \Lambda = 0.5, \beta = 0.2, \mu = 0.1, \alpha_1 = 0.1, \alpha_2 = 0.02, \alpha_3 = 0.03, d = 0.1\) and \(r = 0.5\). By calculation, we have \(R_0 = 0.9524\). In this case, system (3) has a disease-free equilibrium \(E_f(5,0)\). Hence, by Theorem 4(i), \(E_f\) is globally asymptotically stable. Numerical simulation illustrates our result (see Figure 2).

In Figure 3, we choose \(\beta = 0.6\) and do not change the other parameter values. By calculation, we have \(R_0 = 2.8571\) which satisfy Theorem 4(ii); then the disease-free equilibrium is still present and the system (2) has a unique endemic equilibrium \(E^*(1.3625,0.5196)\). Therefore, by Theorem 2 and Theorem 4(ii), \(E_f\) is unstable, while \(E^*\) is globally asymptotically stable. Numerical simulation illustrates our result (see Figure 3).

5. Conclusion

In this paper, we investigated the dynamics of a reaction-diffusion epidemic model with specific nonlinear incidence rate. This specific nonlinear incidence rate includes the traditional bilinear incidence rate, the saturated incidence rate, the Beddington-DeAngelis functional response, and Crowley-Martin functional response. The global dynamics of the model are completely determined by the basic reproduction number \(R_0\). We proved that the disease-free equilibrium is globally asymptotically stable if \(R_0 \leq 1\), which leads to the eradication of disease from population. When \(R_0 > 1\) then disease-free equilibrium becomes unstable and a unique endemic equilibrium exists and is globally asymptotically stable, which means that the disease persists in the population.

From our theoretical and numerical results, we conclude that the spatial diffusion has no effect on the stability behavior.
of equilibria in the case of Neumann conditions and spatially constant coefficients.

Conflict of Interests

The authors declare that they have no conflict of interests regarding the publication of this paper.

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