Hindawi Publishing Corporation
International Journal of Partial Differential Equations
Volume 2014, Article ID 186437, 6 pages
http://dx.doi.org/10.1155/2014/186437

Research Article

Partial Differential Equations of an Epidemic Model with Spatial Diffusion

El Mehdi Lotfi,[1] Mehdi Maziane,[1] Khalid Hattaf,[1,2] and Noura Yousfi[1]

[1] *Department of Mathematics and Computer Science, Faculty of Sciences Ben M'sik, Hassan II University, P.O. Box 7955, Sidi Othman, Casablanca, Morocco*
[2] *Centre Régional des Métiers de l'Education et de la Formation, 20340 Derb Ghalef, Casablanca, Morocco*

Correspondence should be addressed to Khalid Hattaf; k.hattaf@yahoo.fr

Received 30 August 2013; Revised 6 December 2013; Accepted 20 December 2013; Published 10 February 2014

Academic Editor: William E. Fitzgibbon

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 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

$$\begin{aligned}\frac{dS}{dt} &= \Lambda - \mu S - \frac{\beta SI}{1+\alpha_1 S+\alpha_2 I+\alpha_3 SI},\\ \frac{dI}{dt} &= \frac{\beta SI}{1+\alpha_1 S+\alpha_2 I+\alpha_3 SI} - (\mu + d + r)\, I, \qquad (1)\\ \frac{dR}{dt} &= rI - \mu R,\end{aligned}$$

where S, I, and R are susceptible, infectious, and recovered classes, respectively. Λ is the recruitment rate of the population, μ is the natural death rate of the population, d is the death rate due to disease, r is the recovery rate of the infective individuals, β 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:

$$\begin{aligned}\frac{\partial S}{\partial t} &= d_S\Delta S + \Lambda - \mu S(x,t) \\ &\quad - \frac{\beta S(x,t) I(x,t)}{1+\alpha_1 S(x,t)+\alpha_2 I(x,t)+\alpha_3 S(x,t) I(x,t)}, \\ \frac{\partial I}{\partial t} &= d_I\Delta I + \frac{\beta S(x,t) I(x,t)}{1+\alpha_1 S(x,t)+\alpha_2 I(x,t)+\alpha_3 S(x,t) I(x,t)} \\ &\quad - (\mu + d + r) I(x,t), \\ \frac{\partial R}{\partial t} &= d_R\Delta R + rI(x,t) - \mu R(x,t),\end{aligned} \tag{2}$$

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

$$\begin{aligned}\frac{\partial S}{\partial t} &= d_S\Delta S + \Lambda - \mu S(x,t) \\ &\quad - \frac{\beta S(x,t) I(x,t)}{1+\alpha_1 S(x,t)+\alpha_2 I(x,t)+\alpha_3 S(x,t) I(x,t)}, \\ \frac{\partial I}{\partial t} &= d_I\Delta I + \frac{\beta S(x,t) I(x,t)}{1+\alpha_1 S(x,t)+\alpha_2 I(x,t)+\alpha_3 S(x,t) I(x,t)} \\ &\quad - (\mu + d + r) I(x,t),\end{aligned} \tag{3}$$

with homogeneous Neumann boundary conditions

$$\frac{\partial S}{\partial \nu} = \frac{\partial I}{\partial \nu} = 0, \quad \text{on } \partial\Omega \times (0,+\infty), \tag{4}$$

and initial conditions

$$S(x,0) = \phi_1(x) \geq 0, \quad I(x,0) = \phi_2(x) \geq 0, \quad x \in \overline{\Omega}. \tag{5}$$

Here, Ω is a bounded domain in $\mathbb{R}^n$ with smooth boundary $\partial\Omega$. $\partial S/\partial\nu$ and $\partial I/\partial\nu$ 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 $X = C(\overline{\Omega}) \times C(\overline{\Omega})$ of the form

$$\begin{aligned}u'(t) &= Au(t) + F(u(t)), \quad t > 0, \\ u(0) &= u_0 \in X,\end{aligned} \tag{6}$$

where $u = \mathrm{col}(S, I)$, $u_0 = \mathrm{col}(\phi_1, \phi_2)$, $Au(t) = \mathrm{col}(d_S\Delta S, d_I\Delta I)$, and

$$F(u(t)) = \begin{pmatrix} \Lambda - \mu S - \dfrac{\beta SI}{1+\alpha_1 S+\alpha_2 I+\alpha_3 SI} \\ \dfrac{\beta SI}{1+\alpha_1 S+\alpha_2 I+\alpha_3 SI} - (\mu + d + r) I \end{pmatrix}. \tag{7}$$

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

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

$$\begin{aligned}\frac{\partial S}{\partial t} - d_S\Delta S &= F_1(S,I), \\ \frac{\partial I}{\partial t} - d_I\Delta I &= F_2(S,I).\end{aligned} \tag{8}$$

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

$$\begin{aligned}\frac{\partial S}{\partial t} - d_S\Delta S &\leq \Lambda - \mu S, \\ \frac{\partial S}{\partial \nu} &= 0,\end{aligned} \tag{9}$$

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

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

$$\frac{dS_1}{dt} = \Lambda - \mu S_1, \qquad S_1(0) = \|\phi_1\|_\infty. \tag{10}$$

Since $S_1(t) \le \max\{\Lambda/\mu, \|\phi_1\|_\infty\}$ for $t \in [0, \infty)$, we have that

$$S(x,t) \le \max\left\{\frac{\Lambda}{\mu}, \|\phi_1\|_\infty\right\}, \quad \forall (x,t) \in \overline{\Omega} \times [0, T_{\max}). \tag{11}$$

From Theorem 2 given by Alikakos in [12], to establish the L^∞ 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 $(\partial/\partial t)(S+I) - \Delta(d_S S + d_I I) \le \Lambda - \mu(S+I)$, we get

$$\frac{\partial}{\partial t}\left(\int_\Omega (S+I)\,dx\right) \le \operatorname{mes}(\Omega)\,\Lambda - \mu\left(\int_\Omega (S+I)\,dx\right). \tag{12}$$

Hence,

$$\int_\Omega (S+I)\,dx \le \operatorname{mes}(\Omega) \max\left\{\frac{\Lambda}{\mu}, \|\phi_1 + \phi_2\|_\infty\right\}, \tag{13}$$

which implies that $\sup_{t\ge 0}\int_\Omega I(x,t)dx \le K := \operatorname{mes}(\Omega)\max\{\Lambda/\mu, \|\phi_1 + \phi_2\|_\infty\}$. Using [12, Theorem 3.1], we deduce that there exists a positive constant K^* that depends on K and on $\|\phi_1 + \phi_2\|_\infty$ such that

$$\sup_{t\ge 0}\|I(\cdot,t)\|_\infty \le K^*. \tag{14}$$

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

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

$$R_0 = \frac{\beta\Lambda}{(\mu + \alpha_1\Lambda)(\mu + d + r)}, \tag{15}$$

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

$$S^* = \frac{2(a + \alpha_2\Lambda)}{\beta - \alpha_1 a + \alpha_2\mu - \alpha_3\Lambda + \sqrt{\delta}}, \qquad I^* = \frac{\Lambda - \mu S^*}{a}, \tag{16}$$

with $a = \mu + d + r$ and $\delta = (\beta - \alpha_1 a + \alpha_2\mu - \alpha_3\Lambda)^2 + 4\alpha_3\mu(a + \alpha_2\Lambda)$.

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 $\overline{E}(\overline{S}, \overline{I})$ for small space- and time-dependent fluctuations and expand them in Fourier space. For this, let

$$S(\vec{x},t) \sim \overline{S}e^{\lambda t}e^{i\vec{k}\cdot\vec{x}}, \qquad I(\vec{x},t) \sim \overline{I}e^{\lambda t}e^{i\vec{k}\cdot\vec{x}}, \tag{17}$$

where $\vec{x} = (x, y)$ and $\vec{k}\cdot\vec{k} := \langle\vec{k},\vec{k}\rangle := k^2$; $\vec{k}$ and λ are the wavenumber vector and frequency, respectively. Then we can obtain the corresponding characteristic equation as follows:

$$\det\left(J - k^2 D - \lambda I_2\right) = 0, \tag{18}$$

where I_2 is the identity matrix, $D = \operatorname{diag}(d_S, d_I)$ is the diffusion matrix, and J is the Jacobian matrix of (3) without diffusion ($d_S = d_I = 0$) at $\overline{E}$ which is given by

$$J = \begin{pmatrix} -\mu - \dfrac{\beta\overline{I}(1+\alpha_2\overline{I})}{(1+\alpha_1\overline{S}+\alpha_2\overline{I}+\alpha_3\overline{S}\overline{I})^2} & -\dfrac{\beta\overline{S}(1+\alpha_1\overline{S})}{(1+\alpha_1\overline{S}+\alpha_2\overline{I}+\alpha_3\overline{S}\overline{I})^2} \\ \dfrac{\beta\overline{I}(1+\alpha_2\overline{I})}{(1+\alpha_1\overline{S}+\alpha_2\overline{I}+\alpha_3\overline{S}\overline{I})^2} & \dfrac{\beta\overline{S}(1+\alpha_1\overline{S})}{(1+\alpha_1\overline{S}+\alpha_2\overline{I}+\alpha_3\overline{S}\overline{I})^2} - a \end{pmatrix}. \tag{19}$$

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

$$\left(\mu + k^2 d_S + \lambda\right)\left(a(R_0 - 1) - k^2 d_I - \lambda\right) = 0. \tag{20}$$

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 $\lambda_2(k)$ 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. □

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 $E^*(S^*, I^*)$, we have

$$\lambda^2 + a_1(k)\lambda + a_2(k) = 0, \tag{21}$$

where

$$a_1(k) = \mu + k^2(d_S + d_I) + a\left(\frac{I^*(1+\alpha_2 I^*)}{(1+\alpha_1 S^* + \alpha_2 I^* + \alpha_3 S^* I^*)S^*} + \frac{\alpha_2 I^* + \alpha_3 S^* I^*}{1 + \alpha_1 S^* + \alpha_2 I^* + \alpha_3 S^* I^*}\right),$$

$$a_2(k) = \left(\mu + \frac{\beta I^*\left(1+\alpha_2 I^*\right)}{\left(1+\alpha_1 S^*+\alpha_2 I^*+\alpha_3 S^* I^*\right)^2} + k^2 d_S\right) \times \left(k^2 d_I + \frac{\beta S^*\left(\alpha_2 I^*+\alpha_3 S^* I^*\right)}{\left(1+\alpha_1 S^*+\alpha_2 I^*+\alpha_3 S^* I^*\right)^2}\right) + \frac{\beta^2 S^* I^*\left(1+\alpha_2 I^*\right)\left(1+\alpha_1 S^*\right)}{\left(1+\alpha_1 S^*+\alpha_2 I^*+\alpha_3 S^* I^*\right)^4}. \quad (22)$$

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 $f(S,I) = \beta S/(1+\alpha_1 S+\alpha_2 I+\alpha_3 SI)$. To study the global stability of E_f for (1), the authors Hattaf et al. [7] proposed the following Lyapunov functional:

$$V_1 = S - S_0 - \int_{S_0}^{S} \frac{f(S_0,0)}{f(X,0)}dX + I, \quad (23)$$

where $S_0 = \Lambda/\mu$.

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

$$W_1 = \int_\Omega V_1\left(S(x,t), I(x,t)\right)dx. \quad (24)$$

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

$$\frac{dW_1}{dt} = \int_\Omega \left\{-\frac{\mu(S-S_0)^2}{S\left(1+\alpha_1 S_0\right)} + aI\left(\frac{1+\alpha_1 S}{1+\alpha_1 S+\alpha_2 I+\alpha_3 SI}R_0 - 1\right)\right\}dx - \frac{d_S S_0}{1+\alpha_1 S_0}\int_\Omega \frac{|\nabla S|^2}{S^2}dx \le \int_\Omega \left\{-\frac{\mu(S-S_0)^2}{S\left(1+\alpha_1 S_0\right)} + aI\left(R_0-1\right)\right\}dx - \frac{d_S S_0}{1+\alpha_1 S_0}\int_\Omega \frac{|\nabla S|^2}{S^2}dx. \quad (25)$$

Since $R_0 \le 1$, we have $dW_1/dt \le 0$. 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.

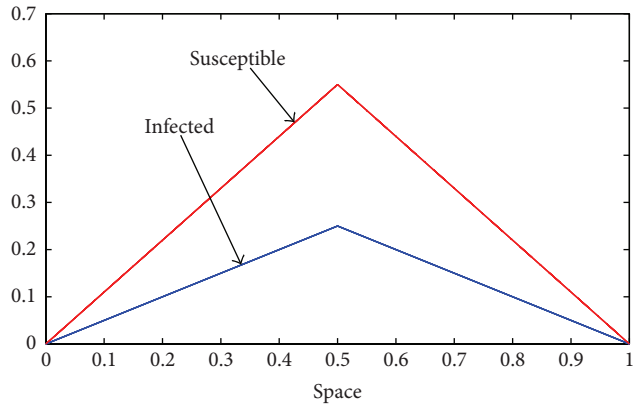

Figure 1: The initial distributions of the numbers of susceptibles and infectious individuals for Figures 2 and 3.

(i) If $R_0 < 1$, then $I = 0$.

(ii) If $R_0 = 1$, from $S = S_0$ and the first equation of (3), we have

$$\frac{\partial S}{\partial t} = \frac{\partial S_0}{\partial t} = d_S \Delta S_0 + \Lambda - \mu S_0 - f\left(S_0, I\right) I = 0. \quad (26)$$

Then, $f(S_0,I)I = (\beta S_0 I)/(1+\alpha_1 S_0+\alpha_2 I+\alpha_3 S_0 I) = 0$. Since $\beta > 0$ and $S_0 > 0$, then $I = 0$.

By the above discussion, we deduce that the largest compact invariant set in $\Gamma = \{(S,I) \mid (dW_1)/dt = 0\}$ is just the singleton 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 $R_0 \le 1$, 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

$$\frac{\partial S}{\partial \nu} = \frac{\partial I}{\partial \nu} = 0, \quad t > 0, \quad x = 0, 1, \quad (27)$$

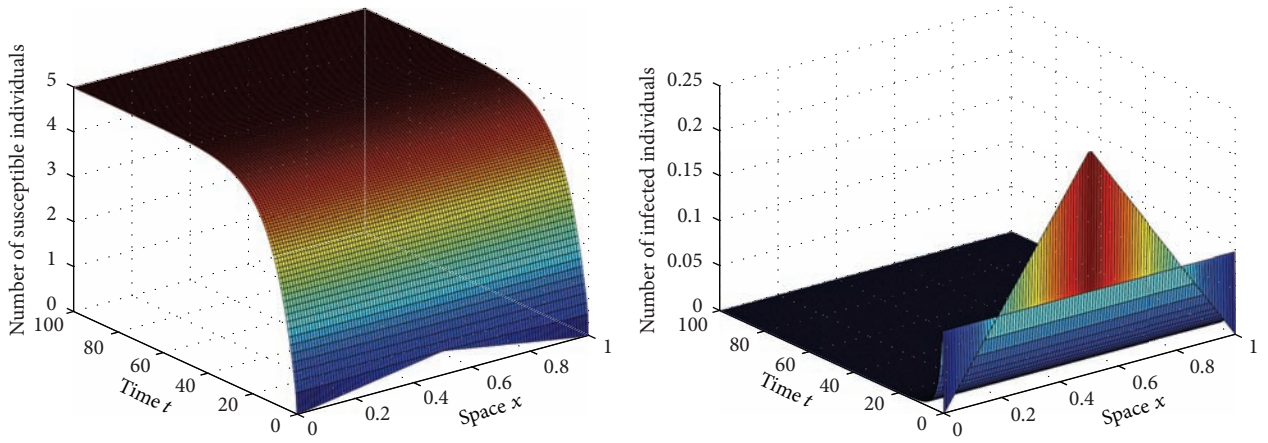

Figure 2: The temporal solution found by numerical of problem (3) with the Neumann boundary conditions (27) and initial conditions (28).

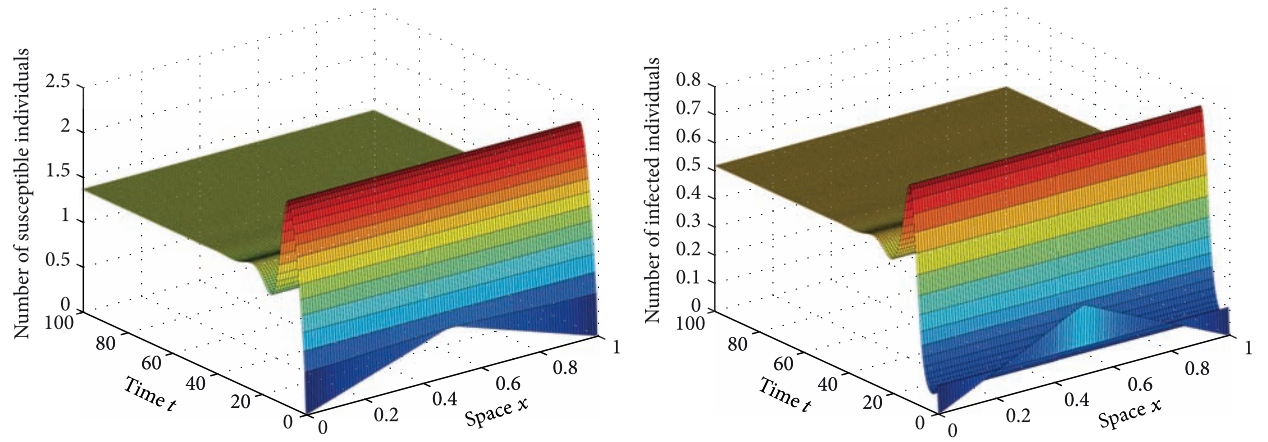

Figure 3: The temporal solution found by numerical of problem (3) with the Neumann boundary conditions (27) and initial conditions (28).

and initial conditions

$$S(x,0) = \begin{cases} 1.1x, & 0 \le x < 0.5, \\ 1.1(1-x), & 0.5 \le x \le 1, \end{cases}$$
$$I(x,0) = \begin{cases} 0.5x, & 0 \le x < 0.5, \\ 0.5(1-x), & 0.5 \le x \le 1. \end{cases} \quad (28)$$

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 $R_0 \le 1$,, 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.

Acknowledgments

The authors would like to thank the editor and the anonymous referees for their valuable remarks and comments which have led to the improvement of the quality of their study.

References

[1] J. R. Beddington, "Mutual interference between parasites or predators and its effect on searching efficiency," *Journal of Animal Ecology*, pp. 331–341, 1975.

[2] D. L. DeAngelis, R. A. Goldsten, and R. Neill, "A model for trophic interaction," *Ecology*, vol. 56, pp. 881–892, 1975.

[3] R. S. Cantrell and C. Cosner, "On the Dynamics of Predator-Prey Models with the Beddington-Deangelis Functional Response," *Journal of Mathematical Analysis and Applications*, vol. 257, no. 1, pp. 206–222, 2001.

[4] P. H. Crowley and E. K. Martin, "Functional responses and interference within and between year classes of a dragonfly population," *Journal of the North American Benthological Society*, vol. 8, pp. 211–221, 1989.

[5] X. Zhou and J. Cui, "Global stability of the viral dynamics with crowley-martin functional response," *Bulletin of the Korean Mathematical Society*, vol. 48, no. 3, pp. 555–574, 2011.

[6] X. Q. Liu, S. M. Zhong, B. D. Tian, and F. X. Zheng, "Asymptotic properties of a stochastic predator-prey model with Crowley-Martin functional response," *Journal of Applied Mathematics and Computing*, vol. 43, no. 1-2, pp. 479–490, 2013.

[7] K. Hattaf, A. A. Lashari, Y. Louartassi, and N. Yousfi, "A delayed SIR epidemic model with general incidence rate," *Electronic Journal of Qualitative Theory of Differential Equations*, vol. 3, pp. 1–9, 2013.

[8] V. Capasso, *Mathematical Structures of Epidemic Systems*, Lecture Notes in Biomathematics, Springer, Germany, 1993.

[9] A. Pazy, *Semigroups of Linear Operators and Applications to Partial Differential Equations*, vol. 44 of *Applied Mathematical Sciences*, Springer, New York, NY, USA, 1983.

[10] J. Smoller, *Shock Waves and Reaction-Diffusion Equations*, Springer, Berlin, Germany, 1983.

[11] M. H. Protter and H. F. Weinberger, *Maximum Principles in Differential Equations,*, Prentice Hall, Englewood Cliffs, NJ, USA, 1967.

[12] N. D. Alikakos, "An application of the invariance principle to reaction-diffusion equations," *Journal of Differential Equations*, vol. 33, no. 2, pp. 201–225, 1979.

[13] D. Henry, *Geometric Theory of Semilinear Parabolic Equations*, vol. 840 of *Lecture Notes in Mathematics*, Springer, Berlin, Germany, 1993.

[14] K. Hattaf and N. Yousfi, "Global stability for reaction-diffusion equations in biology," *Computers and Mathematics with Applications*, vol. 66, pp. 1488–1497, 2013.

[15] J. P. LaSalle, *The Stability of Dynamical Systems*, Regional Conference Series in Applied Mathematics, SIAM, Philadelphia, Pa, USA, 1976.