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Global analysis of an epidemic model with nonmonotone incidence rate

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Abstract

In this paper we study an epidemic model with nonmonotonic incidence rate, which describes the psychological effect of certain serious diseases on the community when the number of infectives is getting larger. By carrying out a global analysis of the model and studying the stability of the disease-free equilibrium and the endemic equilibrium, we show that either the number of infective individuals tends to zero as time evolves or the disease persists.

Keywords: Epidemic; Nonlinear incidence; Psychological effect; Global stability

1. Introduction

Let $S(t)$ be the number of susceptible individuals, $I(t)$ be the number of infective individuals, and $R(t)$ be the number of removed individuals at time t, respectively. After studying the cholera

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epidemic spread in Bari in 1973, Capasso and Serio [2] introduced a saturated incidence rate $g(I)S$ into epidemic models, where $g(I)$ tends to a saturation level when I gets large, i.e.,

$$g(I) = \frac{kI}{1+\alpha I}, \tag{1.1}$$

where kI measures the infection force of the disease and $1/(1+\alpha I)$ measures the inhibition effect from the behavioral change of the susceptible individuals when their number increases or from the crowding effect of the infective individuals. This incidence rate seems more reasonable than the bilinear incidence rate

$$g(I)S = kIS, \tag{1.2}$$

because it includes the behavioral change and crowding effect of the infective individuals and prevents the unboundedness of the contact rate by choosing suitable parameters. Ruan and Wang [12] studied an epidemic model with a specific nonlinear incident rate

$$g(I)S = \frac{kI^2S}{1+\alpha I^2} \tag{1.3}$$

and presented a detailed qualitative and bifurcation analysis of the model. They derived sufficient conditions to ensure that the system has none, one, or two limit cycles and showed that the system undergoes a Bogdanov–Takens bifurcation at the degenerate equilibrium which includes a saddle-node bifurcation, a Hopf bifurcation, and a homoclinic bifurcation. The general incidence rate

$$g(I)S = \frac{kI^pS}{1+\alpha I^q} \tag{1.4}$$

was proposed by Liu et al. [10] and used by a number of authors, see, for example, Derrick and van den Driessche [3], Hethcote [5], Hethcote and Levin [6] and van den Driessche [7], Alexander and Moghadas [1], etc. Nonlinear incidence rates of the form kI^pS^q were investigated by Liu et al. [9,10].

If the function $g(I)$ is nonmonotone, that is, $g(I)$ is increasing when I is small and decreasing when I is large (see Fig. 1), it can be used to interpret the "psychological" effect: for a very large number of infective individuals the infection force may decrease as the number of infective individuals increases, because in the presence of large number of infectives the population may tend to reduce the number of contacts per unit time. The recent epidemic outbreak of severe acute respiratory syndrome (SARS) had such psychological effects on the general public (see [8]), aggressive measures and policies, such as border screening, mask wearing, quarantine, isolation, etc. have

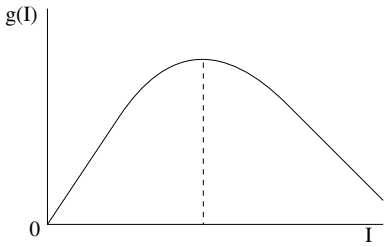

Fig. 1. Nonmonotone incidence function $g(I)$.

been proved to be very effective ([4,13]) in reducing the infective rate at the late stage of the SARS outbreak, even when the number of infective individuals were getting relatively larger. To model this phenomenon, we propose a incidence rate

$$g(I)S = \frac{kIS}{1+\alpha I^2}, \tag{1.5}$$

where kI measures the infection force of the disease and $1/(1+\alpha I^2)$ describes the psychological or inhibitory effect from the behavioral change of the susceptible individuals when the number of infective individuals is very large. This is important because the number of effective contacts between infective individuals and susceptible individuals decreases at high infective levels due to the quarantine of infective individuals or due to the protection measures by the susceptible individuals. Notice that when $\alpha = 0$, the nonmonotone incidence rate (1.5) becomes the bilinear incidence rate (1.2).

The organization of this paper is as follows. In the next section, we present the model and derive the disease-free equilibrium and the endemic equilibrium. In Section 3 we carry out a qualitative analysis of the model. Stability conditions for the disease-free equilibrium and the endemic equilibrium are derived, respectively. A brief discussion and some numerical simulations are given in Section 4.

2. The model

The model to be studied takes the following form

$$\begin{aligned}
\frac{dS}{dt} &= b - dS - \frac{kSI}{1+\alpha I^2} + \gamma R,\\
\frac{dI}{dt} &= \frac{kSI}{1+\alpha I^2} - (d+\mu)I,\\
\frac{dR}{dt} &= \mu I - (d+\gamma)R,
\end{aligned} \tag{2.1}$$

where $S(t)$, $I(t)$ and $R(t)$ denote the numbers of susceptible, infective, and recovered individuals at time t, respectively. b is the recruitment rate of the population, d is the natural death rate of the population, k is the proportionality constant, μ is the natural recovery rate of the infective individuals, γ is the rate at which recovered individuals lose immunity and return to the susceptible class, α is the parameter measures the psychological or inhibitory effect.

Because of the biological meaning of the components $(S(t), I(t), R(t))$, we focus on the model in the first octant of R^3. We first consider the existence of equilibria of system (2.1). For any values of parameters, model (2.1) always has a disease-free equilibrium $E_0 = (b/d, 0, 0)$. To find the positive equilibria, set

$$\begin{aligned}
&b - dS - \frac{kIS}{1+\alpha I^2} + \gamma R = 0,\\
&\frac{kS}{1+\alpha I^2} - (d+\mu) = 0,\\
&\mu I - (d+\gamma)R = 0.
\end{aligned}$$

This yields

$$\alpha d(d+\mu)I^2 + k\left(d+\mu-\frac{\gamma\mu}{d+\gamma}\right)I + d(d+\mu) - kb = 0. \tag{2.2}$$

Define the basic reproduction number as follows

$$R_0 = \frac{kb}{d(d+\mu)}. \tag{2.3}$$

From Eq. (2.2) we can see that

(i) if $R_0 \leqslant 1$, then there is no positive equilibrium;
(ii) if $R_0 > 1$, then there is a unique positive equilibrium $E^* = (S^*, I^*, R^*)$, called the *endemic equilibrium* and given by

$$S^* = \frac{1}{d}\left[b - (d+\mu-\frac{\gamma\mu}{d+\gamma})I^*\right], \tag{2.4}$$

$$I^* = \frac{-k(d+\mu-\frac{\gamma\mu}{d+\gamma}) + \sqrt{\Delta}}{2\alpha d(d+\mu)}, \tag{2.5}$$

$$R^* = \frac{\mu}{d+\gamma}I^*, \tag{2.6}$$

where

$$\Delta = k^2\left(d+\mu-\frac{\gamma\mu}{d+\gamma}\right)^2 - 4\alpha d^2(d+\mu)^2[1-R_0].$$

In the next section, we shall study the property of these equilibria and perform a global qualitative analysis of model (2.1).

3. Mathematical analysis

To study the dynamics of model (2.1), we first present a lemma.

Lemma 3.1. *The plane $S + I + R = b/d$ is an invariant manifold of system (2.1), which is attracting in the first octant.*

Proof. Summing up the three equations in (2.1) and denoting $N(t) = S(t) + I(t) + R(t)$, we have

$$\frac{\mathrm{d}N}{\mathrm{d}t} = b - \mathrm{d}N. \tag{3.1}$$

It is clear that $N(t) = b/d$ is a solution of Eq. (3.1) and for any $N(t_0) \geqslant 0$, the general solution of Eq. (3.1) is

$$N(t) = \frac{1}{d}[b - (b - \mathrm{d}N(t_0))\mathrm{e}^{-d(t-t_0)}].$$

Thus,

$$\lim_{t\to\infty} N(t) = \frac{b}{d},$$

which implies the conclusion. □

It is clear that the limit set of system (2.1) is on the plane $S + I + R = b/d$. Thus, we focus on the reduced system

$$\begin{aligned}\frac{dI}{dt} &= \frac{kI}{1+\alpha I^2}\left(\frac{b}{d} - I - R\right) - (d+\mu) \triangleq P(I,R),\\ \frac{dR}{dt} &= \mu I - (d+\gamma) \triangleq Q(I,R).\end{aligned} \tag{3.2}$$

We have the following result regarding the nonexistence of periodic orbits in system (3.2), which implies the nonexistence of periodic orbits of system (2.1) by Lemma 3.1

Theorem 3.2. *System (3.2) does not have nontrivial periodic orbits.*

Proof. Consider system (3.2) for $I > 0$ and $R > 0$. Take a Dulac function

$$D(I,R) = \frac{1+\alpha I^2}{kI}.$$

We have

$$\frac{\partial(DP)}{\partial I} + \frac{\partial(DQ)}{\partial R} = -1 - \frac{2\alpha(d+\mu)}{k} I - \frac{1+\alpha I^2}{kI}(d+\gamma) < 0.$$

The conclusion follows. □

In order to study the properties of the disease-free equilibrium E_0 and the endemic equilibrium E^*, we rescale (3.2) by

$$x = \frac{k}{d+\gamma} I,\ y = \frac{k}{d+\gamma} R,\ \tau = (d+\gamma)t.$$

Then we obtain

$$\begin{aligned}\frac{dx}{d\tau} &= \frac{x}{1+px^2}(A - x - y) - mx,\\ \frac{dy}{d\tau} &= qx - y,\end{aligned} \tag{3.3}$$

where

$$p = \frac{\alpha(d+\gamma)^2}{k^2},\quad A = \frac{bk}{d(d+\gamma)},\quad m = \frac{d+\mu}{d+\gamma},\quad q = \frac{\mu}{d+\gamma}.$$

Note that the trivial equilibrium $(0,0)$ of system (3.3) is the disease-free equilibrium E_0 of model (2.1) and the unique positive equilibrium (x^*, y^*) of system (3.3) is the endemic equilibrium E^* of model (2.1) if and only if $m - A < 0$, where

$$x^* = \frac{-(1+q) + \sqrt{(1+q)^2 - 4mp(m-A)}}{2mp}, \quad y^* = qx^*.$$

We first determine the stability and topological type of $(0,0)$. The Jacobian matrix of system (3.3) at $(0,0)$ is

$$M_0 = \begin{bmatrix} A-m & 0 \\ q & -1 \end{bmatrix}.$$

If $A - m = 0$, then there exists a small neighborhood N_0 of $(0,0)$ such that the dynamics of system (3.3) are equivalent to that of

$$\begin{aligned} \frac{dx}{d\tau} &= -x^2 - 2xy + O((x,y)^3), \\ \frac{dy}{d\tau} &= qx - y. \end{aligned} \tag{3.4}$$

By Theorem 7.1 of Zhang et al. [14] (pp. 114) or Theorem 2.11.1 of Perko [11] (pp. 150), we know that $(0,0)$ is a saddle-node. Hence, we obtain the following result.

Theorem 3.3. *The disease-free equilibrium* $(0,0)$ *of system* (3.3) *is*

(i) *a stable hyperbolic node if* $m - A > 0$;
(ii) *a saddle-node if* $m - A = 0$;
(iii) *a hyperbolic saddle if* $m - A < 0$.

When $m - A < 0$, we discuss the stability and topological type of the endemic equilibrium (x^*, y^*). The Jacobian matrix of (3.3) at (x^*, y^*) is

$$M_1 = \begin{bmatrix} \dfrac{x^*(px^{*2} + 2pqx^{*2} - 2Apx^* - 1)}{(1+px^{*2})^2} & \dfrac{-x^*}{1+px^{*2}} \\ q & -1 \end{bmatrix}.$$

We have that

$$\begin{aligned} \det(M_1) &= -\frac{x^*(px^{*2} + 2pqx^{*2} - 2Apx^* - 1)}{(1+px^{*2})^2} + \frac{qx^*}{1+px^{*2}} \\ &= \frac{x^*(1 + q + 2Apx^* - (1+q)px^{*2})}{(1+px^{*2})^2}. \end{aligned}$$

The sign of $\det(M_1)$ is determined by

$$S_1 \triangleq 1 + q + 2Apx^* - (1+q)px^{*2}.$$

Note that $mpx^{*2} + (1+q)x^* + m - A = 0$. We have

$$\begin{aligned} mS_1 =& (2Amp + (1+q)^2)x^* + (1+q)(2m - A) \\ =& (2Amp + (1+q)^2)\left[x^* + \frac{(1+q)(2m-A)}{2Amp + (1+q)^2}\right]. \end{aligned}$$

Substituting

$$x^* = \frac{-(1+q) + \Delta_1}{2mp}, \text{ where } \Delta_1 = \sqrt{(1+q)^2 - 4mp(m-A)},$$

into S_1 and using a straightforward calculation, we have

$$\begin{aligned} S_1 =& -\frac{\Delta_1}{m}[(1+q)\Delta_1 - (2Amp + (1+q)^2)] \\ =& \frac{(1+q)\Delta_1}{m}\left[\left(1 + q + \frac{2mpA}{1+q}\right) - \Delta_1\right]. \end{aligned}$$

Since

$$\left(1 + q + \frac{2mpA}{1+q}\right)^2 - \Delta_1^2 = \frac{4m^2p^2A^2}{(1+q)^2} + 4m^2p > 0,$$

it follows that $S_1 > 0$. Hence, $\det(M_1) > 0$ and (x^*, y^*) is a node or a focus or a center. Furthermore, we have the following result on the stability of (x^*, y^*).

Theorem 3.4. *Suppose $m - A < 0$, then there is a unique endemic equilibrium (x^*, y^*) of model (3.3), which is a stable node.*

Proof. We know that the stability of (x^*, y^*) is determined by $\mathrm{tr}(M_1)$. We have

$$\mathrm{tr}(M_1) = \frac{-p^2x^{*4} + (1+2q)px^{*3} - 2(1+A)px^{*2} - x^* - 1}{(1 + px^{*2})^2}.$$

The sign of $\mathrm{tr}(M_1)$ is determined by

$$S_2 = -p^2x^{*4} + (1+2q)px^{*3} - 2(1+A)px^{*2} - x^* - 1.$$

We claim that $S_2 \neq 0$. To see this, note that $mpx^{*2} + (1+q)x^* + m - A = 0$. Then we have

$$m^3pS_2 = (B_1A + B_2)x^* + (B_3A + B_4),$$

where

$$\begin{aligned} B_1 &= mp(2 + 3m + 2q + 4mq), \\ B_2 &= (1+q)[(1+q)^2 + m(1+q)(1+2q) - 2m^3p], \\ B_3 &= -(1+q)^2 - m(1+q)(1+2q) + 2m^3p, \\ B_4 &= m[(1+q)^2 + m(1+q)(1+2q) - p(1+2m)A^2]. \end{aligned}$$

When $m - A < 0$, we can see that $B_1A + B_2 > 0$.

Let $\xi = mpx^{*2} + (1+q)x^* + m - A$. Similarly, we have

$$(B_1A + B_2)^2\xi = m^3pPS_2 + S_3,$$

where P is a polynomial of x^* and

$$S_3 = m^3p(1 + A^2p + 2q + q^2)[(A + 2Am - 2m^2)^2p + (1 + A - m + q)(1 + m + q + 2mq)].$$

Assume that $S_2 = 0$. Since $\xi = 0$, it follows that $S_3 = 0$. However, when $m - A < 0$, we have $S_3 > 0$. Therefore, $S_2 \neq 0$ for any positive value of the parameters p, q and A, that is, $\mathrm{tr}(M_1) \neq 0$. Thus, $m - A < 0$ implies that (x^*, y^*) does not change stability. Take $m = 1$, $A = 2$, $p = 1$, $q = 1$. Then $x^* = -1 + \sqrt{2}$, $y^* = -1 + \sqrt{2}$, $\mathrm{tr}(M_1) = -1.64645 < 0$. By the continuity of $\mathrm{tr}(M_1)$ on the parameters, we know that $\mathrm{tr}(M_1) < 0$ for $m - A < 0$. This completes the proof. □

Summarizing Theorems 3.2–3.4, we have the following results on the dynamics of the original model (2.1).

Theorem 3.5. *Let R_0 be defined by (2.3).*

(i) *If $R_0 < 1$, then model (2.1) has a unique disease-free equilibrium $E_0 = (b/d, 0, 0)$, which is a global attractor in the first octant.*
(ii) *If $R_0 = 1$, then model (2.1) has a unique disease-free equilibrium $E_0 = (b/d, 0, 0)$, which attracts all orbits in the interior of the first octant.*
(iii) *If $R_0 > 1$, then model (2.1) has two equilibria, a disease-free equilibrium $E_0 = (b/d, 0, 0)$ and an endemic equilibrium $E^* = (S^*, I^*, R^*)$. The endemic equilibrium E^* is a global attractor in the interior of the first octant.*

4. Discussions

Several nonlinear incidence rates have been proposed by researchers, see, for example, Capasso and Serio [2], Liu et al. [10], Derrick and van den Driessche [3], Hethcote and van den Driessche [7], etc. Complex dynamics have been observed in epidemiological models with nonlinear incidence rate, such as the existence of multiple equilibria and limit cycles, various types of bifurcations including Hopf, saddle-node, homoclinic and Bagdanov–Takens bifurcations, etc., see Ruan and Wang [12] and references cited therein.

In this paper we proposed a nonmonotone and nonlinear incidence rate of the form $kIS/(1 + \alpha I^2)$, which is increasing when I is small and decreasing when I is large. It can be used to interpret the "psychological" effect: the number of effective contacts between infective individuals and susceptible individuals decreases at high infective levels due to the quarantine of infective individuals or the protection measures by the susceptible individuals. The recent epidemic outbreak of severe acute respiratory syndrome (SARS) had such psychological effects on the general public (see Leung et al. [8], Gumel et al. [4] and Wang and Ruan [13]).

We have carried out a global qualitative analysis of an SIR model with this nonmonotone and nonlinear incidence rate and studied the existence and stability of the disease-free and endemic equilibria. Interestingly, this model does not exhibit complicated dynamics as other epidemic models with other types of incidence rates reported in Liu et al. [10], Derrick and van den Driessche [3], Hethcote and Levin [6], Hethcote and van den Driessche [7], Ruan and Wang [12], etc. In

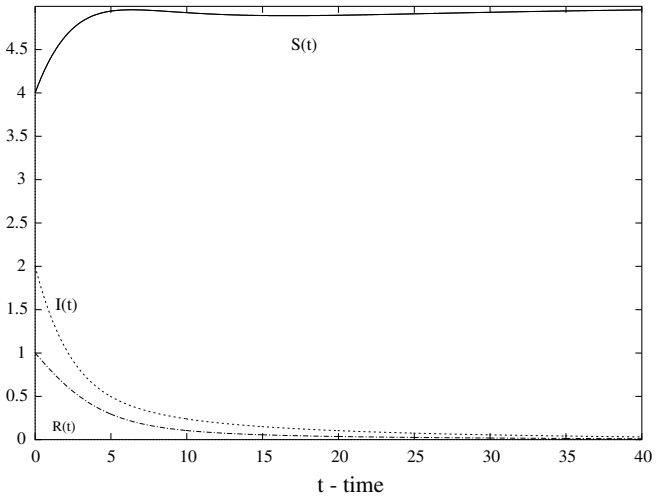

Fig. 2. When $b = 1.0$, $d = 0.2$, $k = 0.2$, $\alpha = 4.0$, $\gamma = 0.3$, $\mu = 0.15$, $R_0 = 6/7 < 1$, $S(t)$ approaches to its steady state value while $I(t)$ and $R(t)$ approach zero as time goes to infinity, the disease dies out.

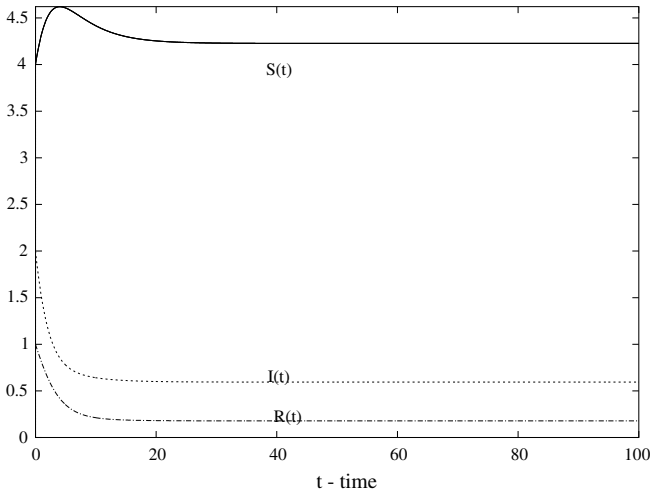

Fig. 3. When $b = 1.0$, $d = 0.2$, $k = 0.2$, $\alpha = 4.0$, $\gamma = 0.3$, $\mu = 0.15$, $R_0 = 20/7 > 1$, all three components, $S(t)$, $I(t)$ and $R(t)$, approach to their steady state values as time goes to infinity, the disease becomes endemic.

terms of the basic reproduction number $R_0 = kb/(d(d + \mu))$, our main results indicate that when $R_0 < 1$, the disease-free equilibrium is globally attractive (see Fig. 2). When $R_0 > 1$, the endemic equilibrium exists and is globally stable (see Fig. 3). Biologically, these indicate that when the proportionality (infection) constant (k) and/or the recruitment rate (b) is sufficiently large and removal rate (death rate (d) plus recovery rate (μ)) is sufficiently small such that $R_0 > 1$, then the disease persists. On the other hand, if the proportionality (infection) constant (k) and/or the recruitment rate (b) is small enough and removal rate (death rate (d) plus recovery rate (μ)) is large enough such that $R_0 < 1$, then the disease dies out. The aggressive control measures and policies, such

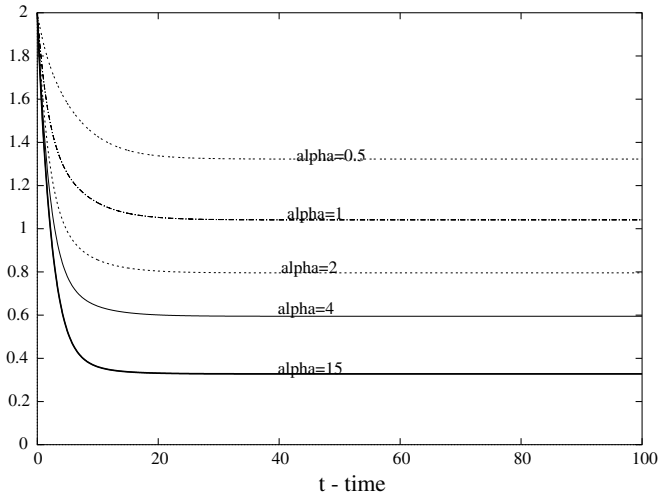

Fig. 4. The dependence of I^* on the parameter α.

as border screening, mask wearing, quarantine, isolation, etc., helped in reducing the infection rate and increasing the removal rate and in the eventual eradication of SARS (Gumel et al. [4] and Wang and Ruan [13]).

Recall that the parameter α describes the psychological effect of the general public toward the infectives. Though the basic reproduction number R_0 does not depend on α explicitly, numerical simulations indicate that when the disease is endemic, the steady state value I^* of the infectives decreases as α increases (see Fig. 4). From the steady state expression (2.5) we can see that I^* approaches zero as α tends to infinity.

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