QUALITATIVE ANALYSIS OF AN EPIDEMIC MODEL

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ABSTRACT. In this paper we consider an epidemic model with a complete form of the non-monotonic incidence rate of [18]. This non-monotonic incidence rate describes the effects of psychological factor, protection measures and intervention policies when a serious disease arouses widespread horror and the number of infective is getting larger. By carrying out the qualitative analysis of the model, we show that when a basic reproduction number is less than one, there exist some values of parameters such that the model has a local stable disease free equilibrium and a local stable endemic equilibrium at the same time, and there also exist other values of parameters such that the model has a global stable disease-free equilibrium. These results reveal rich dynamics in the model.

1 Introduction In the studying of disease transmission models, people often assume that the population can be broken into homogeneous subpopulation denoted by $S, I$ and $R$, respectively such that individuals in a given subpopulation are indistinguishable from one another. $S(t)$ represent the number of individuals who are susceptible to the disease, that is, who are not yet infected at time $t$. $I(t)$ represent the number of infected individuals who infectious and able to spread the disease by contact with susceptible individuals. And $R(t)$ represent the number of individuals who have been infected and then removed from the possibility of being infected again or of spreading at time $t$. A disease transmission model suited to homogeneous subpopulation can be modelled by a system of ordinary differential equations, which describes the evolution of the number of individuals in each subpopulation. For

The first author’s research was supported by Program for New Century Excellent Talents in University, and National Natural Science Foundation of China (No. 10231020).

Keywords: Epidemic, nonmonotonic incidence rate, psychological effect, bistable.

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example, a SIRS epidemic model can be written by
\[
\frac{dS}{dt} = b - dS - g(I)S + \delta R,
\]
(1.1)
\[
\frac{dI}{dt} = g(I)S - (d + \mu)I,
\]
\[
\frac{dR}{dt} = \mu I - (d + \delta)R,
\]
where \(b\) is the recruitment rate of the population, \(d\) is the natural death rate of the population, \(\mu\) is the natural recovery rate of the infective individuals, \(\delta\) is the rate at which recovered individuals lose immunity and return to the susceptible class, and \(g(I)\) is the infection force.

In [10] Kermack and McKendrick assume that the infection force is a linear function of \(I\), or equivalently, that the number of contacts in unit time per infective is proportional to total population size. In [2], according to the data of the cholera epidemic spread in Bari in 1973 Capasso and Serio proposed that the infection force is saturated incidence, the number of contacts in unit time per infective is an increasing but bounded function. The function \(g(I)\) has the following form
\[
g(I) = \frac{kI}{1 + \alpha I},
\]
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. \(g(I)\) tends to a saturation level when \(I\) gets large.

In [13] Liu, Levin and Iwasa proposed a general infection force function
\[
g(I) = \frac{kI^p}{1 + \alpha I^q},
\]
which is used by a number of authors, see, for example, Derrick and van den Driessche in [4], Hethcote and van den Driessche in [9], Alexander and Moghadas in [1], Ruan and Wang in [15], etc.

To model the effects of psychological factor, protection measures and intervention policies when a serious disease arouses widespread horror, Xiao and Ruan in [18] consider a specific infection force
\[
g(I) = \frac{kI}{1 + \alpha I^2}.
\]
$g(I)$ is nonmonotone. This implies that the contact rate and the infection probability are increasing when a new infectious disease emerges and people may ignore the disease (or people has little knowledge about the disease), however, when $I$ is large and some people died of this disease, psychological factor leads people to modify their behavior for reducing the probability of infection, for instance, during the outbreak of epidemic of severe acute respiratory syndrome (SARS) in 2003, people went out with mask wearing and few people went to restaurants. And protection measures and intervention policies, such as quarantining suspected of being infected, postponing conferences, closed recreation ground, etc., were performed. So the infection force decreases at the high infection level. In [18] Xiao and Ruan presented the global analysis of an epidemic model with the nonmonotonic infection force and showed that the model admit threshold dynamics, i.e., the model has a threshold parameter called the basic reproduction number (cf. [5]). More precisely, the disease free equilibrium is globally stable as the basic reproduction number is less than one, and the disease cannot invade the population, but when the basic reproduction number is greater than one, an endemic equilibrium is globally stable and the disease persists as time evolves.

In this paper, we consider a complete form of the non-monotonic infection force

$$g(I) = \frac{kI}{1 + \beta I + \alpha I^2},$$

where $\beta$ is a parameter such that $1 + \beta I + \alpha I^2 > 0$ for all $I \geq 0$, hence, $\beta > -2\sqrt{\alpha}$. When $\beta = 0$, the non-monotonic infection force is considered in [18]. After carrying out a global qualitative analysis, we obtain that the model with the complete form of the non-monotonic infection force has complicated dynamical behaviors. A bistable case, pointed out by Capasso and Wilson in [3], occurs for the model, i.e., when a basic reproduction number is less than one, there exist some values of parameters such that the model has a local stable disease free equilibrium and a local stable endemic equilibrium at the same time, and there also exist other values of parameters such that the model has a global stable disease free equilibrium. These results reveal rich dynamics in the model.

This paper is organized as follows. In Section 2, we consider an epidemic model with a complete form of non-monotonic incidence rate and present qualitative analysis of the model. In Section 3, we give numerical analysis of the model for some parameter values, and show that a bistable occurs, a periodic oscillation appears, etc. A brief discussion is
given in the last section.

2 Qualitative analysis of an epidemic model

We consider an epidemic model with the following form

\[ \begin{align*}
\frac{dS}{dt} &= b - dS - \frac{kSI}{1 + \beta I + \alpha I^2} + \delta R, \\
\frac{dI}{dt} &= \frac{kSI}{1 + \beta I + \alpha I^2} - (d + \mu)I, \\
\frac{dR}{dt} &= \mu I - (d + \delta)R,
\end{align*} \tag{2.1} \]

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, \( \mu \) is the natural recovery rate of the infective individuals, \( \delta \) is the rate at which recovered individuals lose immunity and return to the susceptible class, \( \alpha \) is a positive parameter, \( \beta \) is a parameter such that \( 1 + \beta I + \alpha I^2 > 0 \) for all \( I \geq 0 \), hence, \( \beta > -2\sqrt{\alpha} \).

From the standpoint of biology, we are interested in the dynamics of system (2.1) in the first octant of \( \mathbb{R}^3 \). Before going to details of dynamics, we first present the following lemma for system (2.1).

**Lemma 2.1.** The plane \( S + I + R = b/d \) is an invariant manifold of system (2.1), which is attracting in the first octant of \( \mathbb{R}^3 \).

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

\[ \frac{dN}{dt} = b - dN. \tag{2.2} \]

It is clear that \( N(t) = b/d \) is a solution of equation (2.2) and for any \( N(t_0) \geq 0 \), the general solution of equation (2.2) is

\[ N(t) = \frac{1}{d} \left[ b - (b - dN(t_0))e^{-d(t-t_0)} \right]. \]

Thus,

\[ \lim_{t \to -\infty} N(t) = \frac{b}{d}, \]

which implies the conclusion. \qed
This lemma implies that the limit set of system (2.1) in the first octant of \( \mathbb{R}^3 \) locates on the plane \( S + I + R = \frac{b}{d} \). Therefore, the dynamics of system (2.1) in the first octant of \( \mathbb{R}^3 \) is equivalent to the following system

\[
\begin{align*}
\frac{dI}{dt} &= \frac{kI}{1 + \beta I + \alpha I^2} \left( \frac{b}{d} - I - R \right) - (d + \mu)I, \\
\frac{dR}{dt} &= \mu I - (d + \delta)R,
\end{align*}
\]

in the first quadrant \( \mathbb{R}_+^2 \) of \( \mathbb{R}^2 \). System (2.1) has the disease free equilibrium or the endemic equilibria if and only if system (2.3) has equilibrium \((0, 0)\) or the positive equilibria, respectively. For simplicity, we re-scale (2.3) by

\[
\begin{align*}
x &= \frac{k}{d + \delta} I, & y &= \frac{k}{d + \delta} R, & \tau &= (d + \delta)t.
\end{align*}
\]

Then we obtain

\[
\begin{align*}
\frac{dx}{d\tau} &= \frac{x}{1 + mx + nx^2} (A - x - y) - px \triangleq P(x, y), \\
\frac{dy}{d\tau} &= qx - y \triangleq Q(x, y),
\end{align*}
\]

where

\[
\begin{align*}
m &= \frac{\beta(d + \delta)}{k}, & n &= \frac{\alpha(d + \delta)^2}{k^2}, & A &= \frac{bk}{d(d + \delta)}, \\
p &= \frac{d + \mu}{d + \delta}, & q &= \frac{\mu}{d + \delta}.
\end{align*}
\]

We can see that \( A, p, q \) and \( n \) are positive parameters, and \( m > -2\sqrt{\frac{\alpha}{\alpha(d + \delta)/k}} = -2\sqrt{n} \).

It is clear that system (2.4) has the trivial equilibrium \((0, 0)\) for all parameters. To find the positive equilibria of system (2.4), we set

\[
\begin{align*}
\frac{x}{1 + mx + nx^2} (A - x - y) - px &= 0, \\
qx - y &= 0,
\end{align*}
\]

which yields

\[
pmx^2 + (1 + q + pm)x + p - A = 0.
\]
Let
\[ \Delta = (1 + q + pm)^2 - 4pn(p - A) = -\frac{4\alpha(d + \mu)(d^2 + \mu d - bk)}{dk^2} + \left(1 + \frac{\mu}{d + \delta} + \frac{\beta(d + \mu)}{k}\right)^2. \]

By analyzing the existence of positive roots to (2.5), we obtain the following lemma.

**Lemma 2.2.**

(i) **System (2.4) has a unique equilibrium (0, 0) if and only if one of the following conditions holds:**

(i.1) \( \Delta < 0; \)

(i.2) \( \Delta \geq 0, \ A - p \leq 0 \) and \( 1 + q + pm \geq 0. \)

(ii) **System (2.4) has two equilibria, (0, 0) and a unique positive equilibrium \( E^*(x^*, y^*) \) if and only if one of the following conditions holds:**

(ii.1) \( A - p > 0. \) In this case, \( x^* = \frac{-(1 + q + pm) + \sqrt{\Delta}}{2pn}, \)

\( y^* = qx^*. \)

(ii.2) \( A - p = 0 \) and \( 1 + q + pm < 0. \) In this case, \( x^* = \frac{1 + q + pm}{pm}, \)

\( y^* = qx^*. \)

(ii.3) \( \Delta = 0 \) and \( 1 + q + pm < 0. \) In this case, \( x^* = \frac{1 + q + pm}{2pn}, \)

\( y^* = qx^*. \)

(iii) **System (2.4) has three equilibria, (0, 0) and two positive equilibria \( E_1(x_1, y_1) \) and \( E_2(x_2, y_2) \) if and only if \( \Delta > 0, \ A - p < 0 \) and \( 1 + q + pm < 0. \) In this case,

\[ x_1 = \frac{-(1 + q + pm) - \sqrt{\Delta}}{2pn}, \quad y_1 = qx_1; \]

\[ x_2 = \frac{-(1 + q + pm) + \sqrt{\Delta}}{2pn}, \quad y_2 = qx_2. \]

Now we study the local stability of equilibria of system (2.4). We first study the disease-free equilibrium (0, 0). The Jacobian matrix of system (2.4) at (0, 0) is

\[ M_0 = \begin{bmatrix} A - p & 0 \\ q & -1 \end{bmatrix}. \]
The two eigenvalues of $M_0$ are $\lambda_1 = -1$ and $\lambda_2 = A - p$. When $A - p \neq 0$, equilibrium $(0, 0)$ of system (2.4) is hyperbolic, and equilibrium $(0, 0)$ is a stable node (or a saddle) if $A - p < 0$ ($A - p > 0$, respectively).

Following the method of van den Driessche and Watmough in [3], we can find that the basic reproduction number

$$R_0 = \frac{A}{p} = \frac{bk}{d(d + \mu)}.$$

If $A - p = 0$, i.e., $R_0 = 1$, then equilibrium $(0, 0)$ of system (2.4) is nonhyperbolic. From qualitative analysis and Theorem 7.1 (p. 131) in [19], we obtain that equilibrium $(0, 0)$ is a saddle-node or a stable degenerate node as $1 + q + Am \neq 0$ or $1 + q + Am = 0$, respectively.

Summarizing the above analysis, we obtain the following theorem.

**Theorem 2.3.** The disease free equilibrium $(0, 0)$ of system (2.4) is

(i) a stable hyperbolic node if $R_0 < 1$ (i.e., $\frac{bk}{d(d + \mu)} < 1$);

(ii) a saddle-node if $R_0 = 1$ and $1 + q + Am \neq 0$, i.e., $1 + \frac{b\beta}{d} + \frac{\mu}{d + \delta} \neq 0$; a degenerate stable node if $R_0 = 1$ and $1 + q + Am = 0$, i.e.,

$$1 + \frac{b\beta}{d} + \frac{\mu}{d + \delta} = 0;

(iii) a hyperbolic saddle if $R_0 > 1$, i.e., $\frac{bk}{d(d + \mu)} > 1$.

From Theorem 2.3 we can see that when the basic reproduction number $R_0 = 1$, the ability for an infectious disease to invade a population is related to the quantity $1 + b\beta/d + \mu/(d + \delta)$ and the initial number of $(I, R)$. If both $R_0 = 1$ and $1 + b\beta/d + \mu/(d + \delta) = 0$, then the disease free equilibrium is locally asymptotically stable and the disease may not invade the population. If $R_0 = 1$ and $1 + b\beta/d + \mu/(d + \delta) \neq 0$, then the disease free equilibrium is a saddle-node and the disease can invade the population when the initial number of $(I, R)$ is suitable. To discuss when the disease cannot invade the population, we have to study the global stability of equilibrium $(0, 0)$. We first give conditions that system (2.4) has not nontrivial periodic orbits in $\mathbb{R}^2_+$. 

**Lemma 2.4.** If $m \geq \zeta$, then system (2.4) does not have nontrivial
periodic orbits in the interior of $\mathbb{R}^2_+$, where

$$
\zeta = \max \left\{ \frac{-1}{1+p}, \frac{-2\sqrt{\alpha (d+\delta)}}{k} \right\} 
$$

$$
= \max \left\{ -\frac{d+\delta}{2d+\delta+\mu}, \frac{-2\sqrt{\alpha (d+\delta)}}{k} \right\}.
$$

Proof. We consider system (2.4) in the interior of $\mathbb{R}^2_+$. Taking a Dulac function

$$
D(x, y) = \frac{1 + mx + nx^2}{x},
$$

we have

$$
\frac{\partial (DP)}{\partial x} + \frac{\partial (DQ)}{\partial y} = -1 - m(p + 1) - \frac{1}{x} - nx(2p + 1) < 0,
$$
since $m \geq \zeta$. The conclusion follows.

Remark 2.1. If $\beta \geq 0$, then $m \geq 0$. By means of Lemma 2.4, we know that when $\beta > 0$ system (2.4) does not have nontrivial periodic orbits in the interior of $\mathbb{R}^2_+$. Summing up the above lemmas and theorems, we have

Theorem 2.5. The disease free equilibrium $(b/d, 0, 0)$ of (2.1) is globally asymptotical stable in the interior of $\mathbb{R}^3_+$, and the disease cannot invade the population if one of the following conditions holds.

1. $m \geq \zeta$ and $\Delta < 0$;
2. $m \geq \zeta$, $\Delta \geq 0$ and $R_0 < 1$;
3. $m \geq \zeta$ and $R_0 = 1$.

Proof. Lemma 2.1 implies that the stability of the disease free equilibrium $(b/d, 0, 0)$ of (2.1) in $\mathbb{R}^3_+$ is equivalent to that of equilibrium $(0, 0)$ of (2.4) in $\mathbb{R}^2_+$. Thus, we only discuss the stability of equilibrium $(0, 0)$ of (2.4) in $\mathbb{R}^2_+$.

1. From Lemma 2.2, we know that system (2.4) has a unique equilibrium $(0, 0)$ in $\mathbb{R}^2_+$ if $\Delta < 0$. It is clear that $R_0 < 1$ if $\Delta < 0$. According to the term (i) of Theorem 2.3, $(0, 0)$ is a stable hyperbolic node. Thus, when $m \geq \zeta$, $(0, 0)$ is a global attractor in $\mathbb{R}^2_+$ since system (2.4) has not any nontrivial periodic orbits by Lemma 2.4. Hence, condition (1)
guarantees that \((b/d, 0, 0)\) is globally asymptotical stable in the interior of \(\mathbb{R}^3_+\).

(2) When \(m \geq \zeta\), we have that \(1 + q + pm \geq q + 1/(1 + p) > 0\). From the term (i.2) of Lemma 2.2, we know that system (2.4) has a unique equilibrium \((0, 0, 0)\) if \(m \geq \zeta\), \(\Delta \geq 0\) and \(R_0 < 1\). Therefore, we obtain the conclusion (2) by the same arguments in proof of (1).

(3) Since \(R_0 = 1\), \(A = p\) and \(\Delta \geq 0\). Let \(X = x\), \(Y = y - qx\), \(\tau = -t\), and rename \(X, Y, \tau\) as \(x, y, t\), respectively. Then system (2.4) can be written as

\[
\frac{dx}{dt} = (1 + pm + q)x^2 + xy + O((x, y)^3),
\]

\[
\frac{dy}{dt} = y - q(1 + pm + q)x^2 - qxy + O((x, y)^3).
\]

Note that \(1 + q + pm \geq q + 1/(1 + p) > 0\) since \(m \geq \zeta\). Therefore, there exists a small neighborhood \(N_0\) of \((0, 0)\) such that system (2.6) on a center manifold of \((0, 0)\) becomes

\[
\frac{dx}{dt} = (1 + pm + q)x^2 + q(1 + pm + q)x^3
\]

\[
+ q^2(1 + pm + q)x^4 + O(x^5).
\]

From Theorem 7.1 (p. 131) in [19], we obtain that \((0, 0)\) of system (2.6) is a saddle-node, which has a stable parabolic sector in \(N_0\) if \(1 + pm + q > 0\).

On the other hand, system (2.4) has a unique equilibrium \((0, 0)\) by the term (i.2) of Lemma 2.2 when \(m \geq \zeta\) and \(R_0 = 1\). And system (2.4) has not any nontrivial periodic orbits by Lemma 2.4. Hence, the equilibrium \((0, 0)\) attracts all orbits of system (2.4) in the interior of \(\mathbb{R}^2_+\). We finish the proof.

**Remark 2.2.** By means of Remark 2.1 and Theorem 2.3, we obtain that if \(\beta \geq 0\) and \(R_0 < 1\), then the disease free equilibrium \((b/d, 0, 0)\) of (2.1) is globally asymptotical stable in the interior of \(\mathbb{R}^3_+\).

Next we analyze when invasion of the disease is possible for model (2.1). We first give the topological type of the positive equilibria of system (2.4) as follows.
Lemma 2.6.

(i) When system (2.4) has a unique positive equilibrium \( E^*(x^*, y^*) \), then \( E^*(x^*, y^*) \) is:

(i.1) a node or a focus or a center if \( R_0 > 1 \) or both \( R_0 = 1 \) and \( 1 + q + pm < 0 \);

(i.2) a degenerate equilibrium if both \( \Delta = 0 \) and \( 1 + q + pm < 0 \).

(ii) When system (2.4) has two positive equilibria \( E_1(x_1, y_1) \) and \( E_2(x_2, y_2) \), then \( E_1(x_1, y_1) \) is a hyperbolic saddle, and \( E_2(x_2, y_2) \) is a node, or a focus or a center.

Proof. Let Jacobian matrix of system (2.4) at equilibrium \((x, y)\) be \( M_1 \),

\[
M_1 = \begin{bmatrix}
  x(nx^2 + 2nqx^2 + mqx^2 - 2nAx - Am - 1) & -x \\
  (1 + mx + nx^2)^2 & 1 + mx + nx^2 \\
  q & 1 \\
  1 & -1
\end{bmatrix}.
\]

Then the determinant of \( M_1 \) is

\[
\det(M_1) = \frac{x(1 + q + Am + 2nAx - (1 + q)nx^2)}{(1 + mx + nx^2)^2}.
\]

Its sign is determined by

\[
S_1 \defeq 1 + q + Am + 2nAx - (1 + q)nx^2.
\]

And the trace of \( M_1 \) is

\[
\text{tr}(M_1) = ( - n^2x^4 + n(1 + 2q - 2m)x^3 + (mq - 2nA - m^2 - 2n)x^2 \\
- (Am + 2m + 1)x - 1) / (1 + mx + nx^2)^2,
\]

the sign of which is determined by

\[
S_2 \defeq -n^2x^4 + n(1 + 2q - 2m)x^3 + (mq - 2nA - m^2 - 2n)x^2 \\
- (Am + 2m + 1)x - 1.
\]

Note that \( pmx^2 + (1 + q + pm)x + p - A = 0 \). Thus, we have

\[
pS_1 = [2nAp + (1 + q)(1 + q + mp)]x + Apm + (1 + q)(2p - A),
\]

\[
m^3pS_2 = (B_1A + B_2)x + (B_3A + B_4),
\]


where

\[ B_1 = np(2 + 3p + 2q + 4pq + mp^2), \]
\[ B_2 = (1 + q + mp)((1 + q + mp)^2 - 2np^3 + p(1 + 2q - 2m)(1 + q + mp) + p^2(m^2 - mq)) + 2mnp^4, \]
\[ B_3 = -(1 + q + mp)^2 - p(1 + q + mp)(1 + 2q - 2m) - p^2(m^2 - mq) + 2np^3, \]
\[ B_4 = p((1 + q + mp)^2 + p(1 + q + mp)(1 + 2q - 2m) - n(1 + 2p)A^2 + p^2(m^2 - mq)]. \]

Now we divide into three cases to prove the lemma. (i) When \( R_0 > 1 \), system (2.4) has a unique positive equilibrium \( E^*(x^*, y^*) \), where

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

Substituting the value of \( x^* \) into \( x^* \) of \( pS_1 \) and \( m^3pS_2 \), we obtain

\[ pS_1 = \frac{\Delta_1(1 + q)\left[\left(pm + q + 1 + \frac{2nAp}{1 + q}\right) - \Delta_1\right]}{2mp}, \]
\[ m^3pS_2 = (B_1A + B_2)x^* + (B_3A + B_4), \]

where \( \Delta_1 = \sqrt{(1 + q + pm)^2 - 4pn(p - A)}. \)

Let \( K_1 = pm + q + 1 + \frac{2nAp}{1 + q}. \)

Then \( K_1 > 0 \) by \( R_0 > 1 \) and \(-2\sqrt{n} < m \). In fact,

\[ K_1 = \frac{(1 + q)^2 + pm(1 + q) + 2nAp}{1 + q}. \]

If \( m \geq 0 \), it is obvious that \( K_1 > 0 \). If \(-2\sqrt{n} < m < 0 \), then we consider the following equation of \( y \)

\[ y^2 + pmy + 2nAp = 0. \]
Note that $-2\sqrt{m} < m < 0$ and $p < A$ since $R_0 > 1$. We have
\[
(pm)^2 - 8nAp = p(pm^2 - 8nA) < p(4np - 8nA) = 4np(p - 2A) < 0.
\]
Therefore, $y^2 + pmy + 2nAp > 0$ for all real number $y$. Taking $y = p + 1$, we have $(1 + q)^2 + pm(1 + q) + 2nAp > 0$, which implies that $K_1 > 0$.

Computing
\[
K_1^2 - \Delta_1^2 = 4np^2 \frac{A^2n + Am(1 + q) + (1 + q)^2}{(1 + q)^2},
\]
we have $K_1^2 - \Delta_1^2 > 0$ since $A^2n + Am(1 + q) + (1 + q)^2 > 0$, which can be proved by the similar arguments of $K_1 > 0$. Thus $K_1 - \Delta_1 > 0$, it follows that $S_1 > 0$. Hence, det($M_1$) > 0, which implies that $(x^*, y^*)$ is a non-degenerate node or a focus or a center. Furthermore, it is stable (unstable) if $S_2 < 0$ ($S_2 > 0$, respectively). By numerical computation, we can see that there exist some values of parameters such that $S_2 > 0$ (for example, taking $m = -6$, $n = 20$, $p = 4$, $q = 24$, $A = 25$).

When $\Delta > 0$, $R_0 = 1$ and $1 + q + pm < 0$, system (2.4) has a unique positive equilibrium $E^*(x^*, y^*)$, where $x^* = -(1 + q + pm)/pn$, $y^* = qx^*$. From $1 + q + pm < 0$ we obtain that $-2\sqrt{m} < m < 0$.

Substituting $x^* = -(1 + q + pm)/pn$ into $x$ of $pS_1$, we have
\[
pS_1 = -(1 + q + pm)\frac{nAp + (1 + q)(1 + q + pm)}{pn}.
\]

Note that $nAp + (1 + q)(1 + q + pm) = np^2 + m(1 + q)p + (1 + q)^2$, and
\[
[m(1 + q)]^2 - 4n(1 + q)^2 = (1 + q)^2(m^2 - 4n) < 0.
\]

Thus, $nAp + (1 + q)(1 + q + pm) > 0$, which implies that $S_1 > 0$. Hence, det($M_1$) > 0, which leads to the conclusion.

(ii) When $\Delta = 0$ and $1 + q + pm < 0$, system (2.4) has a unique positive equilibrium $E^*(x^*, y^*)$, where $x^* = -(1 + q + pm)/2pn$, $y^* = qx^*$. Note that $pmx^2 + (1 + q + pm)x^* + p - A = 0$. Simplifying $x^*S_1$, we have
\[
x^*S_1 = -\frac{[2A + 2p + (1 + q + mp)x^*][A - (1 + q)x^*]}{p}.
\]
Substituting $x^* = -(1 + q + pm)/2pn$ into the above formula, we obtain
\[
x^*S_1 = -\frac{[2nAp + (1 + q)(1 + q + mp)][4np(p - A) - (1 + q + mp)^2]}{4n^2p^3} = 0.
\]
Thus, \( \det(M_1) = 0 \), which implies that \((x^*, y^*)\) is a degenerate equilibrium. In next lemma, we will give the topological type of the degenerate equilibrium.

(iii) When \( \Delta > 0 \) and \( R_0 < 1, 1 + q + pm < 0 \), system (2.4) has two positive equilibria \( E_1(x_1, y_1) \) and \( E_2(x_2, y_2) \), where

\[
x_1 = -\frac{(1 + q + pm) - \sqrt{(1 + q + pm)^2 - 4pn(p - A)}}{2pn}, \quad y_1 = qx_1;
\]
\[
x_2 = -\frac{(1 + q + pm) + \sqrt{(1 + q + pm)^2 - 4pn(p - A)}}{2pn}, \quad y_2 = qx_2.
\]

Substituting the values of \( x_1 \) and \( x_2 \) into \( x \) of \( pS_1 \), respectively, we have

\[
pS_1(x_1) = -\frac{\Delta_1(1 + q) \left[ (pm + q + 1 + \frac{2nAp}{1+q}) + \Delta_1 \right]}{2pn} = -\frac{\Delta_1(1 + q)(K_1 + \Delta_1)}{2pn},
\]
\[
pS_1(x_2) = \frac{\Delta_1(1 + q) \left[ (pm + q + 1 + \frac{2nAp}{1+q}) - \Delta_1 \right]}{2pn} = \frac{\Delta_1(1 + q)(K_1 - \Delta_1)}{2pn}.
\]

Let us consider

\[
S_1(x_1)S_1(x_2) = -\frac{\Delta_1^2(1 + q)^2(K_1^2 - \Delta_1^2)}{4p^4n^2}.
\]

Since \( K_1^2 - \Delta_1^2 > 0 \), \( S_1(x_1^*)S_1(x_2^*) < 0 \). Therefore, one of \( E_1 \) and \( E_2 \) is hyperbolic saddle, the other is a nonsaddle elementary equilibrium. The proof of lemma is completed.

We can see that \( R_0 < 1 \) if \( \Delta = 0 \). When \( \Delta = 0 \) and \( 1 + q + pm < 0 \), system (2.4) has only two equilibria \((0, 0)\) and \( E^*(x^*, y^*) \); \((0, 0)\) is a stable hyperbolic node and \( E^*(x^*, y^*) \) is degenerate, whose topological type is as follows.
Lemma 2.7.

(i) Assume that
\[ A = \frac{2p(1 + q + pq)}{1 - mp + q + 2pq} \quad \text{and} \quad n = -\frac{(1 + q + mp)(1 + q + 2pq - mp)}{4p^2}, \]
in which \( p, q \) and \( m \) satisfy that \( p > 0, \ q > 0, \ 1 + q + pm < 0 \) and \( -2\sqrt{n} < m < 0 \). Then the unique positive equilibrium \( E^*(x^*, y^*) \) of system (2.4) is a cusp, where \( x^* = -(1 + q + pm)/2pn, \ y^* = qx^*. \)

(ii) Assume that
\[ A \neq \frac{2p(1 + q + pq)}{1 - mp + q + 2pq} \quad \text{and} \quad n = \frac{(1 + q + mp)^2}{4p(p - A)}, \]
in which \( p, q \) and \( m \) satisfy that \( p > 0, \ q > 0, \ 1 + q + pm < 0 \) and \( -2\sqrt{n} < m < 0 \). Then the unique positive equilibrium \( E^*(x^*, y^*) \) of system (2.4) is a saddle-node, where \( x^* = -(1 + q + pm)/2pn, \ y^* = qx^*. \)

Proof. From Lemma 2.6, we know that the unique equilibrium \( E^*(x^*, y^*) \) of system (2.4) is degenerate if and only if (H1) \( \Delta = 0 \); and (H2) \( 1 + q + pm < 0 \). By (H1) and (H2), we have
\[ x^* = -\frac{1 + q + pm}{2pn}, \quad y^* = qx^*, \]
\[ n = \frac{(1 + q + pm)^2}{4p(p - A)}, \]
\[ -2\sqrt{n} < m < 0. \]

If we further assume that (H3) \( \text{tr}(M_1(x^*)) = 0 \), then
\[ S_2(x^*) = 0. \]

Substituting \( n \) and \( x^* \) in (2.7) into (2.8), we can get
\[ -2p(1 + q) + A(1 - mp + q)\]
\[ \times [2p(1 + q + pq) + A(-1 + mp - q - 2pq)] = 0. \]
Now we claim that $-2p(1 + q) + A(1 - mp + q) \neq 0$, i.e., $A \neq 2p(1 + q)/(1 - mp + q)$. In fact, if $A = 2p(1 + q)/(1 - mp + q)$, then by the second equation of (2.7), we obtain

$$n = -\frac{(1 + q + mp)(1 + q - mp)}{4p^2} = \frac{[m^2p^2 - (1 + q)^2]}{4p^2},$$

which implies that $4n = m^2 - (1 + q)^2/p^2 < m^2$, and it contradicts to the third inequality of (2.7). Therefore, by (2.9) we have

$$(2.10) \quad 2p(1 + q + pq) + A(-1 + mp - q - 2pq) = 0.$$  

Note that $-1 + mp - q - 2pq \neq 0$. Thus,

$$A = \frac{2p(1 + q + pq)}{1 - mp + q + 2pq}.$$  

From the second equation of (2.7), we further obtain that

$$n = -\frac{(1 + q + mp)(1 + q + 2pq - mp)}{4p^2}.$$  

From the above analysis, we can see that the two eigenvalues of $M_1(x^*)$ are all zero if condition (i) in the lemma holds, and only one of the two eigenvalues of $M_1(x^*)$ is zero if condition (ii) in the lemma holds. Using the method in [19], we can obtain the conclusions in lemma. For saving space, we only prove the assertion (i) as follows.

Let $X = x - x^*$, $Y = y - y^*$. And using the Taylor expansion, we can obtain the following system from (2.4) (for simplicity, we still denote $X, Y$ by $x, y$, respectively).

$$\frac{dx}{d\tau} = c_1 x + c_2 y + c_3 x^2 + c_4 xy + P_1(x, y),$$

$$\frac{dy}{d\tau} = qx - y,$$

where $P_1(x, y)$ is a smooth function in $(x, y)$ at least of order three and

$$c_1 = 1, \quad c_2 = -\frac{1}{q},$$

$$c_3 = \frac{(1 + q + 2pq - mp)(2 + p + mp^2 + 2q + 3pq)}{4p^2 q},$$

$$c_4 = -\frac{(1 + q + pq)(1 + q + 2pq - mp)}{2p^2 q^2}.$$
Let \( X = x, Y = x - y/q \), and rename \( X, Y \) as \( x, y \). Then (2.11) is transformed into

\[
\begin{align*}
\frac{dx}{d\tau} &= y + (c_3 + qc_4)x^2 - c_4qxy + P_2(x, y), \\
\frac{dy}{d\tau} &= (c_3 + qc_4)x^2 - c_4qxy + P_2(x, y),
\end{align*}
\]

where \( P_2(x, y) \) is a smooth function in \((x, y)\) at least of order three.

In order to obtain the canonical normal forms, we set \( X = x + (c_4 q/2)x^2, Y = y + (c_3 + qc_4)x^2 \), and rewriting \( X, Y \) as \( x, y \), respectively. Then (2.12) becomes

\[
\begin{align*}
\frac{dx}{d\tau} &= y + P_3(x, y), \\
\frac{dy}{d\tau} &= N_1 x^2 + N_2 xy + P_4(x, y),
\end{align*}
\]

where \( P_3(x, y) \) and \( P_4(x, y) \) are smooth functions in \((x, y)\) at least of order three, and

\[
\begin{align*}
N_1 &= c_3 + qc_4 = \frac{(1 + q + 2pq - mp)(1 + q + mp)}{4pq}, \\
N_2 &= 2c_3 + qc_4 = \frac{(1 + q + 2pq - mp)(1 + p + mp^2 + q + 2pq)}{2p^2q}.
\end{align*}
\]

Note that \( 1 + q + mp < 0 \) and \( 1 + q + 2pq - mp > 0 \). Thus, \( N_1 < 0 \), which implies that \((x^*, y^*)\) is a cusp. The proof is complete.

Lemma 2.7 implies that the disease will not be persistent for some initial number of \((I, R)\) even though an endemic equilibrium exists. On the other hand, the term (i.1) of Lemma 2.6 implies that the disease can invade the population, which leads the disease will be persistent. And the term (i.1) of Lemma 2.6 implies that a bistable case can occur for some values of parameters. More precisely we have

**Theorem 2.8.**

(i) If \( R_0 > 1 \), then system (2.1) has two equilibria, a disease free equilibrium and an endemic equilibrium, and the disease will be persistent for almost all initial number of \((S, I, R)\).
(ii) If $R_0 < 1$, $\Delta = 0$ and $1 + b\beta/d + \mu/(d + \delta) < 0$, then system (2.1) has two equilibria, a disease free equilibrium and an endemic equilibrium, and the disease will die out for almost all initial number of $(S, I, R)$.

(iii) If $R_0 < 1$, $\Delta > 0$ and $1 + b\beta/d + \mu/(d + \delta) < 0$, then system (2.1) has three equilibria, a disease free equilibrium and two endemic equilibria, and there exist parameter values such that both of the disease free equilibrium and an endemic equilibrium are stable.

**Remark 2.3.** It is easy to check that if $\beta \geq 0$ and $R_0 > 1$, then the endemic equilibrium of (2.1) is globally asymptotically stable in the interior of $\mathbb{R}^3_+$ by Lemma 2.4.

From above analysis we know that system (2.1) has rich dynamics as $-2\sqrt{\alpha} < \beta < 0$. Therefore, we will give numerical analysis of the model for $-2\sqrt{\alpha} < \beta < 0$ in the next section.

### 3 Numerical analysis

In this section, we fix some values of parameters of system (2.4) such that some interesting phenomena can be observed.

#### 3.1 $\beta < 0$ and $R_0 > 1$

In this case, we take two sets of parameters values such that system (2.4) has a global asymptotical stable endemic equilibrium, or system (2.4) has a stable limit cycle, respectively.

For example, taking $m = -6$, $n = 20$, $p = 4$, $q = 24$ and $A = 25$, we know that $R_0 = 25 = 4$. System (2.4) has a unique endemic equilibrium, and it is global asymptotically stable in the interior of the first quadrant by Lemma 2.4 (see Figure 3.1).

On the other hand, if we take $m = -3$, $n = 10$, $p = 9$, $q = 47$ and $A = 12$, then system (2.4) becomes

\[
\frac{dx}{d\tau} = \frac{x}{1 - 3x + 10x^2}(12 - x - y) - 9x,
\]
\[
\frac{dy}{d\tau} = 47x - y.
\]

(3.1)

It is easy to compute that $R_0 = 4/3$ and system (3.1) has a unique positive equilibrium $E(x^*, y^*)$, which is a stable weak focus of order one, where $x^* = 1/10$, $y^* = 47/10$. And the weak focus is globally asymptotically stable in the interior of the first quadrant by Lemma 2.4 (see Figure 3.2).
FIGURE 3.1: When $\beta < 0$ and $R_0 > 1$, a global stable endemic equilibrium.

FIGURE 3.2: When $\beta < 0$ and $R_0 > 1$, a global stable weak focus equilibrium.
In the following, we choose \( p \) as a bifurcation parameter such that a stable limit cycle bifurcates from the stable weak focus of order one as \( p \) varies in a small neighborhood of 9. Let \( p = 9 + \epsilon \).

Then we rewrite (3.1) as follows

\[
\begin{align*}
\frac{dx}{dt} &= \frac{x}{1 - 3x + 10x^2} (12 - x - y) - (9 + \epsilon)x, \\
\frac{dy}{dt} &= 47x - y.
\end{align*}
\]

(3.2)

When \( 0 < \epsilon \ll 1 \), system (3.2) has a hyperbolic unstable focus \( E(x_0, y_0) \), where

\[
x_0 = \frac{3\epsilon - 21 + \sqrt{1521 - 366\epsilon - 31\epsilon^2}}{2(90 + 10\epsilon)},
\]

\[
y_0 = 47x_0.
\]

Therefore, there is a supercritical Hopf bifurcation in which a stable limit cycle bifurcates from the equilibrium \( E(x_0, y_0) \) as \( 0 < \epsilon \ll 1 \).

The following Figure 3.3 is the phase portrait of system (3.2) when \( \epsilon = 0.2 \).

3.2 \( \Delta = 0 \) and \( 1 + q + pm < 0 \). In this case, it is clear that \( \beta < 0 \) and system (2.4) has a unique endemic equilibrium, which is degenerate. Taking \((p, A, q, m, n) = (2, 1, 1, -5, 8)\), we have that \( R_0 = 1/2 < 1 \) and system (2.4) has a unique endemic equilibrium \( E(1/4, 1/4) \), which is a cusp of codimension 2 (see Figure 3.4).

Choosing \( A \) and \( p \) as bifurcation parameters, we consider the bifurcation of the cusp of codimension 2 in a neighborhood of \( E(1/4, 1/4) \). Let \( A = 1 + \lambda_1 \), \( p = 2 + \lambda_2 \). Then (2.4) becomes

\[
\begin{align*}
\frac{dx}{d\tau} &= \frac{x}{1 - 5x + 8x^2} (1 + \lambda_1 - x - y) - (2 + \lambda_2)x, \\
\frac{dy}{d\tau} &= x - y,
\end{align*}
\]

(3.3)

where \( |\lambda_1| \ll 1 \) and \( |\lambda_2| \ll 1 \). After a series of transformation, system (3.3) can be transformed into

\[
\begin{align*}
\frac{dx}{dt} &= y, \\
\frac{dy}{dt} &= \eta_1 + \eta_2y + x^2 + xy + w(x, y, \lambda),
\end{align*}
\]

(3.4)
FIGURE 3.3: When $\beta < 0$ and $R_0 > 1$, a unique endemic equilibrium and a stable limit cycle.

FIGURE 3.4: When $\beta < 0$ and $R_0 < 1$, a unique endemic equilibrium and a stable disease free equilibrium.
where \( w(x, y, \lambda) \) is a smooth function of \( x \), \( y \) and \( \lambda \) at least of order three in \( x \) and \( y \), and
\[
\eta_1 = -\frac{4(3 + \lambda_2)4(\lambda_2^3 + 16(\lambda_1 - \lambda_2/4)(\lambda_2 + 4))}{(4 + \lambda_2)^4},
\]
\[
\eta_2 = \frac{2\lambda_2(\lambda_2 + 3)}{4(4 + \lambda_2)^4}.
\]

It is easy to prove that system (3.4) undergoes Bogdanov-Takens bifurcation. Therefore, we have the following local representations of the bifurcation curves in a small neighborhood of the origin of parameters plane.

- The saddle-node bifurcation curve \( SN = \{(\lambda_1, \lambda_2) : \eta_1 = 0, \eta_2 \neq 0\} \);
- The Hopf bifurcation curve \( H = \{(\lambda_1, \lambda_2) : \eta_1 = -\eta_2^2 + O(\eta_2^4), \eta_2 > 0\} \);
- The homoclinic bifurcation curve \( HL = \{(\lambda_1, \lambda_2) : \eta_1 = -49\eta_2^2/25 + O(\eta_2^4), \eta_2 > 0\} \).

When parameters \( (\lambda_1, \lambda_2) \) lies at the Hopf bifurcation curve, system (3.3) has a weak focus of order one, which is unstable. It follows from Hopf bifurcation theorem that there is a subcritical Hopf bifurcation in which an unstable limit cycle bifurcates from the equilibrium as \( (\lambda_1, \lambda_2) \) lies below the Hopf bifurcation curve. And when parameters \( (\lambda_1, \lambda_2) \) lies on the Homoclinic curve, system (3.3) has an unstable homoclinic loop, which connects a hyperbolic saddle.

### 3.3 \( \Delta > 0 \), \( R_0 < 1 \) and \( 1 + q + pm < 0 \)

In this case, system (2.4) has a disease free equilibrium and two endemic equilibria. Let us consider the stability of the disease free equilibrium and one endemic equilibrium. Taking \( p = 1.98, \ A = 1.02, \ q = 1, \ m = -5 \) and \( n = 8 \), system (2.4) has a disease free equilibrium \( O(0,0) \), two endemic equilibria \( E_1(0.20964,0.20964) \) and \( E_2(0.2891,0.2891) \). It is easy to check that \( R_0 = 17/33, \ O(0,0) \) and \( E_2(0.2891,0.2891) \) are local stable, and \( E_1(0.20964,0.20964) \) is a saddle. A bistable case occurs (see Figure 3.5).

### 4 Discussions

In this paper we consider an epidemic model with a complete form of the non-monotonic incidence rate of [18]. Following the definition of a basic reproduction number in [5], we obtain the basic reproduction number of this model
\[
R_0 = \frac{bk}{d(d + \mu)},
\]
which is the same to that of the model in [18]. From the expression of $R_0$, we can see that the basic reproduction number of the two models is independent of parameters $\beta$ or $\alpha$ which measures the psychological or inhibitory effect. By carrying out the qualitative analysis of the model with a complete form of the non-monotonic incidence rate, we have shown that when $\beta \geq 0$ the model has threshold dynamics, i.e. the disease will be extinct if we control the basic reproduction number such that $R_0 < 1$. Conversely, the disease will be persistent if $R_0 > 1$. However, when $0 > \beta - 2\sqrt{\alpha}$, the model has rich dynamical behaviors such as a bistable case, periodic oscillations, etc. In this case, there exist parameter values such that $R_0 < 1$ and both of the disease free equilibrium and the endemic equilibrium are local stable. Hence, the disease will not be extinct yet even though we can control the basic reproduction number such that $R_0 < 1$. The phenomena has also been observed for an epidemic model with nonlinear infection forces in [17].

Let us now consider when $\alpha$ is fixed what $\beta < 0$ implies (see Figure 4.1).

The function $I/(1 + \beta I + \alpha I^2)$ increases more rapidly than the function $I/(1 + \alpha I^2)$ increases in a small neighborhood of $I = 0$. This implies when a new infectious disease emerges, if the infection force of the disease is very strong such that the number of infected individuals
increases rapidly, then it will be difficult to eradicate the disease since the evolution of the disease is depend on the initial number of infected individuals and other parameters $\alpha$, $\beta$ and $\delta$ besides $R_0$. Therefore, The exploration on dynamics of the model indicates that to avoid invasion of the infectious disease, every susceptible individuals should forwardly take protection measures to reduce the number of contacts such that the number of infected individuals increases slowly when a new infectious disease emerges.

Acknowledgements The authors are grateful to the anonymous referees for their valuable comments and suggestions, which led to an improvement of our original manuscript. The authors also thank one referee for pointing us the reference [5].
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