Qualitative and bifurcation analysis using an SIR model with a saturated treatment function

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\textbf{ABSTRACT}

In this paper, we introduce a saturated treatment function into the SIR epidemic model with a bilinear incidence rate and density-dependent demographics, where the treatment function is limited for increasing number of infected individuals. By carrying out global qualitative and bifurcation analysis, it is shown that the system exhibits some new and complicated behaviors: if the basic reproduction number is larger than unity, the number of infected individuals will show persistent behavior, either converging to some positive constant or oscillating; and if the basic reproduction number is below unity, the model may exhibit complicated behaviors including: (i) backward bifurcation; (ii) almost sure disease eradication where the number of infective individuals tends to zero for all initial positions except the interior equilibria; (iii) “oscillating” backward bifurcation where either the number of infective individuals oscillates persistently, if the initial position lies in a region covering the stable endemic equilibrium, or disease eradication, if the initial position lies outside this region; (iv) disease eradication for all initial positions if the basic reproduction number is less than a turning point value. Numerical simulations are presented to illustrate the conclusions.

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1. Introduction

The dynamics of infectious diseases is an important research area in mathematical epidemiology. Understanding the transmission characteristics of infectious diseases in communities, regions and countries can lead to better approaches for decreasing the transmission of these diseases [1]. Recently, ODE models of epidemics have been studied by a number of scholars (see, e.g., [2–4]). The basic and important research subjects for these systems are the existence of the threshold value, which distinguishes whether the infectious disease will die out or not, the local and global stability of the disease-free equilibrium and the endemic equilibrium, the existence of periodic solutions, and the persistence and extinction of the disease. Periodic oscillations have been observed in the incidence of many infectious diseases, including measles, mumps, chickenpox, and influenza (see, e.g., [5]). On the basis of this, there has been a lot of interest in determining how periodic solutions can arise in epidemiological models. For classical SIR epidemic models, a population is divided into three classes: susceptible, infective and recovered. It is common for the basic reproduction number to be a threshold in the sense that a disease is persistent if the basic reproduction number is greater than 1, and dies out if it is below 1. But in recent years, the phenomenon of backward bifurcations has attracted interest in the realm of disease control (see, e.g., [6–9]), and it is found

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that even if the disease-free equilibrium is locally stable, the disease may persist. In this case, the basic reproduction number does not provide a description of the necessary elimination effort; rather the description of the effort is provided through the value of the critical parameter at the turning point. Thus, it is important to identify backward bifurcation in order to obtain thresholds for the control of diseases.

Noting that each country or city has a maximal capacity for the treatment of a disease, Wang [10] introduced a staged treatment function in order to describe the treatment saturated level. The model studied in [10] takes the following form:

\[
\begin{align*}
\dot{S} &= A - dS - \lambda SI, \\
\dot{I} &= \lambda SI - (d + \gamma + \epsilon)I - T(I), \\
\dot{R} &= \gamma I + T(I) - dR,
\end{align*}
\]

(1)

where \(S\), \(I\) and \(R\) denote the numbers of susceptible, infective, and recovered individuals, respectively. \(T(I)\) is the treatment rate function which is proportional to the number of infectives below a fixed constant capacity and is some constant when the number of infectives is greater than the capacity. For system (1), Wang [10] showed that a backward bifurcation occurs if the capacity is small and that there exist bistable endemic equilibria if the capacity is low. A similar model can be found in a later work by Zhang and Liu [4] in which the authors take the saturated treatment function as \(T(I) = \frac{aI}{I + b}\) to show that the medical condition is limited for increasing number of infected individuals. It is shown in [4] that a backward bifurcation will take place when such an effect is strong.

In most of the studies, compartmental models were built by assuming the total population to either be a constant or satisfy exponential growth [1,10,4]. It is more reasonable to assume that the population of a given region obeys logistic growth due to crowding and limited sources. Epidemic models with logistic or generalized logistic demographic structure have been extensively studied (see, e.g., [11–16]). Then the model to be studied takes the following form:

\[
\begin{align*}
\dot{S} &= S(A - S) - kIS, \\
\dot{I} &= kIS - \mu I - \frac{rI}{a + I}, \\
\dot{R} &= \frac{rI}{a + I} - \mu R,
\end{align*}
\]

(2)

where \(S\), \(I\) and \(R\) are defined as in (1). \(A\) is the carrying capacity in the absence of disease, and \(\mu\) is the natural death rate of the population. \(k\) and \(r\) are the infection coefficient and cure rate, respectively. \(a\) is present to measure the extent of the effect of there being a delay in the treatment of infecteds. It is assumed throughout this paper that all the parameters are positive constants.

Before going into any detail, we simplify the model. Since the first two equations of (2) are independent of the third one and its dynamic behavior is trivial when \(I(t_0) = 0\) for some \(t_0 > 0\), it suffices to consider the first two equations with \(I > 0\). Thus, we restrict our attention to the following reduced model:

\[
\begin{align*}
\dot{S} &= S(A - S) - kIS, \\
\dot{I} &= kIS - \mu I - \frac{rI}{a + I}.
\end{align*}
\]

(3)

The organization of this paper is as follows. In the next section, we present preliminary results for our model including the boundedness and existence of equilibria and backward bifurcation. In Section 3, we present a global analysis on disease-free equilibrium. We show the stability of equilibria, and the occurrence of saddle-node bifurcation and supercritical Hopf bifurcation in Section 4. In Section 5, we show that when \(R_0 \geq 1\), the model is persistent in the first quadrant. A brief discussion is given in Section 6.

2. Preliminaries

In this section, we will give some preliminary results from (2), including the boundedness and existence of equilibria and backward bifurcation.

**Lemma 2.1.** System (2) is ultimately bounded, that is, point dissipative.

**Proof.** We shall now show that \(S(t)\), \(I(t)\), \(R(t)\) are bounded on their intervals of existence, which in turn will imply by a standard continuability argument that they are defined on \(0, \infty\). Define \(M_1 = \max\{A, S(0)\}\). Note that any solution of system (2) remains positive when its initial value is positive. Since \(dS(t)/dt \leq S(t)(A - S(t))\), it follows that \(S(t) \leq M_1\) for all \(t\). That is, \(S(t)\) is bounded and consequently defined on \(0, \infty\). Let us consider the Lyapunov function \(U(S(t), I(t), R(t)) = S(t) + I(t) + R(t)\). We now compute the time derivative of \(U(t)\) along the solutions of system (2). One then has

\[
\dot{U}(t) = S(t)(A - S(t)) - \mu I(t) - \mu R(t),
\]

which implies

\[
\dot{U}(t) + \mu U(t) \leq (\mu + A)S(t).
\]
Consequently,
\[ U(S(t), I(t), R(t)) \leq \frac{M_1(\mu + A)}{\mu} + \left( U(S(0), I(0), R(0)) - \frac{M_1(\mu + A)}{\mu} \right) e^{-\mu t}, \]
for all \( t \). This implies that \( I(t), R(t) \) are also bounded and consequently defined on \((0, \infty)\). Finally, we analyze the behavior of solutions which start with initial value \((S, I, R)\) on the boundary of \((0, \infty)^3\).

If \( S_i = 0 \), then \((S(t), I(t), R(t)) \to (0, 0, 0)\) irrespective of the initial values \( I_i, R_i \geq 0 \). If \( S_i > 0 \), then \((S(t), I(t), R(t)) \to (A, 0, 0)\) for \( I_i = R_i = 0 \), while otherwise \((S(t), I(t), R(t))\) enters \((0, \infty)^3\) (and stays there). This completes the proof. \( \square \)

Therefore, the following set is positively invariant for system (2):
\[
\Theta = \left\{ 0 \leq S(t) \leq M_1, 0 \leq S(t) + I(t) + R(t) \leq \frac{M_1(\mu + A)}{\mu}, I(t) \geq 0, R(t) \geq 0 \right\}.
\]
System (3) always admits disease-free equilibria \( E_0(0, 0) \) and \( E_1(A, 0) \). The possible endemic equilibria of (3) are given by
\[
\begin{align*}
S(A - S) - kSI &= 0, \\
kSI - \mu I - \frac{ri}{a + r} &= 0.
\end{align*}
\]
We can define the basic reproduction number as
\[
R_0 = \frac{kA}{\mu + \frac{\mu}{k}},
\]
which is the expected number of new infective individuals produced by a single infective individual in the susceptibles.

In order to obtain the positive solutions of system (4), we solve \( I \) from the first equation of (4), obtaining \( I = \frac{A - S}{k} \). We substitute this into the second equation, which yields
\[
S^2 - \left( ak + A - \frac{\mu}{k} \right) S + \frac{\mu}{k}(ak + A) + r = 0.
\]
Let the discriminant of (5) be \( \Delta = (ak + A - \frac{\mu}{k})^2 - 4r \). Then it is easy to see that
\[
\Delta = \left[ ak + R_0 \left( \frac{\mu}{k} + \frac{r}{ak} \right) - \frac{\mu}{k} \right]^2 - 4r.
\]
Mathematically, \( \Delta = (ak + A - \frac{\mu}{k})^2 - 4r \) is a saddle-node bifurcation surface, where the parameters pass from one side of the surface to the other, describing the number of equilibria of the system changes. Therefore, we have the following simple statements which describe the number and location of equilibria of system (3).
It follows that \( \Delta \geq 0 \) is equivalent to
\[
R_0 \geq 1 - \frac{a^2k^2 + r}{2a\mu + r} + \frac{2ak\sqrt{\Delta}}{2a\mu + r} := p_0,
\]
or
\[
R_0 \leq 1 - \frac{a^2k^2 + r}{2a\mu + r} - \frac{2ak\sqrt{\Delta}}{2a\mu + r}.
\]
Note that \( ak + A - \frac{\mu}{k} > 0 \) is equivalent to
\[
R_0 > -1 - \frac{a^2k^2 - r}{2a\mu + r}.
\]
It follows that \( \Delta \geq 0 \) if and only if (6) holds. Let us suppose that (6) holds. Then (5) has two positive solutions \( S_1 \) and \( S_2 \), where
\[
S_1 = \frac{A + ak + \frac{\mu}{k} + \sqrt{\Delta}}{2}, \quad S_2 = \frac{A + ak + \frac{\mu}{k} - \sqrt{\Delta}}{2},
\]
and \( S_2 < S_1 \). Set \( I_i = (A - S_i)/k \) and \( E_i = (S_i, I_i) \) for \( i = 1, 2 \). Then \( E_i \) is an endemic equilibrium of (3) if \( S_i < A \). Let us consider the conditions under which \( S_i < A \). By the definitions, we see that this is equivalent to
\[
-\sqrt{\Delta} > ak + \frac{\mu}{k} - A.
\]
This implies that
\[ ak + \frac{\mu}{k} - R_0 \left( \frac{\mu}{k} + \frac{r}{ak} \right) < 0. \]

It follows that
\[ R_0 > 1 + \frac{a^2k^2 - r}{au + r} := p_1. \]

Furthermore, (8) implies that
\[ \left( A - ak - \frac{\mu}{k} \right)^2 > \Delta. \]

It follows that (10) is equivalent to
\[ R_0 < 1. \]

Hence, \( S_1 < A \) holds if and only if (9) and (11) are valid. Moreover, if \( R_0 \leq p_1 \) or \( R_0 \geq 1 \), we have \( S_1 \geq A \).

By arguments similar to those above, we see that \( S_2 < A \) if (9) holds or \( 1 < R_0 < p_1 \).

Note that \( ak < \sqrt{r} \) is equivalent to \( p_1 < 1 \). Summarizing the discussion above, we have the following conclusions.

**Theorem 2.1.** The endemic equilibria \( E_1 \) and \( E_2 \) do not exist if \( R_0 < p_0 \). Furthermore, if \( R_0 \geq p_0 \), we have the following conclusions:

(i) If \( ak < \sqrt{r} \), then both \( E_1 = (S_1, I_1) \) and \( E_2 = (S_2, I_2) \) exist when \( p_1 < R_0 < 1 \).

(ii) If \( ak < \sqrt{r} \), then \( E_1 \) does not exist but \( E_2 \) exists if \( R_0 > 1 \).

(iii) Let \( ak \geq \sqrt{r} \). Then \( E_1 \) does not exist. Furthermore, \( E_2 \) exists when \( R_0 > 1 \), and \( E_2 \) does not exist when \( R_0 \leq 1 \).

Note that \( p_0 < 1 \). If \( ak < \sqrt{r} \), then \( p_1 < 1 \). It follows from the discussion for (i) of Theorem 2.1 that \( E_1 \) or \( E_2 \) exists only if \( R_0 > p_1 \). We have the following corollary for giving conditions for such a backward bifurcation to occur.

**Corollary 2.1.** System \((3)\) has a backward bifurcation with endemic equilibria when \( R_0 < 1 \) and \( ak < \sqrt{r} \).

Note that a backward bifurcation with endemic equilibria when \( R_0 < 1 \) is very interesting in applications. The basic reproduction number does not provide a description of the necessary elimination effort; rather the description of the effort is provided through the value of the critical parameter at the turning point. Thus, it is important to identify backward bifurcation to obtain thresholds for the control of diseases.

For \( ak < \sqrt{r} \), a typical bifurcation diagram is illustrated in Fig. 1, where the bifurcation from the disease-free equilibrium at \( R_0 = 1 \) is backward, which gives rise to the existence of multiple endemic equilibria. Further, if \( ak > \sqrt{r} \), the bifurcation at \( R_0 = 1 \) is forward and \((3)\) has one endemic equilibrium for all \( R_0 > 1 \).

**Example 2.1.** Fix \( \mu = 0.2, a = 0.8, A = 2, k = 0.8 \) and \( r = 1.2 \). Then for system \((3)\), a backward bifurcation occurs with two endemic equilibria when \( R_0 < 1 \) and \( p_0 = 0.873 \) in this case (see Fig. 1).

**Remark 2.1.** As shown in Fig. 1, there are two endemic equilibria for an interval of values of \( R_0 \) from a value \( R_0^c \) defined by \( R_0 = p_0 \) to \( R_0 = 1 \). To calculate \( R_0^c \), we substitute the values of \( r, a, \mu, k \) into \( R_0 = p_0 \) to get an expression for \( A \):

\[ A + ak - \frac{\mu}{k} = \pm \sqrt{4r}. \]

Noting that \( ak + \frac{\mu}{k} < A \) and \( R_0 < 1 \), which implies

\[ A^c = \frac{\mu}{k} - ak + \sqrt{4r}, \]

it follows that

\[ R_0^c = \frac{ka^c}{\mu + \frac{\mu}{k}} = p_0. \]

Furthermore, we can conclude that \( R_0^c \) is an increasing function with respect to the parameter \( a \) by directly calculating \( dR_0^c(a)/da > 0 \), which suggests that the dynamics of the model is sensitive to the parameter \( a \).

3. Disease-free equilibrium

By analyzing the eigenvalues of the Jacobian matrices of \((3)\) at the equilibria, we obtain the following results for the stability of the endemic equilibria.
2.5
2
1.5
1
0.5
0
l
0.5 1 1.5 2 2.5
R0
Rc

Fig. 1. The figure for infective sizes at equilibria versus $R_0$ when $\mu = 0.2$, $a = 0.8$, $A = 2$, $r = 1.2$, where backward bifurcation occurs with two equilibria when $R_0 < 1$ and $R_0 = 0.873$.

The Jacobian matrix evaluated at $E_0$ is

$$J_{E_0} = \begin{pmatrix} -\mu & 0 \\ 0 & -k \frac{r}{a} \end{pmatrix}.$$

which implies that $E_0$ is always a saddle.

The Jacobian matrix evaluated at $E_1$ is

$$J_{E_1} = \begin{pmatrix} -A & -kA \\ 0 & kA - \mu - \frac{r}{a} \end{pmatrix},$$

which has negative eigenvalues, implying asymptotic stability of the disease-free equilibrium if and only if

$$\frac{kaA - \mu a - r}{a} < 0,$$

which is equivalent to $R_0 < 1$. So the disease-free equilibrium $E_1$ is locally asymptotically stable if $R_0 < 1$, and is unstable when $R_0 > 1$.

**Theorem 3.1.** The disease-free equilibrium $E_1(A, 0)$ is globally asymptotically stable, i.e., the disease dies out, if and only if the following conditions are satisfied:

$$R_0 < 1 \text{ and } p_1 \geq 1. \quad (12)$$

**Proof.** By (6), we have that $p_0 < 1$. Let us suppose that $p_1 \geq 1$. If $ak < \sqrt{T}$, since $p_1 > 1$, it follows from the discussion for (i), (ii) of Theorem 2.1 that $E_1$ or $E_2$ exists only if $R_0 > p_1$, which is impossible since we have $R_0 < 1$. If $ak \geq \sqrt{T}$ and $R_0 < 1$, it follows from (iii) of Theorem 2.1 that $E_1$ and $E_2$ do not exist. In summary, endemic equilibria do not exist under the assumptions.

It is easy to verify that positive solutions of (3) are ultimately bounded. Note that the nonnegative $S$-axis is positively invariant and the stable manifold partly lies there. Since $E_0$ is always a saddle, its stable manifold lies on the $I$-axis. One can conclude that no trajectory with positive initial condition can have $E_0$ as an omega limit set. Since the initial value is positive, the omega limit set cannot be equal to $E_0$. If it contained $E_0$, then it must also contain an entire orbit different from $E_0$ belonging to the stable manifold of $E_0$. Since $E_1$ is asymptotically stable when $R_0 < 1$, it follows from the Poincaré–Bendixson theorem applied to system (3) that every positive solution of system (3) approaches the disease-free equilibrium $E_1$ as $t$ approaches infinity (see Fig. 3(a)). This completes the proof. \(\square\)

4. Endemic equilibria

4.1. Endemic equilibria for $R_0 = R_0^c$

When $R_0 = R_0^c$, system (3) has a unique interior equilibrium $E^*(S_0, I_0)$, where $S_0 = \frac{ak + A + \frac{\mu}{2}}{r}$, $I_0 = \frac{A - ak - \frac{\mu}{2}}{2k}$. The Jacobian matrix of (3) at $E^*(S_0, I_0)$ is

$$J_{E^*} = \begin{pmatrix} -S_0 & -kS_0 \\ kl_0 & \frac{rl_0}{(a + l_0)^2} \end{pmatrix}.$$
After some calculations, we know that \( E^*(S_0, I_0) \) is a saddle-node type of equilibrium. We first translate the positive equilibrium \( E^+(S_0, I_0) \) of system (3) to the origin. Let \( x = S - S_0, y = I - I_0 \) and \( dt = \frac{dC}{a + y + I_0} \). For the sake of simplicity, we still denote \( x, y, \) and \( t \) by \( S, I, \) and \( t \), respectively. Then system (3) can be written as

\[
\begin{align*}
\dot{S} &= (a + I + I_0)(A - 2S_0 - kI_0)S - kS_0I - kSI - S^2, \\
\dot{I} &= [kS_0 + kS_0 - \mu](a + I + I_0) + (kS_0I_0 - \mu I_0 - r)I.
\end{align*}
\]

Note that \( A - S_0 - kI_0 = 0, kS_0 - \mu - \frac{r}{a + y + I_0} = 0 \), which yields

\[
\begin{align*}
\dot{S} &= -S_0(a + I_0)S - \mu(a + I_0) + rI - (ka + A)SI - (a + I_0)S^2 - kS_0I^2 + [S, I]_3, \\
\dot{I} &= kI_0(a + I_0)S + \frac{rI_0}{a + I_0}I + k(a + 2I_0)SI + \frac{r}{a + I_0}I^2 + [S, I]_3,
\end{align*}
\]

where \([S, I]_3 \) are \( C^\infty \) functions in \((S, I)\) of at least third order.

Performing coordinate transformations via \( x = S - \frac{S_0}{I_0}I , y = I, \) for the sake of simplicity, we still denote \( x, y, \) and \( t \) by \( S, I, \) and \( t \), respectively. Note that \( \Delta = 0. \) Then system (14) can be written as

\[
\begin{align*}
\dot{S} &= [S, I]_2, \\
\dot{I} &= kI_0(a + I_0)S - (aS_0 + S_0I_0 - kS_0S_0)I + [S, I]_2,
\end{align*}
\]

where \([S, I]_2 \) are \( C^\infty \) functions in \((S, I)\) of at least second order.

By Theorem 7.1 of Zhang et al. [17, pp. 114] or Theorem 2.11.1 of [18, pp. 150], we know that \( E^+(S_0, I_0) \) is a saddle node. Furthermore, we can calculate \( aS_0 + S_0I_0 - kS_0S_0 + I_0 = (a + I_0)(S_0 - k^2I_0) \), whose sign is determined by \( ak^2 + (a - A)k^2 + (A + \mu)k + \mu > 0 \). The phase portrait in this case consists of two hyperbolic sectors and one parabolic sector; the hyperbolic sector is between the \( S \)-axis and the equilibrium. The topological structure can be sketched as in Fig. 2(a). In this case, there exist two separatrices. Solutions initiating on one side of the separatrix converge to the interior equilibrium.

(ii) \( ak^2 + (a - A)k^2 + (A + \mu)k + \mu < 0 \). The phase portrait in this case consists of two hyperbolic sectors and one parabolic sector; the parabolic sector lies between the \( S \)-axis and the equilibrium. In this case, there exists one separatrix which converges to the interior equilibrium, and all other solutions tend to the equilibrium \( E_1(A, 0) \). The topological structure is shown in Fig. 2(b).

(iii) \( ak^2 + (a - A)k^2 + (A + \mu)k + \mu = 0 \). The equilibrium \( E^+(S_0, I_0) \) is a cusp which consists of two hyperbolic sectors and two separatrices. One of the separatrices converges to the interior equilibrium \( E^+(S_0, I_0) \) and all other solutions tend to the equilibrium \( E_1(A, 0) \).

4.2. Endemic equilibria for \( R_0^* < R_0 < 1 \)

When \( R_0^* < R_0 < 1 \), system (3) admits two endemic equilibria: \( E_1 = (S_1, I_1), E_2 = (S_2, I_2) \). We begin by analyzing the stability of these two equilibria. Let \( J_i \) be the Jacobian matrix of (3) at \( E_i, i = 1, 2 \); then we get

\[
J_i = \begin{pmatrix}
-S_i & -kS_i \\
kI_i & rI_i/a + I_0^2
\end{pmatrix}.
\]
Fig. 3. Graph (a) shows the case where the disease-free equilibrium \( E_1(A, 0) \) is globally asymptotically stable when \( A = 1, \mu = 0.2, a = 0.008, r = 0.25, k = 2 \), where the condition of Theorem 3.1 is satisfied. Graph (b) shows the case where \( E_1^*(S, I) \) is stable when \( A = 1, \mu = 0.02, a = 0.2, r = 0.2, k = 1 \). Graph (c) shows the case where \( E_2^*(S, I) \) is unstable when \( A = 1, \mu = 0.2, a = 0.07, r = 0.25, k = 2 \). Graph (d) shows the case where \( E_2^*(S, I) \) is unstable and a stable limit cycle occurs when \( A = 1, \mu = 0.2, a = 0.12, r = 0.25, k = 2 \).

Thus, we have

\[
\det(J_1) = S_1 I_1 \left( k^2 - \frac{r}{(a + I_1)^2} \right). \tag{16}
\]

Note that \( R_0 > p_0 \), and \( I_1 = \frac{A - ak - \mu}{k} - \sqrt{\left( \frac{A + ak - \mu}{k} - 4r \right) \frac{2k}{k}} \). It follows from the above two conditions that

\[
(a + I_1)^2 = \left( \frac{A + ak - \mu}{2k} - \sqrt{\left( \frac{A + ak - \mu}{k} - 4r \right)} \right)^2 < \left( \frac{A + ak - \mu}{k} \right)^2 < \frac{r}{k^2}. \tag{17}
\]

i.e., \( \det(J_1) < 0 \). It follows that \( E_1 = (S_1, I_1) \) is a saddle point.

By the same argument, we obtain \( \det(J_2) > 0 \). Thus, \( E_2 = (S_2, I_2) \) is a focus, a node, or a center. Further, we have

\[
\text{tr}(J_2) = \frac{rI_2 - S_2(a + I_2)^2}{(a + I_2)^2}. \tag{18}
\]

Now, we propose the following assumption:

(H1) \( \Delta_{20} = [(k - 1)A - \mu + ak]^2 + 4aA(1 - k)k > 0 \).

Define

\[
H = \frac{ak^2}{k - 1} - \frac{\mu}{k(k - 1)} - \sqrt{[(k - 1)A - \mu + ak]^2 + 4aA(1 - k)k}. \tag{19}
\]

The stability of equilibrium \( E_2 \) is stated in the following theorem.
Theorem 4.1. Let \( ak < \sqrt{r}, p_1 < R_0 < 1 \) and (H1) hold. For system (3), we have

(i) \( E_2 \) is stable (see Fig. 3 (b)) if either

\[
(k - 1)A - \mu + ak \leq 0.
\]

or

\[
(k - 1)A - \mu + ak > 0,
\]

\[
r < \frac{1}{4} \left[ \left( ak + A - \frac{\mu}{k} \right)^2 - H^2 \right].
\]

(ii) \( E_2 \) is unstable (see Fig. 3 (c) and (d)) if

\[
(k - 1)A - \mu + ak > 0,
\]

\[
r > \frac{1}{4} \left[ \left( ak + A - \frac{\mu}{k} \right)^2 - H^2 \right].
\]

where \( H \) is defined in (19).

Proof. Since \( nl_2/(a + l_2) = kS_2l_2 - \mu l_2 \), we see that the trace of \( J_2 \) is

\[
\text{tr}(J_2) = \frac{1}{a + l_2}. \left[ (k - 1)S_2l_2 - aS_2 - \mu l_2 \right].
\]

Thus, the trace is negative if \( k \leq 1 \). Suppose that \( k > 1 \). Let us find the conditions under which \( \text{tr}(J_2) = 0 \). Since \( S_2 = A - kl_2 \), it follows from (23) that \( \text{tr}(J_2) = 0 \) is equivalent to

\[
(1 - k)kl_2^2 + ((k - 1)A - \mu + ak)l_2 - aA = 0.
\]

Thus, the set of \( \text{tr}(J_2) = 0 \) is empty if

\[
(k - 1)A - \mu + ak \leq 0.
\]

Suppose that

\[
(k - 1)A - \mu + ak > 0.
\]

It follows from (H1) that we obtain

\[
I_2 = \frac{[(k - 1)A - \mu + ak]}{2(k - 1)k} \left[ 1 \pm \sqrt{1 + \frac{4aA(1 - k)k}{[(k - 1)A - \mu + ak]^2}} \right].
\]

In view of \( I_2 < A/k \), we have

\[
I_2 = \frac{[(k - 1)A - \mu + ak]}{2(k - 1)k} \left[ 1 - \sqrt{1 + \frac{4aA(1 - k)k}{[(k - 1)A - \mu + ak]^2}} \right].
\]

Hence, after a long and tedious calculation and using Eq. (19), Eq. (27) can be reduced to

\[
r = \frac{1}{4} \left[ \left( ak + A - \frac{\mu}{k} \right)^2 - H^2 \right].
\]

As a consequence, we see that (H1), (26) and (28) are the necessary and sufficient conditions for \( \text{tr}(J_2) = 0 \). The previous discussion show that the stability of \( E_2 \) does not change if (25) holds. It follows from the definition of \( \text{tr}(J_2) = 0 \) that (25) implies that \( \text{tr}(J_2) < 0 \). Therefore, \( E_2 \) is stable if (20) holds. It follows from (23)–(28) that \( \text{tr}(J_2) < 0 \) if (21) is valid and that \( \text{tr}(J_2) > 0 \) if (22) holds. This completes the proof. \( \Box \)

Remark 4.1. As in the discussion above, we find that as for the two endemic equilibria, one is always a saddle; the other may be stable or unstable, that is, \( E_2 \) exhibits a stable state and an unstable state as \( r \) increases from 0. This suggests the possibility that (3) admits a Hopf bifurcation. In Fig. 3(c), endemic equilibria are unstable and there is no limit cycle, so almost all orbits approach the disease-free equilibrium as time tends to infinity.

From the above theorem, we know that the interior equilibrium \( E_2^* (S_2, l_2) \) of system (3) is a center type nonhyperbolic equilibrium when \( \text{tr}(J_2) = 0 \). This suggests the possibility that (3) admits a Hopf bifurcation. Let us now verify the existence of a Hopf bifurcation in (3).

Set

\[
r_0 = \frac{1}{4} \left[ \left( ak + A - \frac{\mu}{k} \right)^2 - H^2 \right].
\]
Theorem 4.2. Let \( ak < \sqrt{r} \), \( p_1 < R_0 < 1 \) and (H1) hold. Assume further that
\[
(k - 1)A - \mu + ak > 0 \quad \text{and} \quad k(a - 1) + 2A + 2 < 0. 
\]
Then there is a family of stable limit cycles if \( r \) is less than and near \( r_0 \), i.e., a supercritical Hopf bifurcation occurs when \( r \) passes through \( r_0 \).

Proof. Suppose \( r = r_0 \); then \( \text{Tr}(J_2) = 0 \). First of all, perform coordinate transformations using \( x = S - S_2, y = I - I_2 \) and \( \frac{dx}{dt} = \frac{dy}{dt} \). For the sake of simplicity, we still denote \( x, y, \) and \( \tau \) by \( S, I, \) and \( t \), respectively.

It follows from \( A - S_2 - kl_2 = 0, kS_2 - \mu - \frac{r}{\sigma + l_2} = 0 \) and \( rl_2 - S_2(a + l_2)^2 = 0 \) that system (3) becomes
\[
\begin{cases}
\dot{S} = -S_2(a + l_2)S - kS_2(a + l_2)I - (k(a + A)S - (a + l_2)S^2 - kS_2^2 - kSl^2 - S^2l), \\
\dot{I} = kl_2(a + l_2)S + S_2(a + l_2)I + k(a + 2l_2)Sl + \frac{r}{a + l_2}l^2 + kSl^2.
\end{cases}
\]

Set
\[
\omega = \sqrt{\det(J_2)} > 0. 
\]
Then the eigenvalues of \( J_2 \) are \( \lambda_1 = \omega i \) and \( \lambda_2 = -\omega i \).

Defining \( m = S_2(a + l_2) \) and \( p = kl_2(a + l_2) \), then
\[
\omega = \sqrt{pm - m^2} = \sqrt{S_2(a + l_2)^2(k^2l_2 - S_2)} > 0. 
\]
Setting \( x = pS + ml, y = o\omega \) and using \( \text{tr}(J_2) = 0 \), we obtain
\[
\begin{cases}
\dot{x} = -\omega y + f(x, y), \\
\dot{y} = o\alpha x + g(x, y),
\end{cases}
\]
where \( f(x, y), g(x, y) \) are \( C^\infty \) functions and satisfy \( f(0, 0) = g(0, 0) = 0 \);
\[
f(x, y) = \frac{2m(a + l_2) - p(ka + A) + mk(a + 2l_2)}{\omega}xy + \frac{pm(ka + A) - km^2l_2 - kpl^2S_2 - (a + l_2)m^2}{\omega}y^2 \\
- \frac{a + l_2}{p}x^2 - \frac{1}{\omega}xy + \frac{2m - pk + mk}{\omega}xy^2 + \frac{\omega^2 - km^2}{\omega^3}y^3,
\]
\[
g(x, y) = \frac{k(a + 2l_2)}{p}xy - \frac{km^2}{\omega}y^2 + \frac{k}{\omega}xy^2 - \frac{km}{\omega^2}y^3. 
\]

According to the formula for the third focal value (i.e., the Lyapunov number) of a multiple focus (p. 253 of Andronov [19]), we have the first Lyapunov number \( \alpha \) of the equilibrium \( (0, 0) \) of system (30) as follows:
\[
\alpha = \left| \frac{f_{xx} + f_{xy} + g_{xx} + g_{yy} + f_{yy}(f_{xx} + f_{yy}) - g_{xy}(g_{xx} + g_{yy}) - f_{xxy}g_{xx} + f_{yy}g_{yy}}{16\omega} \right|_{x=0,y=0}. 
\]

After elementary but lengthy computations, we have
\[
\alpha = \frac{(a + l_2)[2S_2(1 - k) - k^2l_2^2]}{8\omega p^2} + \frac{(a + l_2)^3}{16p^2\omega^3} [(2 + k)S_2(a + l_2) + kS_2l_2 - kl_2(ka + A)](-2k^2S_2l_2^2) \\
+ \frac{(a + l_2)^3}{16p^2\omega^4} [k^3(a + 2l_2)S_2l_2 - k(a + 2l_2)S_2^2 - 2ak^2S_2l_2 - 2S_2^2(a + l_2)l_2]kS_2l_2] \\
= \frac{2k(a + l_2)^3S_2l_2}{16p^2\omega^4} [(ka - 1) + 2A + 2S_2 - kA - aS_2^2](k^2l_2 - S_2) \\
+ \frac{2k(a + l_2)^3S_2l_2}{16p^2\omega^4} [-2AS_2(a + l_2) - 3ak^3S_2l_2 - (A - ka - 2kl_2)k^2l_2]. 
\]

It follows from (31) and \( l_2 < (A - ak)/2k \) that \( \alpha < 0 \). The conclusion of this theorem also follows from Theorem 3.4.2 of [20]. \( \square \)

Remark 4.2. Theorems 4.1 and 4.2 imply that the Allee effect occurs because endemic equilibrium \( E_2 \) and the disease-free equilibrium can be stable at the same time, or a stable limit cycle and the disease-free equilibrium can be stable at the same time.
Suppose that $R_0 = 1$ and $k > 1$, then the equilibrium $E^*_2(S^*_2, I^*_2)$ goes to the equilibrium $E_1(A, 0)$ such that the equilibrium is a complex equilibrium. By analysis we readily find that the equilibrium $E_1(A, 0)$ is a saddle-node point and $E^*_2(S^*_2, I^*_2)$ is a locally asymptotically stable equilibrium. Moreover, there are two limit cycles in the interior of the first quadrant (see Fig. 5).

4.3. Endemic equilibria for $R_0 > 1$

Theorem 4.3. Suppose that $R_0 > 1$, $k < 1$ and (20), (21) hold; then system (3) has a globally asymptotically stable equilibrium $E^*_2(S^*_2, I^*_2)$ in the interior of the first quadrant.

Proof. It is easy to check that the equilibrium $E^*_2(S^*_2, I^*_2)$ is a stable focus (or node). By Lemma 2.1, system (3) is ultimately bounded. Next we prove that system (3) has no periodic orbits in the interior of the first quadrant. It is clear that the dynamics of system (3) is equivalent to that of the system

\[
\begin{cases}
\dot{S} = S(A - S) - kl = f(S, I), \\
\dot{I} = I(kS - \mu - \frac{r}{a + I}) = g(S, I).
\end{cases}
\]

Take the Dulac function $B(S, I) = \frac{a + I}{S}$ for system (33), which yields

\[
\frac{\partial(Bf)}{\partial S} + \frac{\partial(Bg)}{\partial I} = k - 1 - \frac{\mu}{S} - \frac{a}{I}.
\]

Since $k < 1$, we have $\frac{\partial(Bf)}{\partial S} + \frac{\partial(Bg)}{\partial I} < 0$. It follows directly from the Bendixson–Dulac criterion that system (3) has no limit cycle in the interior of the first quadrant. Note that positive solutions stay eventually in the compact region $\mathcal{O}$. It follows that the endemic equilibrium $E^*_2(S^*_2, I^*_2)$ is globally asymptotically stable in the interior of the first quadrant (see Fig. 4(a)).

Consequently, we have:

Theorem 4.4. Suppose that $R_0 > 1$ and (22) hold; then system (3) has at least one limit cycle in the interior of the first quadrant.

Proof. We have proved the ultimate boundedness of the system in Lemma 2.1. On the other hand, we know that the equilibrium $E_1(A, 0)$ is unstable if $R_0 > 1$, that is to say, no solution with positive initial condition can have $E_2$ as an omega limit point. Also the omega limit set cannot contain an unbounded orbit. Therefore, the existence of a periodic orbit follows directly from the Poincaré–Bendixson theorem. Moreover, at least one periodic orbit is stable in the interior of the first quadrant. The phase portrait is shown in Fig. 4(b).

5. Persistence of disease as $R_0 \geq 1$

In order to prove the uniform persistence of system (3), we present the persistent theorem for an infinite dimensional system from paper [21]. Let $X$ be a complete metric space. Suppose that $X^0, X_0 \subset X, X^0 \cap X_0 = \emptyset$ and $X^0 \cup X_0 = X$. Assume that $T(t)$ is a $C_0$ semigroup on $X$ satisfying

\[
T(t): X^0 \to X^0, \quad T(t): X_0 \to X_0.
\]

Let $T_0(t) = T(t)|_{X_0}, T_0(t) = T(t)|_{X^0},$ and $A_0$ be the global attractor for $T_0(t)$.
Fig. 5. The phase portrait of system (3) is given in graph (a) when $A = 2.6, \mu = 0.2, a = 0.05, r = 0.25, k = 2$. Graph (b) shows that $E_2^*$ is local asymptotically stable. Graphs (b) and (d) show the existence of an unstable limit cycle (graph (c)) which corresponds to the smaller cycle that appeared in graph (a). Graphs (d) and (f) show the existence of a stable limit cycle (graph (e)) which corresponds to the larger cycle that appeared in graph (a).

Lemma 5.1 ([21]). Suppose that $T(t)$ satisfies (3) and that the following conditions are satisfied:

(i) There is a $t_0 > 0$ such that $T(t)$ is compact for $t > t_0$.
(ii) $T(t)$ is point dissipative in $X$.
(iii) $\overline{A}_0 = \bigcup_{x \in A_0} W(x)$ is isolated and has an acyclic covering $M$, where

$$M = \{M_1, M_2, \ldots, M_3\}.$$ 

Then $T(t)$ is uniformly persistent if $W^i(M_i) \cap X^0 = \emptyset (i = 1, 2, \ldots, n)$, where $W(x)$ is the $\omega$-limit set, and $W^i(M_i)$ is the stable manifold of invariable set.
Let us define: \( X = \{(S, I) : S \geq 0, I \geq 0\} \); \( X^0 = \{(S, I) : S \geq 0, I > 0\} \); \( X_0 = \{(S, I) : S \geq 0, I = 0\} \). Thus \( X^0 \) is open and dense in \( X \) with \( X^0 \cup X_0 = X \) and \( X^0 \cap X_0 = \emptyset \). We only need to show that \( E_1(A, 0) \) is a weaker repeller for \( X^0 \).

**Theorem 5.1.** If \( R_0 \geq 1 \), then the system (3) is uniformly persistent in the first quadrant.

**Proof.** We have shown the ultimate boundedness of the system in Lemma 2.1. It is easy to see that \( X^0 \) and \( X_0 \) are positive invariant, and conditions (i), (ii) and (iii) are clearly satisfied. We now show that \( W^s(X_0) \cap X^0 = \emptyset \). There exists a constant solution \( E_1(A, 0) \) in \( X_0 \). If there is a solution which has the initial condition in \( X_0 \), we can get \( (S(t), I(t)) \to (A, 0) \) when \( t \to \infty \). This means that \( E_1(A, 0) \) is isolated and is an acyclic covering. Suppose that \( W^s(X_0) \cap X^0 \neq \emptyset \); this implies the existence of a positive solution \( (S(t), I(t)) \) with positive initial condition which has \( E_1 \) as an omega limit point such that \( (S(t), I(t)) \to (A, 0) \) when \( t \to \infty \). As \( R_0 > 1 \), there exists an arbitrarily small \( \varepsilon > 0 \) such that
\[
\varepsilon_1 = kA - \mu - \frac{r}{a} - ke > 0.
\]

When \( t \) is large enough, we have
\[
A - \varepsilon < S(t) < A + \varepsilon, \quad I(t) < \varepsilon.
\]

Thus
\[
\frac{dI}{dt} = \left(kS - \mu - \frac{r}{a + I} \right) I > \left(k(A - \varepsilon) - \mu - \frac{r}{a} \right) I = \varepsilon_1 I > 0.
\]

So \( \lim_{t \to \infty} I(t) \to \infty \), which is a contradiction to \( \lim_{t \to \infty} I(t) \to 0 \). It follows that \( W^s(X_0) \cap X^0 = \emptyset \). When \( kA - \mu - \frac{r}{a} = 0 \), which is equivalent to \( R_0 = 1 \), \( E_1(A, 0) \) being a saddle node, there is no solution with positive initial condition which has \( E_1 \) as an omega limit point. This completes the proof. \( \square \)

By carrying out global qualitative and bifurcation analysis, it is shown that, when some conditions are satisfied, either the number of infective individuals tends to zero as time evolves or there is a region such that the disease will be persistent if the initial position lies in the region and the disease will disappear if the initial position lies outside this region.

**Example 5.1.** In Fig. 2(a), when we change \( a \) from 0.2 to 0.1, there is no endemic equilibrium, and the disease-free equilibrium is globally asymptotically stable. When we change \( r \) from 0.25 to 0.26, there is only one equilibrium point; it is globally asymptotically stable.

**Example 5.2.** In Fig. 2(b), when we change \( r \) from 0.25 to 0.28, there is only one equilibrium, it is globally asymptotically stable.

6. **Discussion**

In this paper, by combining qualitative and bifurcation analysis, we have studied an SIR epidemic model with a saturated treatment function theoretically and found rich dynamics of the model. More precisely, \( E_0(0, 0) \) always exists and is a saddle. \( E_1(A, 0) \) is locally stable when \( R_0 < 1 \) and is unstable when \( R_0 > 1 \), where \( R_0 \) is introduced as the basic reproduction number. When \( R_0 > 1 \) there is a unique endemic equilibrium \( E^*_2 \); otherwise, when \( R_0 < 1 \), there may exist no equilibria, one equilibrium \( E^*_1 \), or two endemic equilibria \( E^*_1 \) and \( E^*_2 \), which implies the existence of backward bifurcation. As for \( E^*_1 \), it is a saddle node; we simulate the saddle-node bifurcation in Fig. 2(a) and (b). As for the two endemic equilibria, \( E^*_1 \) is always a saddle, and \( E^*_2 \) may be stable or unstable.

When there is a backward bifurcation, it is not enough to drive the basic reproduction number below 1 to eradicate the disease. We should do more work to reduce the basic reproduction number further as well. Detailed analysis of the stability of \( E^*_2 \) shows that stability switches for \( E^*_2 \) exist, which indicates that when \( R_0 \) increases, the stability condition of \( E^*_2 \) will change from stable to unstable. We also study the Hopf bifurcation and obtain the criteria for judging its stability by calculating Lyapunov coefficients. We establish Theorem 4.2 for the existence of a supercritical Hopf bifurcation. Further, Theorems 4.1 and 4.2 imply that the Allee effect occurs because endemic equilibrium \( E^*_2 \) and the disease-free equilibrium can be stable at the same time, or a stable limit cycle and the disease-free equilibrium can be stable at the same time. Combining simulations when \( R_0 \) increases for \( R_0 < 1 \), we find that the eventual behavior of the system is sensitive to the initial positions, which makes the model realistic. By the Bendixson–Dulac criteria and the Poincaré–Bendixson theorem, we obtain global asymptotic stability of \( E^*_2 \) under some suitable conditions. Our results suggest that the treatment of the infecteds being delayed is one of the origins of the backward bifurcation. As for \( dR^*_0(a)/da > 0 \), this suggests that the dynamics of the model is sensitive to the parameter \( a \). Hence in order to eradicate the disease, we should improve our medical technology and invest more in medicines, beds etc. to give patients timely treatment. Therefore, it is indeed the saturated treatment function that produces the complicated dynamics of epidemic models and makes the models more reasonable and practical.

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