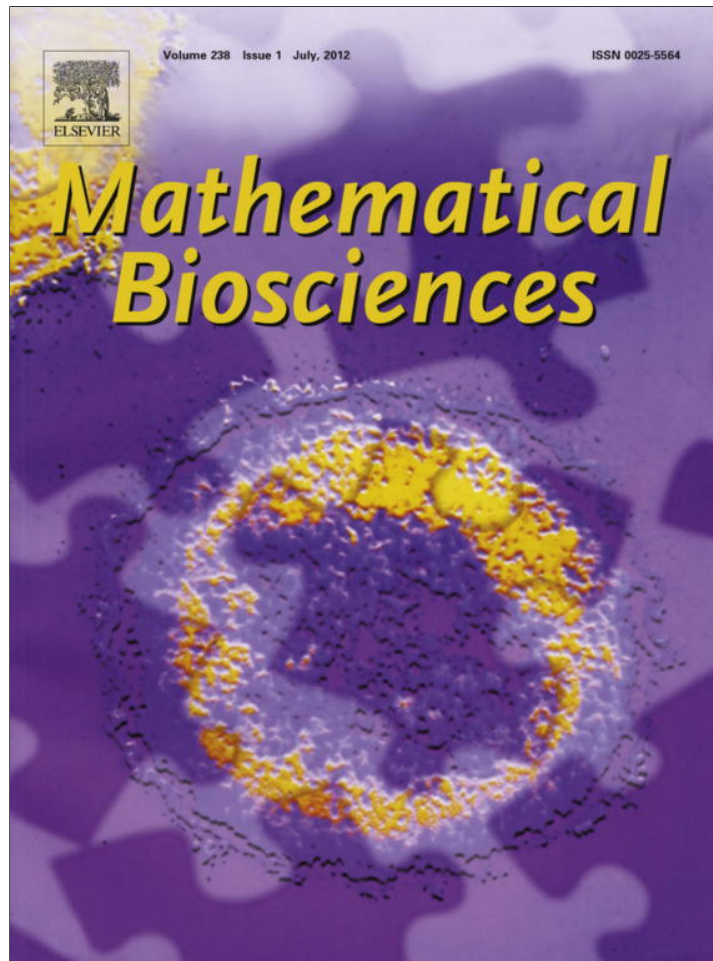


Provided for non-commercial research and education use.  
Not for reproduction, distribution or commercial use.



This article appeared in a journal published by Elsevier. The attached copy is furnished to the author for internal non-commercial research and education use, including for instruction at the authors institution and sharing with colleagues.

Other uses, including reproduction and distribution, or selling or licensing copies, or posting to personal, institutional or third party websites are prohibited.

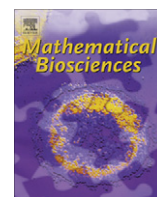
In most cases authors are permitted to post their version of the article (e.g. in Word or Tex form) to their personal website or institutional repository. Authors requiring further information regarding Elsevier's archiving and manuscript policies are encouraged to visit:

<http://www.elsevier.com/copyright>



Contents lists available at SciVerse ScienceDirect

## Mathematical Biosciences

journal homepage: [www.elsevier.com/locate/mbs](http://www.elsevier.com/locate/mbs)

## Analysis of SIR epidemic models with nonlinear incidence rate and treatment

Zhixing Hu<sup>a,1</sup>, Wanbiao Ma<sup>a</sup>, Shigui Ruan<sup>b,\*,2</sup><sup>a</sup> Department of Applied Mathematics, University of Science and Technology Beijing, Beijing 100083, PR China<sup>b</sup> Department of Mathematics, University of Miami, Coral Gables, FL 33124-4250, USA

## ARTICLE INFO

## Article history:

Received 27 March 2011

Received in revised form 26 March 2012

Accepted 27 March 2012

Available online 9 April 2012

## Keywords:

Epidemic model

Treatment rate

Nonlinear incidence rate

Backward bifurcation

Stability

## ABSTRACT

This paper deals with the nonlinear dynamics of a susceptible-infectious-recovered (SIR) epidemic model with nonlinear incidence rate, vertical transmission, vaccination for the newborns of susceptible and recovered individuals, and the capacity of treatment. It is assumed that the treatment rate is proportional to the number of infectives when it is below the capacity and constant when the number of infectives reaches the capacity. Under some conditions, it is shown that there exists a backward bifurcation from an endemic equilibrium, which implies that the disease-free equilibrium coexists with an endemic equilibrium. In such a case, reducing the basic reproduction number less than unity is not enough to control and eradicate the disease, extra measures are needed to ensure that the solutions approach the disease-free equilibrium. When the basic reproduction number is greater than unity, the model can have multiple endemic equilibria due to the effect of treatment, vaccination and other parameters. The existence and stability of the endemic equilibria of the model are analyzed and sufficient conditions on the existence and stability of a limit cycle are obtained. Numerical simulations are presented to illustrate the analytical results.

© 2012 Elsevier Inc. All rights reserved.

## 1. Introduction

One of the focuses of theoretical studies in mathematical epidemiology is to understand the nonlinear dynamics of various epidemic models. Classical susceptible-infectious-recovered (SIR) models with a bilinear incidence rate usually have a disease-free equilibrium and at most one endemic equilibrium (see Capasso and Serio [7] and Hethcote [20]). In such a case, the basic reproduction number is a threshold: when it is less than unity the disease-free equilibrium exists and is stable, which indicates that the disease will die out; when it is greater than unity the disease-free equilibrium becomes unstable and an endemic equilibrium exists, which demonstrates that the disease will persist. The bifurcation leading from a disease-free equilibrium to an endemic equilibrium is called *forward*.

Recently there has been a great interest in investigating the nonlinear dynamics (such as Hopf bifurcation, saddle-node bifurcation, Bogdanov–Takens bifurcation, existence of periodic and homoclinic orbits, coexistence of limit cycles and homoclinic orbits) in epidemic models with multiple endemic equilibria due to social

groups with different susceptibilities, nonlinear or nonmonotone incidence rate, stage structure, behavioral change of susceptibles, etc. (see Alexander and Moghadas [1], Derrick and van den Driessche [12], Feng and Thieme [14], Hu et al. [21], Liu et al. [26,27], Ruan and Wang [30], Tang et al. [33], and Xiao and Ruan [39]). For instance, Liu et al. [26] proposed a nonlinear saturated incidence function  $\beta SI^\ell / (1 + \alpha I^h)$  to model the effect of behavioral changes to certain communicable diseases, where  $\beta SI^\ell$  describes the infection force of the disease,  $1/(1 + \alpha I^h)$  measures the inhibition effect from the behavioral change of the susceptible individuals when the number of infectious individuals increases,  $\ell$ ,  $h$ , and  $\beta$  are all positive constants, and  $\alpha$  is a nonnegative constant. The case when  $\ell = h = 1$ , i.e. the function  $\beta SI / (1 + \alpha I)$ , was used by Capasso and Serio [7] to represent a “crowding effect” or “protection measure” in modeling the cholera epidemics in Bari in 1973. Because of the nonlinearity and saturation property of these incidence functions, it has been shown that SIR epidemic models with such nonlinear incidence rates can possess multiple endemic equilibria. Moreover, such models can exhibit Hopf bifurcation, saddle-node bifurcation and Bogdanov–Takens bifurcation [1,12,26,27,30], the existence of two limit cycles [21], and the coexistence of a limit cycle and a homoclinic cycle [33]. These results demonstrate that the nonlinear dynamics of these epidemic models are very sensitive to the model parameters and various outcomes could occur depending on the initial population sizes. The disease may be eradicated (solutions approach a disease-free equilibrium), persistent (solutions approach an endemic equilibrium), or occurring periodically

\* Corresponding author.

E-mail addresses: [bkdzhx@163.com](mailto:bkdzhx@163.com) (Z. Hu), [ruan@math.miami.edu](mailto:ruan@math.miami.edu) (S. Ruan).<sup>1</sup> Research was partially supported by the National Natural Science Foundation of China (11071013).<sup>2</sup> Research was partially supported by the National Science Foundation (DMS-1022728).

(solutions approach a limit cycle). Understand such nonlinear dynamics and identify the underlie factors is very important in the control and prevention of spread of communicable diseases.

The phenomenon that the disease-free equilibrium coexists with an endemic equilibrium when the basic reproduction number is less than unity was first observed by Castillo-Chavez et al. [8,9] and Huang et al. [23] in multi-group HIV/AIDS models. It was termed as *backward bifurcation* by Hadelor and Castillo-Chavez [17] and Hadelor and van den Driessche [18] and was shown to exist in epidemic models that include behavioral responses (such as via education) to perceived disease risk. The existence of backward bifurcation in epidemic models has important qualitative implications since the disease now cannot be eradicated by simply reducing the basic reproduction number to be less than unity. Dushoff et al. [13] provided a general framework for the mechanisms behind backward bifurcations in simple epidemic models and discussed the biological interpretation of the features of these models that induce backward bifurcations. Therefore, it is also important to study backward bifurcation in epidemic models in order to seek for conditions for the control of diseases (see Arino et al. [3], Blayneh et al. [4], Brauer [5,6], Greenhalgh and Griffiths [15], Kribs-Zaleta and Velasco-Hernandez [24], Regula et al. [29], Safan et al. [31], van den Driessche and Watmough [34], and Wan and Zhu [35]). In this case, the basic reproduction number does not give information on disease elimination; rather disease elimination is determined by the values of critical parameters at the turning points.

Treatment is an important and effective method to prevent and control the spread of various infectious diseases. In classical epidemic models, the treatment rate of the infectives is assumed to be proportional to the number of the infective individuals [2]. During the SARS outbreaks in 2003, the dramatically increasing of SARS cases in Beijing challenged the normal public-health system and capacity in Beijing City and forced the Chinese government to create the first and only SARS hospital, Beijing Xiaotangshan Hospital, to treat the large number of SARS patients [37]. After this experience, researchers started to consider the capacity of the health-care system from both modeling and analyzing points of view. Wang and Ruan [38] considered a SIR model in which the capacity for the treatment of a disease in a community is a constant. Namely, they used the following function

$$T(I) = \begin{cases} k, & \text{if } I > 0, \\ 0, & \text{if } I = 0 \end{cases} \quad (1.1)$$

to describe the treatment rate and studied the effect of treatment parameter  $k$  on the dynamics of the model. They found that the model undergoes a sequence of bifurcations including saddle-node, Hopf, and homoclinic bifurcations and exhibits homoclinic orbits even though incidence rate is assumed to be bilinear. Moreover, it is shown that it may not be necessary to set  $k$  so high to eliminate endemic equilibria since such equilibria may be unstable. Wang [36] proposed the following piecewise linear treatment function

$$T(I) = \begin{cases} kI, & \text{if } 0 \leq I \leq I_0, \\ u = kI_0, & \text{if } I > I_0, \end{cases} \quad (1.2)$$

in a SIR model, where  $I_0$  is the infective level at which the health-care system reaches capacity; that is, treatment increases linearly with  $I$  before the capacity is reached and is constant afterward. It was shown that the model has bistable endemic equilibria when  $I_0$  is low and backward bifurcation can occur.

The treatment function (1.2) has been used by some other researchers. For example, Zhang and Liu [41] studied a model with a general incidence rate  $\lambda(I+S)^{n-1}SI$  ( $0 \leq n \leq 1$ ) and the treatment function (1.2). Hu et al. [22] considered an epidemic model with standard incidence rate  $\beta SI/N$  and the treatment function (1.2). Li

et al. [25] studied an epidemic model with nonlinear incidence rate  $\beta I/(1+\alpha I)$  with the treatment function (1.2) and analyzed the stability and bifurcation of the system. Other types of treatment functions have also been proposed. For instance, Zhang and Liu [40] used a saturated treatment function  $T(I) = rI/(1+\mu I)$ ,  $r > 0$ ,  $\mu \geq 0$ , and found that the saturated function has the advantage of giving near-linear treatment response when  $I$  is low and approaches a constant capacity as  $I$  gets large. Eckalbar and Eckalbar [11] introduced a new treatment function,  $T(I) = \max\{\gamma I - gI^2, 0\}$ ,  $\gamma > 0$ ,  $g > 0$ , into a SIR model with bilinear incidence rate. It was found that the system could have up to four equilibria with possible bi-stability, backward bifurcations, and limit cycles. See also Li et al. [25], Zhou and Fan [42], etc.

In this paper, we consider a SIR epidemic model with the nonlinear incidence rate  $\beta SI/(1+\alpha I)$ , the treatment rate function (1.2), vertical transmission, and vaccination for the newborns of the susceptible and recovered individuals. To formulate our model, let  $S(t)$ ,  $I(t)$  and  $R(t)$  be the number of susceptible, infective and recovered individuals at time  $t$ , respectively. The basic assumptions are as follows.

- (i) The total population size at time  $t$  (day) is denoted by  $N = S + I + R$ . The newborns of  $S$  and  $R$  are susceptible individuals, and the newborns of  $I$  who are not vertically infected are also susceptible individuals.
- (ii) The positive constant  $b$  (per day) denotes the death rate and birth rate of susceptible and recovered individuals. The positive constant  $\delta$  (per day) denotes the death rate and birth rate of infective individuals. The positive constant  $\gamma$  (per day) is the natural recovery rate of infective individuals. The positive constant  $q$  ( $q \leq 1$ ) (per day) is the vertical transmission rate, and note  $p = 1 - q$  (per day), then  $0 \leq p \leq 1$ . Fraction  $m'$  of all newborns with mothers in the susceptible and recovered classes are vaccinated and appeared in the recovered class, while the remaining fraction,  $m = 1 - m'$ , appears in the susceptible class.
- (iii) The incidence rate is described by a nonlinear function  $\beta SI/(1+\alpha I)$ , where  $\beta$  (per day) is a positive constant describing the infection rate and  $\alpha$  (per person) is a nonnegative constant represents the half saturation constant.
- (iv) The treatment rate of a disease is  $T(I)$  given in (1.2).

Under the above assumptions, the SIR epidemic model takes the following form:

$$\begin{cases} \frac{dS}{dt} = bm(S+R) - \beta \frac{SI}{1+\alpha I} - bS + p\delta I, \\ \frac{dI}{dt} = \beta \frac{SI}{1+\alpha I} + q\delta I - \delta I - \gamma I - T(I), \\ \frac{dR}{dt} = \gamma I - bR + bm'(S+R) + T(I). \end{cases} \quad (1.3)$$

Because

$$\frac{dN}{dt} = 0,$$

the total number of population  $N$  is a constant. For convenience, it is assumed that  $N = S + I + R = 1$ , thus  $S$ ,  $I$  and  $R$  are taken as the proportions of susceptible, infective and recovered individuals in the total population. By using  $S + R = 1 - I$ , the first two equations of system (1.3) do not contain the variable  $R$ . Therefore, system (1.3) is equivalent to the following 2-dimensional system:

$$\begin{cases} \frac{dS}{dt} = -\beta \frac{SI}{1+\alpha I} - bS + bm(1-I) + p\delta I, \\ \frac{dI}{dt} = \beta \frac{SI}{1+\alpha I} - p\delta I - \gamma I - T(I). \end{cases} \quad (1.4)$$

It is easy to verify that the positive invariant set of system (1.4) is

$$D = \{(S, I) | S \geq 0, I \geq 0, S + I \leq 1\}.$$

When  $0 \leq I \leq I_0$ , system (1.4) is

$$\begin{cases} \frac{dS}{dt} = -\beta \frac{SI}{1+\alpha I} - bS + bm(1-I) + p\delta I, \\ \frac{dI}{dt} = \beta \frac{SI}{1+\alpha I} - p\delta I - \gamma I - kI. \end{cases} \quad (1.5)$$

When  $I > I_0$ , system (1.4) becomes ( $u = kI_0$ )

$$\begin{cases} \frac{dS}{dt} = -\beta \frac{SI}{1+\alpha I} - bS + bm(1-I) + p\delta I, \\ \frac{dI}{dt} = \beta \frac{SI}{1+\alpha I} - p\delta I - \gamma I - u. \end{cases} \quad (1.6)$$

The purpose of this paper is to study the nonlinear dynamics of system (1.4). Under some conditions, it is shown that there exists a backward bifurcation from an endemic equilibrium, which implies that the disease-free equilibrium coexists with an endemic equilibrium. In such a case, reducing the basic reproduction number less than unity is not enough to control and eradicate the disease, extra measures are needed to ensure that the solutions approach the disease-free equilibrium. When the basic reproduction number is greater than unity, model (1.4) can have multiple endemic equilibria due to the effect of treatment, vaccination and other parameters. The existence and stability of the endemic equilibria of the model are analyzed and sufficient conditions on the existence and stability of a limit cycle are obtained. Numerical simulations are presented to illustrate the analytical results.

The organization of this paper is as follows. In next section, we analyze the existence and bifurcations of equilibria for (1.4). In Section 3, we study the stability of various equilibria and the existence and stability of a limit cycle in (1.4). In Section 4, we give some numerical simulations to verify our results. A brief discussion is presented in Section 5.

## 2. Existence of equilibria

In this section, we consider the equilibria of system (1.4). Obviously,  $E_0(m, 0)$  is the disease-free equilibrium of (1.4).

For the positive equilibrium  $E^*(S^*, I^*)$  of system (1.4), when  $0 < I^* \leq I_0$ , let the right side of (1.5) be equal to zero. Then  $S^*$  and  $I^*$  satisfy the following equations:

$$\begin{cases} -\beta \frac{S^* I^*}{1+\alpha I^*} - bS^* + bm(1-I^*) + p\delta I^* = 0, \\ \beta \frac{S^* I^*}{1+\alpha I^*} - (p\delta + \gamma + k)I^* = 0. \end{cases} \quad (2.1)$$

When  $I^* > I_0$ , let the right side of (1.6) be equal to zero. Then  $S^*$  and  $I^*$  satisfy the following equations

$$\begin{cases} -\beta \frac{S^* I^*}{1+\alpha I^*} - bS^* + bm(1-I^*) + p\delta I^* = 0, \\ \beta \frac{S^* I^*}{1+\alpha I^*} - (p\delta + \gamma)I^* - u = 0. \end{cases} \quad (2.2)$$

From (2.1) we obtain that

$$I^* = \frac{b(p\delta + \gamma + k)(R_0 - 1)}{\beta(bm + \gamma + k) + \alpha b(k + \gamma + \delta p)}, \quad (2.3)$$

where

$$R_0 = \frac{\beta m}{p\delta + \gamma + k} \quad (2.4)$$

is called the *basic reproduction number* of (1.4); if  $R_0 > 1$ ,  $I^*$  in (2.3) is positive. At the same time,  $I^*$  in (2.3) must satisfy  $I^* \leq I_0$ , which is equivalent to

$$u \geq \frac{kb(p\delta + \gamma + k)(R_0 - 1)}{\beta(bm + \gamma + k) + \alpha b(k + \gamma + \delta p)} = u_2. \quad (2.5)$$

Therefore,  $E_0(m, 0)$  is always the disease-free equilibrium of (1.4).  $E^*(S^*, I^*)$  is the endemic equilibrium of system (1.4) if and only if  $R_0 > 1$  and  $u \geq u_2$ , where

$$S^* = \frac{\alpha bm(p\delta + \gamma + k) + (bm + \gamma + k)(p\delta + \gamma + k)}{\beta(bm + \gamma + k) + \alpha b(p\delta + \gamma + k)}. \quad (2.6)$$

According to (2.2),  $I^*$  satisfies the following equation

$$b_0(I^*)^2 + b_1 I^* + b_2 = 0, \quad (2.7)$$

where

$$\begin{aligned} b_0 &= \beta(bm + \gamma) + \alpha b(p\delta + \gamma), \\ b_1 &= b(p\delta + \gamma - \beta m) + u(\alpha b + \beta), \\ b_2 &= bu. \end{aligned} \quad (2.8)$$

If  $b_1 \geq 0$ , it is clear that Eq. (2.7) does not have a positive solution. Let us suppose that  $b_1 < 0$ . Clearly,  $b_1 < 0$  is equivalent to

$$\beta m > p\delta + \gamma \quad \text{and} \quad u < \frac{b(\beta m - p\delta - \gamma)}{b\alpha + \beta}. \quad (2.9)$$

From (2.8), we get

$$\begin{aligned} \Delta &= b_1^2 - 4b_0 b_2 \\ &= (\alpha b + \beta)^2 u^2 - 2b\{(\beta m - p\delta - \gamma)(\alpha b + \beta) + 2[\beta(bm + \gamma) + \alpha b(p\delta + \gamma)]\}u + b^2(p\delta + \gamma - \beta m)^2. \end{aligned}$$

Note that

$$\begin{aligned} &\{-2b\{(\beta m - p\delta - \gamma)(\alpha b + \beta) + 2[\beta(bm + \gamma) + \alpha b(p\delta + \gamma)]\} \\ &\quad - 4(\alpha b + \beta)^2 b^2(p\delta + \gamma - \beta m)^2 \\ &= 4[\beta(bm + \gamma) + \alpha b(p\delta + \gamma)][\beta(bm + \gamma) + \alpha b(p\delta + \gamma) \\ &\quad + (\beta m - p\delta - \gamma)(\alpha b + \beta)] > 0. \end{aligned}$$

Denote

$$\eta = [\beta(bm + \gamma) + \alpha b(p\delta + \gamma)][\beta(bm + \gamma) + \alpha b(p\delta + \gamma) + (\beta m - p\delta - \gamma)(\alpha b + \beta)]^{\frac{1}{2}}.$$

It follows that  $\Delta \geq 0$  is equivalent to

$$u \geq \frac{b\{(\beta m - p\delta - \gamma)(\alpha b + \beta) + 2[\beta(bm + \gamma) + \alpha b(p\delta + \gamma)] + 2\eta\}}{(\alpha b + \beta)^2} \quad (2.10)$$

or

$$u \leq \frac{b\{(\beta m - p\delta - \gamma)(\alpha b + \beta) + 2[\beta(bm + \gamma) + \alpha b(p\delta + \gamma)] - 2\eta\}}{(\alpha b + \beta)^2} = u_0. \quad (2.11)$$

As  $\beta m > p\delta + \gamma$ , we have

$$u_0 < \frac{b(\beta m - p\delta - \gamma)}{\alpha b + \beta}.$$

Therefore,  $b_1 < 0$ ,  $\Delta \geq 0$  if and only if  $\beta m > p\delta + \gamma$  and  $u \leq u_0$ .

Suppose that  $u \leq u_0$  and  $\beta m > p\delta + \gamma$ , then (2.7) has two positive solutions  $I_1$  and  $I_2$ , where

$$I_1 = \frac{-b_1 - \sqrt{\Delta}}{2b_0}, \quad I_2 = \frac{-b_1 + \sqrt{\Delta}}{2b_0}. \quad (2.12)$$

Let

$$S_j = \frac{bm - u - (bm + \gamma)I_j}{b}.$$

If  $I_j > I_0$  ( $j = 1, 2$ ), then  $E_j(S_j, I_j)$  ( $j = 1, 2$ ) is an endemic equilibrium of (1.4).

By the expression of  $I_1$ , we notice that  $I_1 > I_0$  is equivalent to  $-\sqrt{\Delta} > 2b_0 I_0 + b_1$ .

This implies that  $2b_0 I_0 + b_1 < 0$ . It is easy to prove that  $2b_0 I_0 + b_1 < 0$  is equivalent to

$$u < \frac{bk(\beta m - p\delta - \gamma)}{2[\beta(bm + \gamma) + b\alpha(p\delta + \gamma)] + k(b\alpha + \beta)} = u_1. \quad (2.14)$$

Furthermore,  $I_1 > I_0$  requires that

$$(b_1 + 2b_0I_0)^2 - \Delta > 0.$$

Following some calculations, we obtain that

$$(b_1 + 2b_0I_0)^2 - \Delta = \frac{4b_0I_0}{k} \{ kb[p\delta + \gamma + k - \beta m] + [\beta(bm + \gamma + k) + \alpha b(p\delta + \gamma + k)]u \}.$$

- (1) If  $p\delta + \gamma < \beta m \leq p\delta + \gamma + k$ , then  $(b_1 + 2b_0I_0)^2 - \Delta > 0$ .
- (2) If  $\beta m > p\delta + \gamma + k$ , that is,  $R_0 > 1$ , then the inequality  $(b_1 + 2b_0I_0)^2 - \Delta > 0$  is equivalent to  $u > u_2$ .

Therefore,  $I_1 > I_0$  holds if and only if either  $u < u_1$ ,  $u \leq u_0$  and  $\beta m > p\delta + \gamma$  or  $u < u_1$ ,  $u > u_2$ ,  $u \leq u_0$  and  $R_0 > 1$ . Similarly,  $I_2 > I_0$  holds if and only if either  $u \leq \min\{u_0, u_1\}$  and  $\beta m > p\delta + \gamma$ , or  $u < \min\{u_0, u_2\}$ ,  $u > u_1$  and  $R_0 > 1$ .

Note that the sign of  $u_2 - u_1$  is determined by

$$\Phi = R_0 - R_0^*,$$

where

$$R_0^* = 1 + \frac{k(\beta(bm + \gamma + k) + \alpha b(p\delta + \gamma + k))}{(p\delta + \gamma + k)[\beta(bm + \gamma) + \alpha b(p\delta + \gamma)]}. \quad (2.15)$$

It follows that  $u_1 > u_2$  if  $1 < R_0 < R_0^*$ , and  $u_1 < u_2$  if  $R_0 > R_0^*$ .

Summarizing the above discussions, we have the following conclusions for the existence of equilibria.

**Theorem 2.1.** (i) For system (1.4), the disease-free equilibrium  $E_0(m, 0)$  always exists. (ii)  $E^*(S^*, I^*)$  is an endemic equilibrium of system (1.4) if and only if  $R_0 > 1$  and  $u \geq u_2$ . Furthermore, suppose  $R_0 > 1$ ,  $u \geq u_2$  and one of the following conditions is satisfied:

- (a)  $u > u_0$ ;
- (b)  $u_1 < u < u_0$ .

Then  $E^*$  is the unique endemic equilibrium of system (1.4).

**Theorem 2.2.** The endemic equilibria  $E_1(S_1, I_1)$  and  $E_2(S_2, I_2)$  of system (1.4) do not exist if  $\beta m \leq p\delta + \gamma$  or  $u > u_0$ . On the other hand, suppose  $u \leq u_0$  and  $\beta m > p\delta + \gamma$ , we have the following results.

- (i) If  $R_0 \leq 1$  and  $u < u_1$ , then the equilibria  $E_1$  and  $E_2$  of system (1.4) exist.
- (ii) If  $1 < R_0 < R_0^*$  and  $u_2 < u < u_1$ , then the equilibria  $E_1$  and  $E_2$  of system (1.4) exist.
- (iii) If  $1 < R_0 < R_0^*$  and  $u < u_2$ , then the equilibrium  $E_2$  of system (1.4) exists but  $E_1$  does not exist, and the equilibrium  $E_2$  does not exist if  $u > u_1$ .
- (iv) If  $1 < R_0^* < R_0$ , then  $E_1$  does not exist. Furthermore,  $E_2$  exists if  $u < u_2$  and does not exist if  $u > u_2$ .

We can see that under the conditions in Theorem 2.1 (i), the disease-free equilibrium  $E_0$  coexists with two endemic equilibria  $E_1$  and  $E_2$ . In fact, we have the following result.

**Corollary 2.3.** If  $R_0 < 1$ ,  $\beta m > p\delta + \gamma$ , and  $u < \min\{u_0, u_1\}$ , then system (1.4) has a backward bifurcation of endemic equilibria.

We now present examples to show that, for various parameter values, system (1.4) has a forward bifurcation from one endemic equilibrium to another endemic equilibrium (see Example 2.4)

and a backward bifurcation with a disease-free equilibrium and two endemic equilibria (Example 2.5). Note that by Theorem 2.1 (ii) and Theorem 2.2 (ii), there are conditions that guarantee the existence of all three endemic equilibria  $E_1, E_2$ , and  $E^*$  (Example 2.6).

**Example 2.4.** We choose the parameter values as follows:  $\beta = 0.34$ ,  $\alpha = 0.4$ ,  $\gamma = 0.01$ ,  $\delta = 0.01$ ,  $b = 0.2$ ,  $p = 0.02$ ,  $k = 0.03$  and  $m = 0.3$ . By calculations,  $R_0 = 2.53731$ ,  $R_0^* = 2.12826$ ,  $R_0 > R_0^*$  and  $u_2 = 0.00996346$ , case (iv) of Theorem 2.2 holds. When  $u \leq u_2$ , a bifurcation diagram is illustrated in Fig. 1, the bifurcation at  $u = u_2$  is forward when the parameter  $u$  decreases, and system (1.4) has a unique endemic equilibrium for all  $u > 0$ .

**Example 2.5.** We choose the parameter values as follows:  $\beta = 0.2$ ,  $\alpha = 0.4$ ,  $\gamma = 0.01$ ,  $\delta = 0.01$ ,  $b = 0.2$ ,  $p = 0.02$ ,  $k = 0.03$  and  $m = 0.1$ . By calculations, we have  $R_0 = 0.497512 < 1$ ,  $u_0 = 0.000590643$ ,  $u_1 = 0.00266885$ , so case (i) of Theorem 2.2 holds. A backward bifurcation diagram is illustrated in Fig. 2, where the horizontal line denotes the disease-free equilibrium  $E_0$ . Two endemic equilibria appear simultaneously at  $u = u_0$  when the parameter  $u$  decreases.

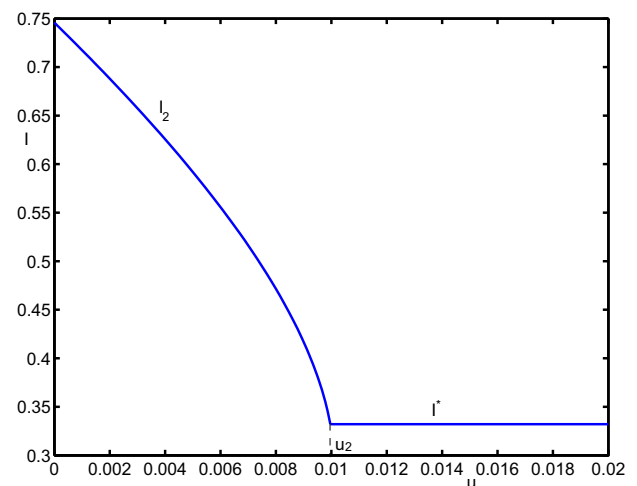


Fig. 1. The forward bifurcation diagram from  $I^*$  to  $I_2$  versus  $u$  for (1.4).

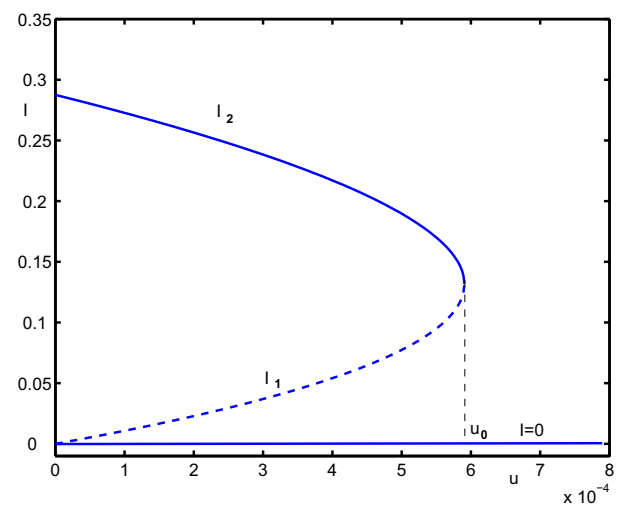


Fig. 2. The backward bifurcation diagram of  $I_1$  and  $I_2$  versus  $u$ .

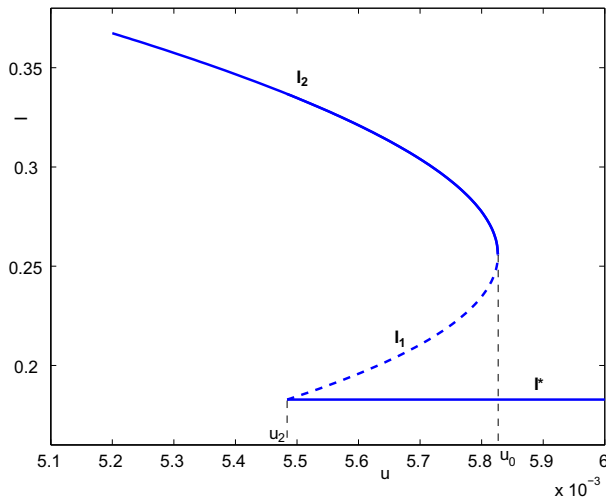


Fig. 3. The bifurcation diagram with multiple endemic equilibria versus  $u$ .

**Example 2.6.** Choose  $\beta = 0.34$ ,  $\alpha = 0.4$ ,  $\gamma = 0.01$ ,  $\delta = 0.01$ ,  $b = 0.2$ ,  $p = 0.02$ ,  $k = 0.03$  and  $m = 0.2$ . We have  $R_0 = 1.69154$ ,  $R_0^* = 2.27405$ ,  $u_0 = 0.00582652$ ,  $u_1 = 0.00719025$  and  $u_2 = 0.00548396$ , so case (ii) of Theorem 2.2 holds. A bifurcation diagram is illustrated in Fig. 3, where the horizontal line denotes the endemic equilibrium  $E^*$ . It shows that there is a bifurcation at  $u = u_0$  when the parameter  $u$  decreases, which gives rise to the existence of multiple endemic equilibria  $E^*$ ,  $E_1$ , and  $E_2$ .

### 3. Stability of equilibria

For the stability of the disease-free equilibrium  $E_0(m, 0)$ , we have the following theorem.

**Theorem 3.1.** The disease-free equilibrium  $E_0(m, 0)$  is locally asymptotically stable if  $R_0 < 1$  and unstable if  $R_0 > 1$ . Moreover,  $E_0(m, 0)$  is globally asymptotically stable in  $D$  if  $R_0 < 1$  and  $u > u_0$ .

**Proof.** It is easy to obtain that the characteristic roots to the linearized equation of system (1.4) at  $E_0(m, 0)$  are  $\lambda_1 = -b < 0$  and  $\lambda_2 = (p\delta + \gamma + k)(R_0 - 1)$ . Thus,  $E_0$  is locally asymptotically stable if  $R_0 < 1$  and unstable if  $R_0 > 1$ .

Next, if  $R_0 < 1$ ,  $E^*$  does not exist by Theorem 2.1. By Theorem 2.2,  $E_1$  and  $E_2$  do not exist if  $u > u_0$ . Therefore,  $E_0$  is the unique equilibrium of system (1.4). Since  $D$  is the invariant set of system (1.4) and  $E_0$  is locally asymptotically stable, it follows from Bendixson Theorem that every solution of system (1.4) in  $D$  approaches  $E_0$  when  $t$  tends to positive infinity.  $\square$

For the endemic equilibrium  $E^*(S^*, I^*)$ , we have the following theorem.

**Theorem 3.2.** If the endemic equilibrium  $E^*(S^*, I^*)$  of system (1.4) exists, then it is locally asymptotically stable.

**Proof.** According to Theorem 2.1, the endemic equilibrium  $E^*(S^*, I^*)$  exists if and only if  $R_0 > 1$  and  $u \geq u_2$ . The Jacobian matrix of system (1.4) at  $E^*(S^*, I^*)$  is

$$J^* = \begin{pmatrix} -b - \beta \frac{I^*}{1+\alpha I^*} & -\beta \frac{S^*}{(1+\alpha I^*)^2} - bm + p\delta \\ \beta \frac{I^*}{1+\alpha I^*} & \beta \frac{S^*}{(1+\alpha I^*)^2} - p\delta - \gamma - k \end{pmatrix}. \tag{3.1}$$

Because  $S^*$  and  $I^*$  satisfy Eq. (2.1), by means of (2.1), the trace and determinant of  $J^*$  are simplified into

$$\begin{aligned} \text{tr}(J^*) &= -\frac{b + [\beta + \alpha(b + p\delta + \gamma + k)]I^*}{1 + \alpha I^*} < 0, \\ \det(J^*) &= \frac{b\alpha(p\delta + \gamma + k) + \beta(bm + \gamma + k)}{1 + \alpha I^*} I^* > 0. \end{aligned}$$

Therefore, all eigenvalues of matrix  $J^*$  have negative real parts when  $R_0 > 1$ ,  $u \geq u_2$ . It follows that  $E^*(S^*, I^*)$  is locally asymptotically stable.  $\square$

Now we discuss the stability of endemic equilibria  $E_j(S_j, I_j)$  ( $j = 1, 2$ ). The Jacobian matrix of system (1.4) at  $E_j(S_j, I_j)$  is

$$J_j = \begin{pmatrix} -b - \beta \frac{I_j}{1+\alpha I_j} & -\beta \frac{S_j}{(1+\alpha I_j)^2} - bm + p\delta \\ \beta \frac{I_j}{1+\alpha I_j} & \beta \frac{S_j}{(1+\alpha I_j)^2} - p\delta - \gamma \end{pmatrix}, \quad j = 1, 2. \tag{3.2}$$

**Theorem 3.3.** If the endemic equilibrium  $E_1(S_1, I_1)$  of system (1.4) exists, then it is unstable.

**Proof.** For the endemic equilibrium  $E_1(S_1, I_1)$ ,  $S_1$  and  $I_1$  satisfy (2.2). By (2.2), (2.7) and (2.12), after some calculations, the determinant of the matrix  $J_1$  is

$$\det(J_1) = \frac{-\sqrt{\Delta}}{1 + \alpha I_1} < 0. \tag{3.3}$$

Therefore,  $E_1(S_1, I_1)$  is a saddle and unstable.  $\square$

For the endemic equilibrium  $E_2(S_2, I_2)$ , similarly, the determinant of  $J_2$  is

$$\det(J_2) = \frac{\sqrt{\Delta}}{1 + \alpha I_2} > 0. \tag{3.4}$$

Thus,  $E_2$  may be a node, focus, or center.

By (2.2), (2.7) and (2.12), the trace  $\text{tr}(J_2)$  of matrix  $J_2$  is very complicated, but the sign of  $\text{tr}(J_2)$  is determined by

$$\begin{aligned} \varphi &= -2bb_0b_2 + b_1u[b(\beta + b\alpha) - \beta(bm + \gamma)] \\ &\quad - [b_2(\beta + \alpha(b + p\delta + \gamma)) + b_0u]\sqrt{\Delta}. \end{aligned} \tag{3.5}$$

- (1) According to  $b_1 < 0$  and (3.5), if  $b(\beta + b\alpha) \geq \beta(bm + \gamma)$ , then  $\varphi < 0$ .
- (2) If  $b(\beta + b\alpha) < \beta(bm + \gamma)$ , because

$$\begin{aligned} &-2bb_0b_2 + b_1u[b(\beta + b\alpha) - \beta(bm + \gamma)] \\ &= u \left\{ -2b^2[\beta(bm + \gamma) + b\alpha(p\delta + \gamma)] + b(p\delta + \gamma - m\beta)[b(\beta + b\alpha) - \beta(bm + \gamma)] \right. \\ &\quad \left. + (b\alpha + \beta)[b(\beta + b\alpha) - \beta(bm + \gamma)]u \right\}, \end{aligned} \tag{3.6}$$

it is known from (3.6) that if

$$0 < \beta m - p\delta - \gamma \leq \frac{2b[\beta(bm + \gamma) + b\alpha(p\delta + \gamma)]}{\beta(bm + \gamma) - b(\beta + b\alpha)} = \eta_1, \tag{3.7}$$

$$u \geq \frac{-2b^2[\beta(bm + \gamma) + b\alpha(p\delta + \gamma)] + b(p\delta + \gamma - m\beta)[b(\beta + b\alpha) - \beta(bm + \gamma)]}{(b\alpha + \beta)[\beta(bm + \gamma) - b(\beta + b\alpha)]} = u_3, \tag{3.8}$$

then  $\varphi < 0$ .

(3) It follows from (3.6) that if  $b(\beta + b\alpha) < \beta(bm + \gamma)$ ,  $\beta m - p\delta - \gamma > \eta_1$  and then  $\varphi < 0$ .

(4) It is known from (3.5) and (3.6) that if  $b(\beta + b\alpha) < \beta(bm + \gamma)$ ,  $\beta m - p\delta - \gamma > \eta_1$ ,  $u < u_3$  and

$$\chi = \Delta - \frac{\{-2bb_0b_2 + b_1u[b(\beta + b\alpha) - \beta(bm + \gamma)]\}^2}{[b_2(\beta + \alpha(b + p\delta + \gamma)) + b_0u]^2} > 0, \tag{3.9}$$

then  $\varphi < 0$ .

Summarizing the above discussions, we have the following results on the stability of the equilibrium  $E_2(S_2, I_2)$ .

**Theorem 3.4.** Suppose that the endemic equilibrium  $E_2(S_2, I_2)$  exists, if one of following conditions is satisfied:

- (i)  $b(\beta + b\alpha) \geq \beta(bm + \gamma)$ ;
- (ii)  $b(\beta + b\alpha) < \beta(bm + \gamma)$  and  $\beta m - p\delta - \gamma \leq \eta_1$ ;
- (iii)  $b(\beta + b\alpha) < \beta(bm + \gamma)$ ,  $\beta m - p\delta - \gamma > \eta_1$  and  $u \geq u_3$ ;
- (iv)  $b(\beta + b\alpha) < \beta(bm + \gamma)$ ,  $\beta m - p\delta - \gamma > \eta_1$ ,  $u < u_3$  and  $\chi > 0$ ,

then  $E_2(S_2, I_2)$  is locally asymptotically stable. It is unstable if  $b(\beta + b\alpha) < \beta(bm + \gamma)$ ,  $\beta m - p\delta - \gamma > \eta_1$ ,  $u < u_3$  and  $\chi < 0$ .

The existence of limit cycles plays an important role in determining the dynamical behaviors of the system. For example, if there is no limit cycle in system (1.4) and its endemic equilibrium is unique and locally asymptotically stable, then it must be globally stable. Now, we consider the existence of limit cycles in system (1.4).

**Theorem 3.5.** Suppose  $R_0 > R_0^*$  and  $u < \min\{u_0, u_2\}$ . If  $\varphi > 0$ , then system (1.4) has at least a stable limit cycle which encircles  $E_2$ .

**Proof.** As  $R_0 > R_0^* > 1$  and  $u < u_2$ , it is known from Theorem 2.1 that the equilibrium  $E^*$  of system (1.4) does not exist. Again, because  $R_0 > R_0^*$ ,  $u < u_0$  and  $u < u_2$ , it follows from Theorem 2.2 that the equilibrium  $E_1$  of system (1.4) does not exist, but the equilibrium  $E_2$  exists.

It is known from  $\varphi > 0$  that  $E_2$  is an unstable focus or node. It is easy to check that the unstable manifold at  $E_0(m, 0)$  which is a saddle point, is in the first quadrant. As the set  $D$  is positively invariant for system (1.4), and system (1.4) does not have any

equilibrium in the interior of  $D \setminus \{E_2\}$ . It follows from Poincaré–Bendixon theorem that system (1.4) has at least a stable limit cycle which encircles  $E_2$ .  $\square$

#### 4. Numerical simulations

In this section, we present some numerical simulations of system (1.4) to illustrate our results.

**Example 4.1** (Example 2.4 continued). Choose  $\beta = 0.34, \alpha = 0.4, \gamma = 0.01, \delta = 0.01, b = 0.2, p = 0.02, k = 0.03$  and  $m = 0.3$ . We have  $R_0 = 2.53731, R_0^* = 2.12826, R_0 > R_0^*, u_0 = 0.0101133$  and  $u_2 = 0.00996346$ . A forward bifurcation diagram was given in Fig. 1.

If we select  $I_0 = 0.4$ , then  $u = 0.012 > u_2$ , the equilibrium  $E^*(0.133942, 0.332115)$  exists, but  $E_1$  and  $E_2$  do not exist (see Theorem 2.1(ii)). Its phase portrait is given in Fig. 4, which shows that the unique equilibrium  $E^*$  is globally asymptotically stable.

If we choose  $I_0 = 0.2$ , then  $u = 0.006 < u_2$ , the equilibrium  $E_2(0.0754805, 0.55577)$  exists, but  $E_1$  and  $E^*$  do not exist (see Theorem 2.2(iv)). Its phase portrait is illustrated in Fig. 5. The unique equilibrium  $E_2$  is globally asymptotically stable in  $D$ .

**Example 4.2** (Example 2.5 continued). Choose  $\beta = 0.2, \alpha = 0.4, \gamma = 0.01, \delta = 0.01, b = 0.2, p = 0.02, k = 0.03$  and  $m = 0.1$ . By calculations,  $R_0 = 0.497512 < 1, u_0 = 0.000590643, u_1 = 0.00266885$  and  $R_0 < 1$ . A backward bifurcation diagram was given in Fig. 2. If we choose  $I_0 = 0.01$ , then  $u = 0.0003 < u_0$ , the equilibria  $E_1(0.0929588, 0.036941)$  and  $E_2(0.060531, 0.238294)$  exist, but the equilibrium  $E^*$  does not exist. Its phase portrait is illustrated in Fig. 6. It shows that the equilibria  $E_0$  and  $E_2$  are asymptotically stable.

**Example 4.3** (Example 2.6 continued). For system (1.4), we choose parameter values as follows:  $\beta = 0.34, \alpha = 0.4, \gamma = 0.01, \delta = 0.01, b = 0.2, p = 0.02, k = 0.03, m = 0.2$  and  $I_0 = 0.1867$ . Then we have  $R_0 = 1.69154, R_0^* = 2.27405, b(\beta + b\alpha) - \beta(bm + \gamma) = 0.067 > 0, u_0 = 0.00582652, u_1 = 0.00719025, u_2 = 0.00548396$  and  $u = 0.0056$ . The parameter values satisfy the conditions of Theorem 3.2, the condition (ii) of Theorem 2.2 and the condition (i) of Theorem 3.4. Therefore, all four equilibria  $E_0(0.2, 0), E^*(0.126881, 0.182799), E_1(0.123039, 0.195843)$  and

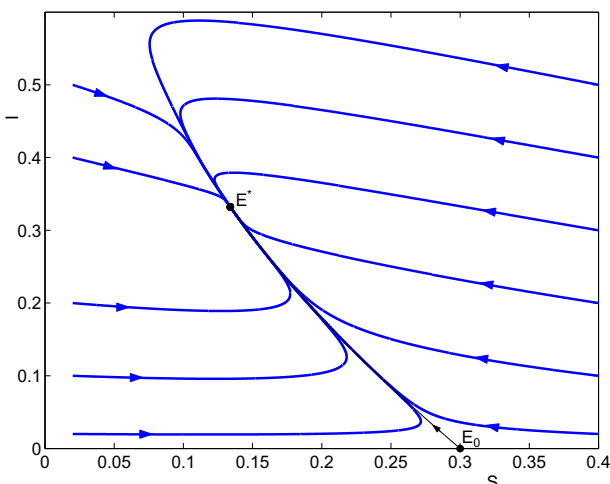


Fig. 4. The phase portrait of system (1.4) when  $E^*$  is globally asymptotically stable.

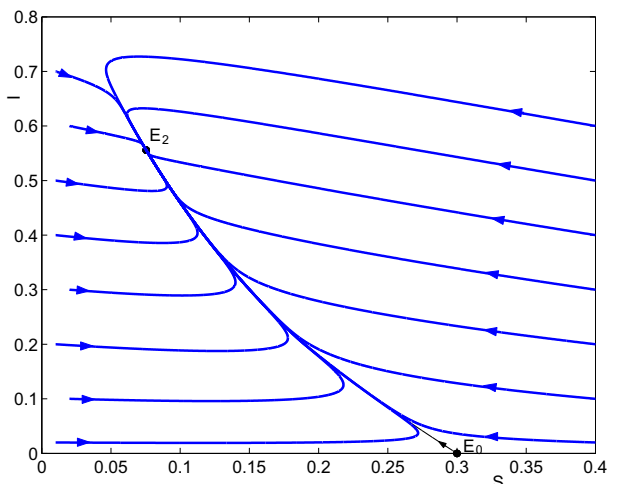


Fig. 5. The phase portrait of system (1.4) when  $E_2$  is globally asymptotically stable.

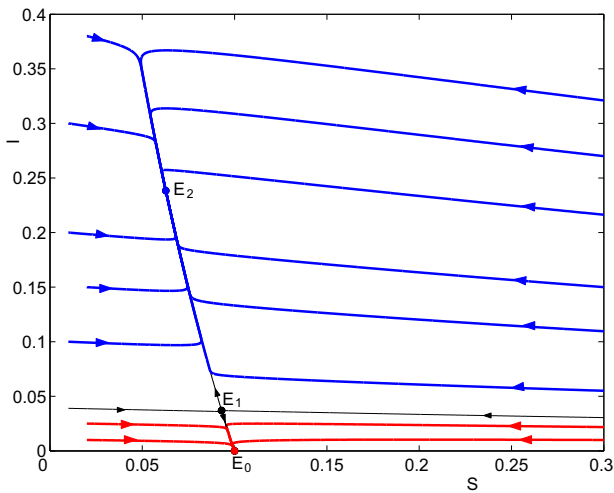


Fig. 6. The phase portrait of system (1.4) when  $E_2$  and  $E_0$  are stable and  $E_1$  is unstable.

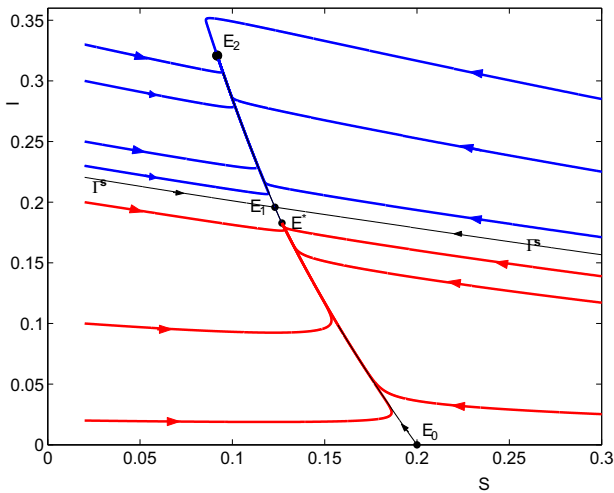


Fig. 7. The phase portrait of system (1.4) with bistable endemic equilibria.

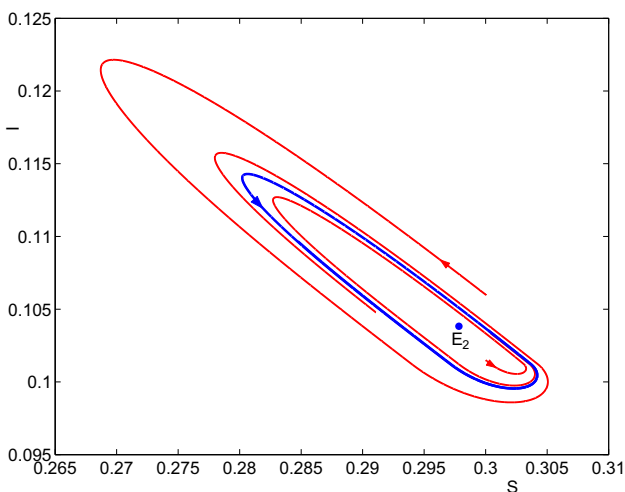


Fig. 8. A stable limit cycle of system (1.4) encircling the unstable equilibrium  $E_2$ .

$E_2(0.0917512, 0.320995)$  exist. The phase portrait of system (1.4) is illustrated in Fig. 7, where the black thin lines are the separatrices of saddle points  $E_0$  and  $E_1$ . It is known from Fig. 7 that the equilibria  $E_2$  and  $E^*$  are asymptotically stable, and  $E_0$  and  $E_1$  are unstable. Thus, system (1.4) has bistable endemic equilibria  $E^*$  and  $E_2$ .

We can see from Fig. 7 that the stable separatrices  $\Gamma^s$  of saddle point  $E_1$  separate the positive invariant set  $D$  into two regions, the basin of attraction for the stable equilibrium  $E_2$  is the region above  $\Gamma^s$  and the basin of attraction for the stable equilibrium  $E^*$  is the region below  $\Gamma^s$ .

**Example 4.4.** Choose  $\beta = 0.2, \alpha = 0.8, \gamma = 0.01, \delta = 0.03, b = 0.01, p = 0.2, k = 0.04$  and  $m = 0.9$ . We have  $R_0 = 3.21429, R_0^* = 3.22723, u_0 = 0.00404965, u_1 = 0.00405539$  and  $u_2 = 0.00404964$ . If we choose  $I_0 = 0.101225$ , then  $u = 0.004049 < u_0, \text{tr}(J_2) = 0.00407523 > 0$ , the equilibria  $E_2(0.29783, 0.103826)$  exists but is unstable, and the equilibria  $E^*$  and  $E_1$  do not exist. The parameter values satisfy the conditions of Theorem 3.5. Its phase portrait is given in Fig. 8, which shows that system (1.4) has a stable limit cycle which encircles  $E_2$ . Therefore, under some conditions, system (1.4) has a stable periodic orbit which encircles the equilibrium  $E_2$ .

### 5. Discussion

In this paper, we have analyzed a SIR epidemic model to study the effect of limited resources for the treatment of patients in the public-health system, which could occur when there is a very large number of patients but the medical facilities are insufficient, the number of beds is limited, or the number of health-care workers is short-handed. We also considered nonlinear incidence rate, vertical transmission and vaccination for the newborns of the susceptible and recovered individuals in the model. Theorem 2.2 and Corollary 2.3 imply that a backward bifurcation occurs when  $R_0 < 1$ , that is, the disease-free equilibrium coexists with an endemic equilibrium. Theorems 2.2 and 3.4 indicate that system (1.4) has multiple endemic equilibrium when  $R_0 > 1$  where a bifurcation diagram displays forward bifurcations. When there are two endemic equilibria, one of them is always unstable and the other one is stable under certain conditions. When there are three endemic equilibria, bistable endemic equilibria can occur. Numerical simulation confirmed that system (1.4) has a stable periodic orbit which encircles an endemic equilibrium under some conditions. The existence and stability of equilibria of system (1.4) can be summarized in Table 1 (DNE = does not exist).

We can see that when the basic reproduction number  $R_0 < 1$  and the treatment term  $u < \min\{u_0, u_1\}$ , a backward bifurcation occurs with a disease-free equilibrium and two endemic equilibria. Note that the disease-free equilibrium is always stable since  $R_0 < 1$ , the stability of the endemic equilibria depends on other conditions. Recall that  $u = kI_0$ , where  $k$  is the treatment parameter and  $I_0$  represents the infective level at which the health-care system reaches capacity,

$$R_0 = \frac{\beta m}{p\delta + \gamma + k},$$

$$u_0 = \frac{b\{(\beta m - p\delta - \gamma)(\alpha b + \beta) + 2[\beta(bm + \gamma) + \alpha b(p\delta + \gamma)] - 2\eta\}}{(\alpha b + \beta)^2},$$

$$u_1 = \frac{bk(\beta m - p\delta - \gamma)}{2[\beta(bm + \gamma) + b\alpha(p\delta + \gamma)] + k(b\alpha + \beta)},$$



**Table 1**  
Existence and stability of equilibria for system (1.4).

Conditions	$E_0$	$E^*$	$E_1$	$E_2$	Figure
$R_0 < 1$	Stable	DNE	DNE	DNE	
$u < \min\{u_0, u_1\}$	Stable	DNE	Unstable	Stable	Fig. 6
$R_0 > 1$	Unstable	Stable	DNE	DNE	Fig. 4
$R_0 < R_0^*$	Unstable	Stable	Unstable	Stable	Fig. 7
$R_0^* < R_0$	Unstable	DNE	DNE	Stable	Fig. 5
				Unstable	Fig. 8

and

$$u_2 = \frac{kb(p\delta + \gamma + k)(R_0 - 1)}{\beta(bm + \gamma + k) + \alpha b(k + \gamma + \delta p)}.$$

Therefore, in order to eradicate the disease, the basic reproduction number  $R_0$  must be lowered than a threshold or the treatment  $u > \max\{u_0, u_1\}$  by other control measures, possibly a combination of different types of measures. Epidemic models with two different types of interventions have been studied by some other researchers, see Brauer [6] and Sun and Yang [32]. For example, reduce the rate of vertical transmission rate  $q(=1-p)$ , increase the vaccination rate  $m'$ , or increase the treatment rate  $k$ , so that the disease approaches a lower endemic steady state for a range of parameters. If the capacity for treatment increases further to a certain level, then the disease may be eradicated.

We would like to mention that related models have been studied and similar results have been obtained by other researchers, for example, Cui et al. [10], Li et al. [25], and Hu et al. [22]. However, we have considered more factors and components in our model and obtained different and new results. For example, we assumed that the disease can be transmitted vertically and vaccination applies to the newborns of susceptible and recovered individuals. Moreover, we established the existence and stability of a limit cycle when there is a unique unstable endemic equilibrium.

As pointed out by Greenhalgh and Griffiths [15], the study of backward bifurcation in epidemic modeling is relatively new and there are many issues deserving further investigation for this new and interesting phenomenon. So far most studies focus on the theoretical aspects of backward bifurcation and very few relate to real communicable diseases (see Greenhalgh and Griffiths [15] on a Bovine Respiratory Syncytial virus epidemic model and Blayneh et al. [4] and Wan and Zhu [35] on West Nile virus epidemic models). As mentioned in the Introduction, we started the project with modeling the SARS outbreaks in 2003 [38]. The second SARS outbreak in Toronto, Canada in 2003 (see Gumel et al. [16]) may be explained as a result of backward bifurcation since the basic reproduction number was certainly less than unity after the first outbreak when restrictive and suitable infection-control procedures were being taken [19]. We expect that our results in this paper might be helpful to study the endemic of hepatitis B virus in China [43] and the outbreaks of cholera in Haiti or Zimbabwe [28]. We propose to study these in the future.

**Acknowledgments**

We are very grateful to the referees and the handling editor for their helpful comments and suggestions. We also would like to thank Professor Wendi Wang for helpful discussions.

**References**

[1] M.E. Alexander, S.M. Moghadas, Periodicity in an epidemic model with a generalized non-linear incidence, *Math. Biosci.* 189 (2004) 75.  
 [2] R.M. Anderson, R.M. May, *Infectious Diseases of Humans*, Oxford University Press, London, 1991.

[3] J. Arino, C.C. McCluskey, P. van den Driessche, Global results for an epidemic model with vaccination that exhibits back bifurcation, *SIAM J. Appl. Math.* 64 (2003) 260.  
 [4] K.W. Blayneh, A.B. Gumel, S. Lenhart, T. Clayton, Backward bifurcation and optimal control in transmission dynamics of West Nile virus, *Bull. Math. Biol.* 72 (2010) 1006.  
 [5] F. Brauer, Backward bifurcations in simple vaccination models, *J. Math. Anal. Appl.* 298 (2004) 418.  
 [6] F. Brauer, Backward bifurcations in simple vaccination/treatment models, *J. Biol. Dynam.* 5 (2011) 410.  
 [7] V. Capasso, G. Serio, A generalization of the Kermack–McKendrick deterministic epidemic model, *Math. Biosci.* 42 (1978) 41.  
 [8] C. Castillo-Chavez, K. Cooke, W. Huang, S.A. Levin, The role of long incubation periods in the dynamics of HIV/AIDS. Part 1: Single population models, *J. Math. Biol.* 27 (1989) 373.  
 [9] C. Castillo-Chavez, K. Cooke, W. Huang, S.A. Levin, The role of long incubation periods in the dynamics of HIV/AIDS. Part 2: Multiple group models, in: C. Castillo-Chavez (Ed.), *Mathematical and Statistical Approaches to AIDS Epidemiology*, Lecture Notes in Biomathematics, vol. 83, Springer-Verlag, 1989, pp. 200–217.  
 [10] J. Cui, X. Mu, H. Wan, Saturation recovery leads to multiple endemic equilibria and backward bifurcation, *J. Theor. Biol.* 254 (2008) 275.  
 [11] J.C. Eckalbar, W.L. Eckalbar, Dynamics of an epidemic model with quadratic treatment, *Nonlinear Anal. RWA* 12 (2011) 320.  
 [12] W.R. Derrick, P. van den Driessche, Homoclinic orbits in a disease transmission model with nonlinear incidence and nonconstant population, *Discrete Contin. Dynam. Syst. Ser. B* 2 (2003) 299.  
 [13] J. Dushoff, W. Huang, C. Castillo-Chavez, Backwards bifurcations and catastrophe in simple models of fatal diseases, *J. Math. Biol.* 36 (1998) 227.  
 [14] Z. Feng, H.R. Thieme, Recurrent outbreaks of childhood disease revisited: the impact of isolation, *Math. Biosci.* 128 (1995) 93.  
 [15] D. Greenhalgh, M. Griffiths, Backward bifurcation, equilibrium and stability phenomena in a three-stage extended BRSV epidemic model, *J. Math. Biol.* 59 (2009) 1.  
 [16] A.B. Gumel, S. Ruan, T. Day, J. Watmough, F. Brauer, P. van den Driessche, D. Gabrielson, C. Bowman, M.E. Alexander, S. Ardal, J. Wu, B.M. Sahai, Modeling strategies for controlling SARS outbreaks, *Proc. R. Soc.* 271B (2004) 2223.  
 [17] K.P. Haderler, C. Castillo-Chavez, A core group model for disease transmission, *Math. Biosci.* 128 (1995) 41.  
 [18] K.P. Haderler, P. van den Driessche, Backward bifurcation in epidemic in control, *Math. Biosci.* 146 (1997) 15.  
 [19] Health Canada, *Learning from SARS – Renewal of Public Health in Canada*, Health Canada, Ottawa, 2003.  
 [20] H.W. Hethcote, The mathematics of infectious disease, *SIAM Rev.* 42 (2000) 599.  
 [21] Z. Hu, P. Bi, W. Ma, S. Ruan, Bifurcations of an SIRS epidemic model with nonlinear incidence rate, *Discrete Contin. Dynam. Syst. Ser. B* 18 (2011) 93.  
 [22] Z. Hu, S. Liu, H. Wang, Backward bifurcation of an epidemic model with standard incidence rate and treatment rate, *Nonlinear Anal. RWA* 9 (2008) 2302.  
 [23] W. Huang, K.L. Cooke, C. Castillo-Chavez, Stability and bifurcation for a multiplegroup model for the dynamics of HIV/AIDS transmission, *SIAM J. Appl. Math.* 52 (1992) 835.  
 [24] C.M. Kribs-Zaleta, J.X. Velasco-Hernandez, A simple vaccination model with multiple endemic states, *Math. Biosci.* 164 (2000) 183.  
 [25] X.-Z. Li, W.-S. Li, M. Ghosh, Stability and bifurcation of an SIR epidemic model with nonlinear incidence and treatment, *Appl. Math. Comput.* 210 (2009) 141.  
 [26] W.M. Liu, H.W. Hethcote, S.A. Levin, Influence of nonlinear incidence rates upon the behavior of SIRS epidemiological model, *J. Math. Biol.* 23 (1986) 187.  
 [27] W.M. Liu, H.W. Hethcote, S.A. Levin, Dynamical behavior of epidemiological models with nonlinear incidence rates, *J. Math. Biol.* 25 (1987) 359.  
 [28] Z. Mukandavire, S. Liao, J. Wang, H. Gaff, D.L. Smith, J. Glenn Morris Jr., Estimating the reproductive numbers for the 2008–2009 cholera outbreaks in Zimbabwe, *Proc. Natl. Acad. Sci. USA* 108 (2011) 8767–8772.  
 [29] T.C. Reluga, J. Medlock, A.S. Perelson, Backward bifurcations and multiple equilibria in epidemic models with structured immunity, *J. Theor. Biol.* 252 (2008) 155.  
 [30] S. Ruan, W. Wang, Dynamical behavior of an epidemic model with a nonlinear incidence rate, *J. Different. Equat.* 188 (2003) 135.  
 [31] M. Safan, H. Heesterbeek, K. Dietz, The minimum effort required to eradicate infections in models with backward bifurcation, *J. Math. Biol.* 53 (2006) 703.

- [32] C. Sun, W. Yang, Global results for an SIRS model with vaccination and isolation, *Nonlinear Anal. RWA* 11 (2010) 4223.
- [33] Y. Tang, D. Huang, S. Ruan, W. Zhang, Coexistence of limit cycles and homoclinic loops in a SIRS model with a nonlinear incidence rate, *SIAM J. Appl. Math.* 69 (2008) 621.
- [34] P. van den Driessche, J. Watmough, A simple SIS epidemic model with a backward bifurcation, *J. Math. Biol.* 40 (2000) 525.
- [35] H. Wan, H. Zhu, The backward bifurcation in compartmental models for West Nile virus, *Math. Biosci.* 227 (2010) 20.
- [36] W. Wang, Backward bifurcation of an epidemic model with treatment, *Math. Biosci.* 201 (2006) 58.
- [37] W. Wang, S. Ruan, Simulating the SARS outbreak in Beijing with limited data, *J. Theor. Biol.* 227 (2004) 369.
- [38] W. Wang, S. Ruan, Bifurcation in an epidemic model with constant removal rate of the infectives, *J. Math. Anal. Appl.* 291 (2004) 775.
- [39] D. Xiao, S. Ruan, Global analysis of an epidemic model with nonmonotone incidence rate, *Math. Biosci.* 208 (2007) 419.
- [40] X. Zhang, X. Liu, Backward bifurcation of an epidemic model with saturated treatment function, *J. Math. Anal. Appl.* 348 (2008) 433.
- [41] X. Zhang, X. Liu, Backward bifurcation and global dynamics of an SIS epidemic model with general incidence rate and treatment, *Nonlinear Anal. RWA* 10 (2009) 565.
- [42] L. Zhou, M. Fan, Dynamics of an SIR epidemic model with limited medical resources revisited, *Nonlinear Anal. RWA* 13 (2012) 312.
- [43] L. Zou, W. Zhang, S. Ruan, Modelling the transmission dynamics and control of Hepatitis B virus in China, *J. Theor. Biol.* 262 (2009) 330.