A bifurcation study on epidemic models with density-dependent treatments

Xiaoguang Zhang[1], Baojun Song[2]

[1] Department of Mathematics, North University of China,

Shanxi, Taiyuan, 030051, People's Republic of China

[2] Department of Mathematical Sciences, Montclair State University,

Montclair, NJ 07043, USA

Abstract

Numerically studies have shown that the bi-stability and backward bifurcation are not automatically connected in epidemic models. In fact, when a backward bifurcation occurs, the disease-free equilibrium may be globally stable or it may support to a stable limit cycle. In this paper, a continuous epidemic model that incorporates density-dependent treatments is analyzed. It is shown that it supports new types of backward bifurcations. We proceed to find bifurcation curves in a subset of entire parameter space. Finally, we analyze an analog discrete-time epidemic model.

Keywords: Epidemic models, Bifurcation, Density-dependent treatments, Dynamic models

1 Introduction

More and more mathematical models describing the dynamics of human infectious diseases are proposed to understand the mechanism of disease transmission. This is necessary and useful to give tutorial control measures in epidemiology. Usually, classical epidemic models only have a stable disease-free equilibrium when $R_0 < 1$ and which is unstable when $R_0 > 1$; and the unique stable endemic equilibrium exist when the basic reproduction number $R_0 > 1$, hence the bifurcation at $R_0 = 1$ is forward[1, 2, 3]. Some epidemic models, however, exhibit more complicated behaviors. Another equilibrium may bifurcate from the disease-free equilibrium when $R_0 < 1$. This is so called backwards bifurcation. In this case, the basic reproduction number alone cannot be used to describe disease elimination effort. Then it is important and necessary to investigate backward bifurcations and establish thresholds for the control of diseases.

There are a lot of works that studied the backward bifurcations. For instance, the works in [13, 14, 15, 16, 17, 18, 19, 22, 23] successfully used the continuous epidemic models to investigate the dynamical behavior when backward bifurcation occurs. The work in [13] provided a general framework for the mechanisms behind backwards bifurcations in simple disease models and discussed the biological interpretation of the features of the model that produce these bifurcations. Paper [14] studied the global behavior of an epidemic model with a saturated incidence rate $\frac{kI^2S}{1+\alpha I^2}$, and showed that, although the reproduction number is zero, the disease can still persist under some conditions. Paper [18] demonstrated the existence of backward bifurcations in SIR models

with bilinear incidence βSI and piecewise treatment $T(I)$, which is

$$T(I) = \begin{cases} \gamma I, & \text{if } 0 \geq I \leq I_0, \\ k, & \text{if } I > I_0. \end{cases} \tag{1.1}$$

Following [16] modified the incidence as standard incidence $\frac{\beta SI}{N}$ and adopted the same piecewise treatment function in [18]. This model can also lead to the backward bifurcation. [17] modified the model with the nonlinear incidence function $\frac{\lambda SI}{1+\alpha I}$ and showed the dynamic behavior when backward bifurcation occurs. The work in [19] used nonlinear incidence function $\frac{\lambda SI}{1+\alpha I}$ and the saturated treatment function $\frac{\gamma I}{1+\alpha I}$ and showed that the bifurcation can occur. These models all found that there exist bistable equilibria when a backward bifurcation occurs.

We can see that the particular treatment function can lead to backward bifurcation and it seems that a backward bifurcation can lead to bistable dynamics (called Type-I backward bifurcation [23]).

Although a lot of works on backward bifurcation have been done, the general conditions for the occurrence of a backward bifurcation have not been found. The dynamic behaviors of the system when a backward bifurcation occurs are still not completely clear. Paper [12] gave an criterion of the occurrence of a backward bifurcation. [15] proposed an SIS model with bilinear incidence βSI and saturated treatment function $\frac{cI}{b+I}$ and showed the existence of backward bifurcation. It was shown that the oscillations occur when the basic reproduction number $R_0 < 1$. Furthermore, [23] introduced a general form for the treatment function is $T(I) = p(I)I$, and gave some general results

to SIS models with standard incidence $\frac{\beta SI}{N}$. For particular $p(I)$ backward bifurcation can occur. Specifically, the two density-dependent treatment functions $p(I) = \alpha_1 e^{-\gamma I}$ and $p(I) = \alpha_2 + \alpha_1 e^{-\gamma I}$ were used. If $p(I) = \alpha_1 e^{-\gamma I}$, the disease-free equilibrium coexists with an endemic equilibrium when $R_0 < 1$ and an addition condition $R_1 < 1$. It is called Type-I backward bifurcation[23]. However, if $p(I) = \alpha_2 + \alpha_1 e^{-\gamma I}$, two new types of dynamics were found through numerical simulations.

(1) When a backward bifurcation occurs, the disease-free equilibrium is globally stable. One positive equilibrium is an unstable spiral and the other is a saddle; there is a heteroclinic cycle orbit that connects the saddle with the disease-free equilibrium. This is called Type-II backward bifurcation [23].

(2) When a backward bifurcation occurs, the disease may persist in a periodic fashion. Neither positive equilibrium is stable, and there is a stable limit cycle. This is called Type-III backward bifurcation [23].

These new dynamic behaviors show that backward bifurcations cannot always generate bi-stability. The existence of Type-II and Type-III backward bifurcations were discovered numerically. In this paper, we study the same model and show these bifurcations theoretically.

The rest of this paper is organized as follows: In section 2 we give a continuous SIS epidemic model with density-dependent treatments; Section 3 focuses on the analysis of the continuous model, including the stability of equilibria, the different type of backward bifurcations, saddle-node bifurcation and hopf bifurcation; Section 4 studies a corresponding discrete-time epidemic model; Finally, in Section 5, we collect some

observations and conclusions.

2 Continuous-time model with density-dependent treatments

As a review to [23], the epidemic models and some results are relisted below since these will be used in the later analysis.

The model based on the SIS model with the standard incidence and treatment function $p(I) = \alpha_2 + \alpha_1 e^{-\gamma I}$ takes the following form

$$\frac{dS}{dt} = \Lambda - \beta S\frac{I}{N} - \mu S + (\alpha_2 + \alpha_1 e^{-\gamma I})I, \tag{2.1}$$

$$\frac{dI}{dt} = \beta S\frac{I}{N} - (\mu + d)I - (\alpha_2 + \alpha_1 e^{-\gamma I})I, \tag{2.2}$$

$$N = S + I, \tag{2.3}$$

where all the parameters are positive, and Λ is the recruitment of susceptible population; μ the natural death rate; d the additional death rate caused by the disease. The infected individuals are treated with a rate $(\alpha_2 + \alpha_1 e^{-\gamma I})I$. The standard incidence rate $\beta S\frac{I}{N}$ is used, where β is transmission rate.

Model (2.1)-(2.3) always has a disease-free equilibrium $E_0 = (\frac{\Lambda}{\mu}, 0)$. We use the next generation method [21, 20] to obtain the basic reproduction number

$$R_0 = \frac{\beta}{\mu + d + \alpha_1 + \alpha_2}. \tag{2.4}$$

The Jacobian matrix of the system (2.1)-(2.3) is

$$J = \begin{pmatrix} -\beta\frac{I^2}{(S+I)^2} - \mu & -\beta\frac{S^2}{(S+I)^2} + \alpha_2 + \alpha_1 e^{-\gamma I} - \gamma\alpha_1 I e^{-\gamma I} \\ \beta\frac{I^2}{(S+I)^2} & \beta\frac{S^2}{(S+I)^2} - \mu - d - \alpha_2 - \alpha_1 e^{-\gamma I} + \gamma\alpha_1 I e^{-\gamma I} \end{pmatrix}.$$

so the matrix of linearization of (2.1)-(2.3) at the disease-free equilibrium is

$$J(E_0) = \begin{pmatrix} -\mu & -\beta + \alpha_1 + \alpha_2 \\ 0 & \beta - \mu - d - \alpha_1 - \alpha_2 \end{pmatrix}.$$

The characteristic equation at E_0 is

$$(\lambda + \mu)(\lambda + (\mu + d + \alpha_1 + \alpha_2)(1 - R_0)) = 0, \tag{2.5}$$

all the eigenvalues have negative real parts, implying the asymptotic stability of the disease-free equilibrium, if and only if $R_0 < 1$. Hence we have the following result

Theorem 2.1. *For model (2.1)-(2.3), the disease-free equilibrium E_0 is locally asymptotically stable if $R_0 < 1$, and is unstable when $R_0 > 1$.*

For model (2.1)-(2.3), we can classify the type of the bifurcation at $R_0 = 1$ according to the theorem from [12].

Theorem 2.2. *For model (2.1)-(2.3), if $R_1 = \frac{\beta}{\frac{\Lambda}{\mu}\alpha_1\gamma} < 1$, the bifurcation at $R_0 = 1$ is backward, while $R_1 > 1$ the bifurcation at $R_0 = 1$ is forward.*

3 Endemic equilibria

The endemic equilibria of model (2.1)-(2.3) are solutions to

$$\Lambda - \beta S\frac{I}{S+I} - \mu S + (\alpha_2 + \alpha_1 e^{-\gamma I})I = 0, \tag{3.1}$$

$$\beta\frac{S}{S+I} - (\mu + d) - (\alpha_2 + \alpha_1 e^{-\gamma I}) = 0. \tag{3.2}$$

In order to obtain positive solution of (3.1)-(3.2), we eliminate S using the sum of equation (3.1) and (3.2) $\Lambda - \mu(S+I) - dI = 0$, and then substitute it into (3.2). The (S, I) coordinates of an equilibrium must satisfy

$$S = \frac{\Lambda - (\mu + d)I}{\mu}, \quad h(I) \triangleq a_0 + a_1 I + a_2 e^{-\gamma I} + a_3 I e^{-\gamma I} = 0, \tag{3.3}$$

where

$$\begin{aligned}
a_0 =& \Lambda(\beta - \mu - d - \alpha_2) = \Lambda(\mu + d + \alpha_2)(\frac{\beta}{\mu + d + \alpha_2} - 1) \\
=& \Lambda(\mu + d + \alpha_1 + \alpha_2)(R_0 - 1) + \Lambda\alpha_1, \\
a_1 =& d(\mu + d + \alpha_2) - \beta(\mu + d) = -d(\mu + d + \alpha_2)(\frac{\beta}{\mu + d + \alpha_2}\frac{\mu + d}{d} - 1) \\
=& -d(\mu + d + \alpha_2)(R_0\frac{\mu + d + \alpha_1 + \alpha_2}{\mu + d + \alpha_2}\frac{\mu + d}{d} - 1), \\
a_2 =& -\Lambda\alpha_1 < 0, \\
a_3 =& d\alpha_1 > 0.
\end{aligned}$$

We introduce functions $f(I)$ and $g(I)$, and $h(I)$ can be rewritten as $h(I) = g(I) -$

$f(I)$:

$$f(I) \triangleq -(a_2 + a_3 I)e^{-\gamma I}, \quad g(I) \triangleq a_0 + a_1 I. \tag{3.4}$$

The endemic equilibrium $E^* = (S^*, I^*)$ can be found by solving S and I from the following equations

$$S = \frac{\Lambda - (\mu + d)I}{\mu}, \quad f(I) = g(I). \tag{3.5}$$

In order to have $E^* > 0$, we need $0 < I^* \leq \frac{\Lambda}{\mu+d}$.

Denote $I_m = \frac{\Lambda}{\mu+d}$. The first and second order derivations of $f(I)$ are,

$$f'(I) = (\gamma a_3 I + \gamma a_2 - a_3)e^{-\gamma I} = \alpha_1(\gamma d I - \gamma\Lambda - d)e^{-\gamma I},$$

$$f''(I) = -\gamma(\gamma a_3 I + \gamma a_2 - 2a_3)e^{-\gamma I} = -\gamma\alpha_1(\gamma d I - \gamma\Lambda - 2d)e^{-\gamma I},$$

we obtain the following properties of $f(I)$: (i) $f(I)$ decreases in interval $(0, I_c)$, and increases in interval $(I_c, +\infty)$, where $I_c = \frac{a_3 - \gamma a_2}{\gamma a_3} = \frac{d+\gamma\Lambda}{\gamma d} > 0$; (ii) $f(I)$ is concave down in interval $(0, I_h)$, and is concave upward in interval $(I_h, +\infty)$, where $I_h = \frac{2a_3 - \gamma a_2}{\gamma a_3} = \frac{2d+\gamma\Lambda}{\gamma d} > I_c$; (iii) Since $\lim_{I\to+\infty} f(I) = 0$, so the maximum of $f(I)$ is $f(0) = -a_2 = \Lambda\alpha_1 > 0$, and the minimum is $f(I_c) = -\frac{a_3}{\gamma}e^{-\gamma I_c} = -\frac{d\alpha_1}{\gamma}e^{-1-\frac{\gamma\Lambda}{d}} < 0$. A graph of $f(I)$ is sketched in Fig. 1.

To compute the biologically feasible equilibrium, we need to consider the intersections between $f(I)$ and $g(I)$ on the interval $(0, I_m]$. Because $0 < I_m < I_c < I_h$, $f'(I) < 0$ and $f''(I) > 0$ hold when $I \in (0, I_m]$. It is easy to conclude that $f(I)$ is monotone decreasing and concave down in interval $(0, I_m]$ (see Fig.1). And $f(I_m) \geq f(I) < f(0)$, where $f(0) = \Lambda\alpha_1$, $f(I_m) = f(\frac{\Lambda}{\mu+d}) = \frac{\mu\Lambda\alpha_1}{\mu+d}e^{-\frac{\gamma\Lambda}{\mu+d}}$.

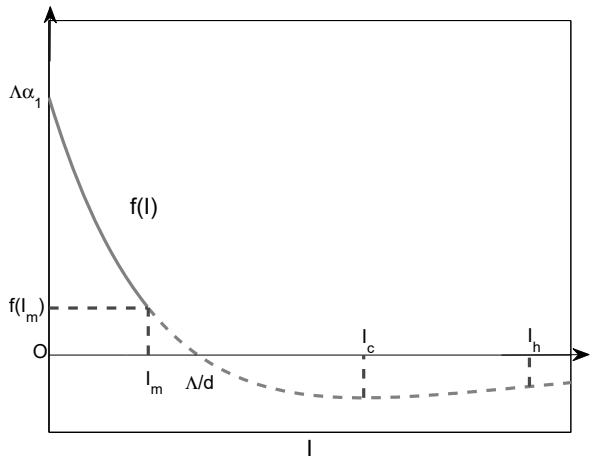

Figure 1: Plot of function $f(I)$. On interval $[0, I_m]$, $f(I)$ is a monotone decreasing function. It reaches its maximum and minimum values at point $I = 0$ and $I = I_m$, respectively, and $f(0) > f(I_m) > 0$.

Let

$$R^* = \frac{\beta}{\mu + d + \alpha_2}, \quad R^{**} = \Big(\frac{\beta}{\mu + d + \alpha_2}\Big)\Big(\frac{\mu + d}{d}\Big),$$

which corresponds to $a_0 = 0$ and $a_1 = 0$, respectively. Now we discuss the number of intersections between $f(I)$ and $g(I)$, the straight line $g(I)$ is determined by the signs of a_0 and a_1, so we should study the intersections between $f(I)$ and $g(I)$ for following distinct cases:

Case 1: $1 < R_0 < R^* < R^{**}$;

In this case $a_0 > \Lambda\alpha_1 = f(0)$ and $a_1 < 0$ always hold, hence $g(I)$ is decreasing on interval $(0, I_m]$, and we have $g(0) = a_0 > f(0)$, $g(I_m) = -\frac{\mu\Lambda}{\mu+d}(\mu+d+\alpha_2) < 0 < f(I_m)$. There is one and only one intersection between $f(I)$ and $g(I)$, as is shown in Fig.2. That is, when $R_0 > 1$, the system (2.1)-(2.3) has an unique endemic equilibrium.

Furthermore due to $p''(I) = \gamma^2\alpha_1 e^{-\gamma I} > 0$, and using Theorem 4.2 in [23], we can

conclude the following theorem:

Theorem 3.1. *For model (2.1)-(2.3), when $R_0 > 1$, there is an unique equilibrium and which is locally asymptotically stable.*

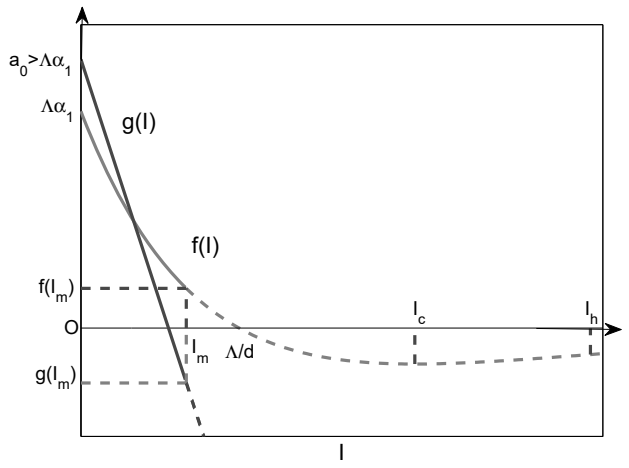

Figure 2: Case 1, when $R_0 > 1$, plot of the intersection between $f(I)$ and $g(I)$. On interval $[0, I_m]$, both $f(I)$ and $g(I)$ are monotone decreasing, and $g(0) > f(0) > 0$, $f(I_m) > 0 > g(I_m)$ hold. The intersection between $f(I)$ and $g(I)$ on interval $[0, I_m]$ is unique.

Case 2: $R_0 < 1 < R^* < R^{**}$;

In this case $0 < a_0 < \Lambda\alpha_1 = f(0)$, $a_1 < 0$. There may be zero, one or two intersections between $f(I)$ and $g(I)$, as is shown in Fig.3.

Case 3: $R_0 < R^* < 1 < R^{**}$;

In this case $a_0 < 0, a_1 < 0$. We have $\max\limits_{I\in[0,I_m]} g(I) = g(0) = a_0 < 0$ and $\min\limits_{I\in[0,I_m]} f(I) = f(I_m) = \frac{\mu\Lambda\alpha_1}{\mu+d} e^{-\frac{\gamma\Lambda}{\mu+d}} > 0$, then $\max\limits_{I\in[0,I_m]} g(I) < \min\limits_{I\in[0,I_m]} f(I)$, thereforce it is clearly that there is no intersection between $f(I)$ and $g(I)$ on interval $[0, I_m]$, as is shown in Fig.4.

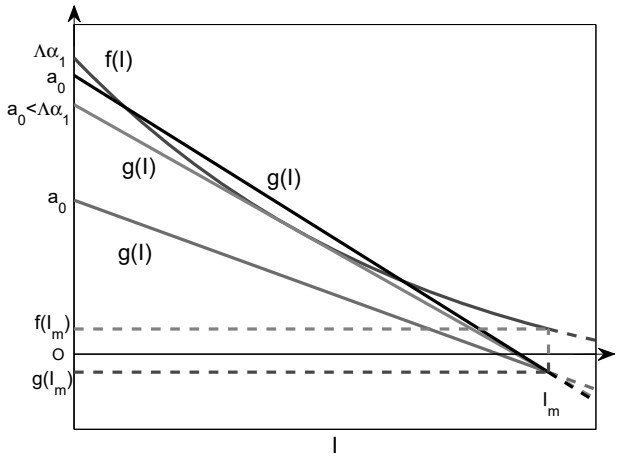

Figure 3: Case 2, when $R_0 < 1 < R^* < R^{**}$, plot of the intersections between $f(I)$ and $g(I)$. On interval $[0, I_m]$, the black line of $g(I)$ has two intersections with the function $f(I)$. The red line of $g(I)$ is tangent with function $f(I)$, so there is only one intersection. And the green line of $g(I)$ has no intersection with the function $f(I)$.

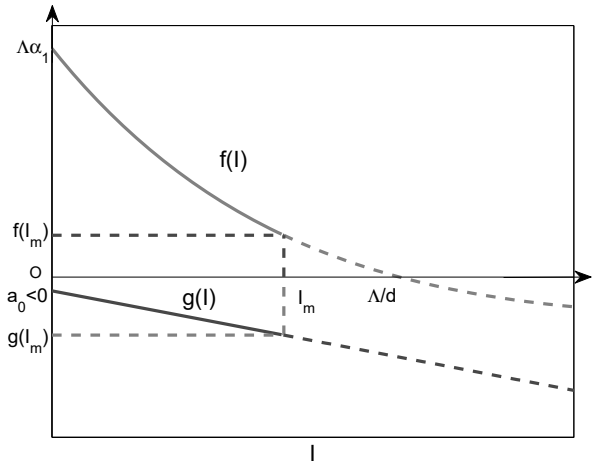

Figure 4: Case 3, when $R_0 < R^* < 1 < R^{**}$, plot of the intersection of the function $f(I)$ the $g(I)$. Since $g(I) < f(I)$ always holds in interval $[0, I_m]$, so there is no intersection for the function $f(I)$ and $g(I)$ in interval $[0, I_m]$.

Case 4: $R_0 < R^* < R^{**} < 1$;

In this case $a_0 < 0, a_1 > 0$. We have $\max\limits_{I\in[0,I_m]} g(I) = g(I_m) = -\frac{\mu\Lambda}{\mu+d}(\mu + d + \alpha_2) < 0$ and $\min\limits_{I\in[0,I_m]} f(I) = f(I_m) = \frac{\mu\Lambda\alpha_1}{\mu+d}e^{-\frac{\gamma\Lambda}{\mu+d}} > 0$, then $\max\limits_{I\in[0,I_m]} g(I) < \min\limits_{I\in[0,I_m]} f(I)$, hence it is clearly that there is no intersection between $f(I)$ and $g(I)$ on interval $[0, I_m]$, as is shown in Fig.5.

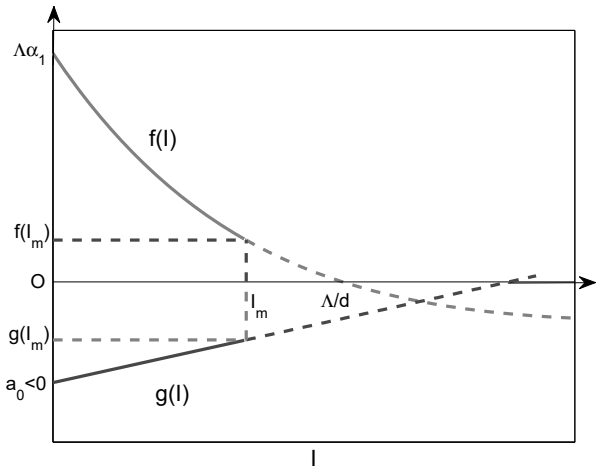

Figure 5: Case 4, when $R_0 < R^* < R^{**} < 1$, plot of the intersection between $f(I)$ and $g(I)$. Since $g(I) < f(I)$ always holds on interval $[0, I_m]$, so there is no intersection between $f(I)$ and $g(I)$ on interval $[0, I_m]$.

3.1 Saddle-node bifurcation

The existence of bifurcation depends on several parameters. To investigate the impact of transmission rate and treatment, we choose β and α_2 as our bifurcation parameters and consider bifurcations in the (β, α_2) plane.

In this case, the Implicit Function Theorem provides the (local) existence of two

smooth functions:

$$f(I) = g(I),$$

$$f'(I) = g'(I),$$

i.e.

$$a_0 + a_1 I = -a_2 e^{-\gamma I} - a_3 I e^{-\gamma I}, \tag{3.6}$$

$$a_1 = (\gamma a_2 + \gamma a_3 I - a_3) e^{-\gamma I}, \tag{3.7}$$

solving a_0 and a_1 from (3.6)-(3.7):

$$a_0 = -(a_2 + \gamma a_2 I + \gamma a_3 I^2) e^{-\gamma I},$$

$$a_1 = (\gamma a_2 + \gamma a_3 I - a_3) e^{-\gamma I},$$

so the parameter function of β and α_2 is obtained:

$$(\mu + d)\beta - d\alpha_2 = d(\mu + d) + \alpha_1(\gamma\Lambda + d - \gamma d I) e^{-\gamma I},$$

$$\beta - \alpha_2 = \alpha_1(1 + \gamma I + \frac{\gamma d I^2}{\Lambda}) e^{-\gamma I},$$

then if $f(I)$ is tangent to $g(I)$ at point I, the parameter β and α_2 satisfies

$$\beta(I) = \gamma(\Lambda - 2dI + \frac{d^2}{\Lambda} I^2) \frac{\alpha_1}{\mu} e^{-\gamma I} = \frac{\gamma \alpha_1}{\Lambda \mu} e^{-\gamma I} (\Lambda - dI)^2, \tag{3.8}$$

$$\alpha_2(I) = -(\mu + d) + (\gamma\Lambda - \mu - \gamma(\mu + 2d)I + \frac{\gamma d(\mu + d)}{\Lambda} I^2)\frac{\alpha_1}{\mu} e^{-\gamma I}, \tag{3.9}$$

following we need to $I \in [0, I_m]$. Since

$$\frac{\partial \beta}{\partial I} = -\frac{\gamma\alpha_1}{\Lambda\mu} e^{-\gamma I}(\Lambda - dI)(\gamma(\Lambda - dI) + 2d) < 0, \tag{3.10}$$

$$\frac{\partial \alpha_2}{\partial I} = -\frac{\gamma\alpha_1}{\Lambda\mu} e^{-\gamma I}(\Lambda - (\mu + d)I)(\gamma(\Lambda - dI) + 2d) < 0, \tag{3.11}$$

when $I \in [0, I_m]$. So both $\beta(I)$ and $\alpha_2(I)$ are decreasing on interval $[0, I_m]$. $\beta(I) > 0$ is always holds on interval $[0, I_m]$. $\alpha_2(I) \in [\alpha_2(I_m), \alpha_2(0)]$, to make $\alpha_2(I) > 0$ holds for some $I \in [0, I_m]$, $\alpha_2(0) > 0$ is sufficient and necessary. Hence for model (2.1)-(2.3), we obtain the necessary and sufficient condition for the saddle-node bifurcation occurs

$$\gamma\Lambda\alpha_1 - \mu(\mu + d + \alpha_1) > 0. \tag{3.12}$$

Denote $p(I) \triangleq (1 + \gamma I(1 - \frac{d}{\Lambda} I))e^{-\gamma I}$. From (3.8) and (3.9) we can obtain that when a saddle-node bifurcation occurs

$$\alpha_2(I) - \beta(I) = -(\mu + d) - p(I)\alpha_1. \tag{3.13}$$

The derivation of $p(I)$ is

$$p\prime(I) = \gamma e^{-\gamma I} I(\frac{\gamma d}{\Lambda} I - (\gamma + \frac{2d}{\Lambda})). \tag{3.14}$$

It is easily to verify that $p\prime(I) < 0$ on interval $[0, I_m]$, so $p(I_m) \leq p(I) < p(0)$ holds

when $I \in (0, I_m]$, then we can derive to that $0 < p(I) < 1$ on interval $(0, I_m]$. From (3.13), we obtain the threshold condition of saddle-node bifurcation occurs satisfies that

$$-(\mu + d + \alpha_1) < \alpha_2 - \beta < -(\mu + d) \tag{3.15}$$

when $I \in (0, I_m]$.

Furthermore, from (3.8) and (3.9) we can calculate the first and second order derivations of α_2 with respect to β

$$\frac{\partial \alpha_2}{\partial \beta} = \frac{\Lambda - (\mu + d)I}{\Lambda - dI}, \tag{3.16}$$

$$\frac{\partial^2 \alpha_2}{\partial \beta^2} = \frac{\mu^2 \Lambda^2 e^{\gamma I}}{\gamma \alpha_1 (\Lambda - dI)^3 (\gamma(\Lambda - dI) + 2d)}. \tag{3.17}$$

Then we can also obtain that $0 \leq \frac{\partial \alpha_2}{\partial \beta} < 1$ and $\frac{\partial^2 \alpha_2}{\partial \beta^2} > 0$ hold when $I \in (0, I_m]$, hence the function α_2 is monotonous increasing with slope is less than 1, and is concave down with respect to β when $I \in (0, I_m]$.

Denote

$$\hat{R}_0 = \frac{\beta(I)}{\mu + d + \alpha_1 + \alpha_2(I)} = \frac{\beta(I)}{\beta(I) + (1 - p(I))\alpha_1}, \tag{3.18}$$

$0 < \hat{R}_0 < 1$ holds since $0 < p(I) < 1$. From the above analysis we get the result regarding the number of endemic equilibrium.

Theorem 3.2. *For model (2.1)-(2.3), we have*

(1) When $R_0 \geq 1$, there is a unique endemic equilibrium.

(2) when $\hat{R}_0 < R_0 < 1$, there are two endemic equilibria.

(3) when $R_0 = \hat{R}_0$, the two endemic equilibria coalesce at a unique endemic equilibrium of multiplicity 2.

(4) when $R_0 < \hat{R}_0$, there is no endemic equilibrium.

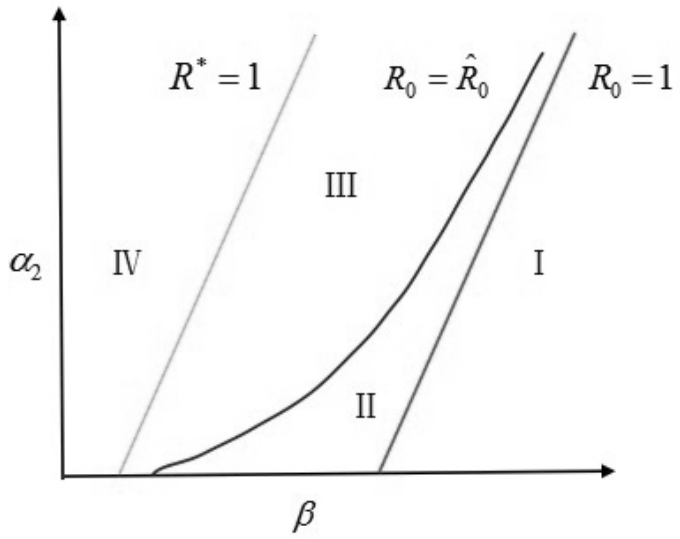

Figure 6: The distribution of endemic equilibria on the plane of (β, α_2).

3.2 Hopf bifurcation

We investigate the Hopf bifurcation in region $\hat{R}_0 < R_0 < 1$, in which there are two endemic equilibria. In this region, (3.12) and $p(I)\alpha_1 < \beta - (\mu + d + \alpha_2) < \alpha_1$ hold. The variational matrix about any equilibrium $E^* = (S^*, I^*)$ of system (2.1)-(2.3) is

$$J(E^*) = \begin{pmatrix} -\beta\frac{I^{*2}}{(S^*+I^*)^2} - \mu & -\beta\frac{S^{*2}}{(S^*+I^*)^2} + \alpha_2 + \alpha_1 e^{-\gamma I^*} - \gamma\alpha_1 I^* e^{-\gamma I^*} \\ \beta\frac{I^{*2}}{(S^*+I^*)^2} & \beta\frac{S^{*2}}{(S^*+I^*)^2} - \mu - d - \alpha_2 - \alpha_1 e^{-\gamma I^*} + \gamma\alpha_1 I^* e^{-\gamma I^*} \end{pmatrix},$$

so the trace of $J(E^*)$ is

$$tr(J(E^*)) = \beta\frac{S^* - I^*}{S^* + I^*} - \alpha_1 e^{-\gamma I^*} + \gamma\alpha_1 I^* e^{-\gamma I^*} - 2\mu - d - \alpha_2, \tag{3.19}$$

from (3.3) we also have

$$S^* = \frac{\Lambda - (\mu + d)I^*}{\mu}, \quad e^{-\gamma I^*} = -\frac{a_0 + a_1 I^*}{a_2 + a_3 I^*}, \tag{3.20}$$

substitute the (3.20) to (3.19), we can determine a threshold condition for Hopf bifurcation:

$$tr(J(E^*)) = \frac{1}{\Lambda - dI^*}(b_0 I^{*2} + b_1 I^* + b_2), \tag{3.21}$$

where

$$\begin{aligned} b_0 =& \gamma a_1 = \gamma d(\mu + d + \alpha_2) - \gamma\beta(\mu + d), \\ b_1 =& \gamma a_0 - \mu(\beta - d) = \gamma\Lambda(\beta - \mu - d - \alpha_2) - \mu(\beta - d), \\ b_2 =& -\mu\Lambda < 0. \end{aligned}$$

From the analysis before we know that $I^* \in [0, I_m]$ implies that $a_0 > 0$, i.e $\beta > \mu + d + \alpha_2$ holds, which can also derive to that $a_1 < 0$, so we can obtain that for equation (3.21), $b_0 < 0$, since $b_2 < 0$, in order to let the roots of (3.21) are positive, it is clearly that

the conditions $\Delta = {b_1}^2 - 4b_0b_2 \geq 0$ and $b_1 > 0$ must hold:

$$\begin{aligned}\Delta &= {b_1}^2 - 4b_0b_2 \\ &= ((\gamma\Lambda - \mu)\beta - \gamma\Lambda(\mu + d + \alpha_2) - \mu d)^2 - 4\mu^2\beta(\gamma\Lambda + d) \\ &= (b_1 - 2\mu d)^2 - 4\mu^2\beta(\gamma\Lambda + d) \\ &\geq 0,\end{aligned} \tag{3.22}$$

from (3.22), we can derive to

$$b_1 \geq 2\mu d + 2\mu\sqrt{\gamma\Lambda + d}\sqrt{\beta}, \tag{3.23}$$

or

$$b_1 \leq 2\mu d - 2\mu\sqrt{\gamma\Lambda + d}\sqrt{\beta} < 0, \tag{3.24}$$

(3.24) is a contradiction with $b_1 > 0$, hence we just consider the condition (3.23). so when (3.23) holds, the (3.21) will have two positive roots:

$$I_1 = \frac{-b_1 + \sqrt{\Delta}}{2b_0}, \qquad I_2 = \frac{-b_1 - \sqrt{\Delta}}{2b_0}. \tag{3.25}$$

In the same way, we can get the determinant of $J(E^*)$ is

$$det(J(E^*)) = \mu(\mu + d + \alpha_2) - \beta\mu\frac{S^* - I^*}{S^* + I^*} + d\beta\frac{{I^*}^2}{(S^* + I^*)^2} + \mu\alpha_1 e^{-\gamma I^*}(1 - \gamma I^*), \tag{3.26}$$

furthermore

$$det(J(E^*)) = \frac{1}{(\Lambda - dI^*)^2}\mu I^*(c_0 I^{*2} + c_1 I^* + c_2), \tag{3.27}$$

where

$$\begin{aligned} c_0 =& \gamma d a_1 = \gamma d(d(\mu + d + \alpha_2) - \beta(\mu + d)), \\ c_1 =& \gamma(2d a_0 + \beta\Lambda\mu) = \gamma\Lambda(2d(\beta - \mu - d - \alpha_2) + \beta\mu), \\ c_2 =& -\Lambda(\gamma a_0 - \beta\mu) = -\Lambda(\gamma\Lambda(\beta - \mu - d - \alpha_2) - \beta\mu). \end{aligned}$$

(3.26) can been rewritten as

$$det(J(E^*)) = d(b_0 I^{*2} + b_1 I^* + b_2) + (d\gamma a_0 + d\mu(\beta - d) + d\gamma\beta\Lambda)I^* - \Lambda(b_1 - 2d\mu), \tag{3.28}$$

we can verify that

$$det(J(E(S_1, I_1))) < 0, \qquad det(J(E(S_2, I_2))) > 0, \tag{3.29}$$

so endemic equilibrium $E(S_1, I_1)$ is an saddle, we continue to consider endemic equilibrium $E(S_2, I_2)$. Since

$$\frac{\partial}{\partial\beta} tr(J(E_2)) \neq 0, \quad \frac{\partial}{\partial\alpha_2} tr(J(E_2)) \neq 0,$$

we need

$$h(I_2) = 0,$$

which conclude the critical condition of hopf bifurcation occurs

$$a_0 + a_1 I_2 + a_2 e^{-\gamma I_2} + a_3 I_2 e^{-\gamma I_2} = 0. \tag{3.30}$$

4 Discrete-time model with density-dependent treatments

Continuous epidemic models have played a important role on investigating the transmitting law of epidemic diseases and predicting the development trend of their spread. Meanwhile, discrete epidemic models have gained more popularity[6, 7, 8, 4, 5, 9, 10, 11], since epidemiological data are usually collected at discrete times and it becomes easier to compare data with models. The backward bifurcations are also found in discrete epidemic models[6, 7, 4, 5, 9, 10, 11]. [6, 7] developed models for the study of disease dynamics in populations with discrete generations and potentially complex (chaotic) disease-free dynamics. The work in [8] introduced a discrete-epidemic framework, and the similarities between single-outbreak comparable classical continuous-time epidemic models and the discrete-time models are introduced through analysis. [10] formulated an extended Ricker model by incorporating Allee effects based on the classical discrete Ricker population model, and showed that the model exhibits period-doubling bifurcations and stability cycles. [11] proposed a set of discrete SEIS models with exogenous reinfections and a variety of treatment strategies, and period doubling, backward, forward-backward, and multiple backward bifurcations are identified from the models.

In this section, we divide the population into two subpopulations: susceptible sub-

population (S) and infectious subpopulation (I), and construct a discrete SIS epidemic models with density-dependent treatments, and the parameters in the model are introduced by corresponding probability. We let he time step be 1, which is also the unit time step. S_n represents the number of the susceptible individuals at time n, and I_n is the number of infected individuals at time n. To formulate the model, we give some assumptions in the following:

(i) Let Λ be the constant recruitment rate and all the which are susceptible against the infection.

(ii) The probability of an susceptible still alive via a time step is a constant, denoted by π; and the probability of an infected individual still alive is π_1, it is rational to let $0 < \pi_1 \leq \pi < 1$. Then the number of susceptible and infected individuals still alive in the period $[n, n+1)$ are πS_n and $\pi_1 I_n$, respectively.

(iii) In period $[n, n+1)$, the probability of a susceptible individual not being infected is $e^{-\beta I_n}$. Hence, in this period, the number of susceptible individuals alive and remaining in S is $\pi S_n e^{-\beta I_n}$, and the number of new infected individuals is $\pi S_n(1 - e^{-\beta I_n})$.

(iv) In period $[n, n+1)$, the probability of an infectious individual removing from I is a density-dependent treatments function: $\alpha_2 + \alpha_1 e^{-\gamma I_n}$, then the probability that an infectious individual survived and infectious in period $[n, n+1)$ is $\pi_1(1-(\alpha_2+\alpha_1 e^{-\gamma I_n}))$. Therefore, via the period $[n, n+1)$, the number of individuals being alive and infectious is $\pi_1(1 - (\alpha_2 + \alpha_1 e^{-\gamma I_n}))I_n$, and the number of individuals being alive and removed from I is $\pi_1 I_n(\alpha_2 + \alpha_1 e^{-\gamma I_n})$. Then the discrete SIS model is as follows:

$$\begin{cases} S_{n+1} = \Lambda + \pi S_n e^{-\beta I_n} + \pi_1 I_n(\alpha_2 + \alpha_1 e^{-\gamma I_n}), \\ \\ I_{n+1} = \pi S_n(1 - e^{-\beta I_n}) + \pi_1 I_n(1 - (\alpha_2 + \alpha_1 e^{-\gamma I_n})), \end{cases} \tag{4.1}$$

We impose the following conditions on the parameters so that solutions of (4.1) remain nonnegative: $0 < \alpha_1 + \alpha_2 < 1$.

4.1 Analysis

Assume $\pi \neq \pi_1$, for the model (4.1), the disease-free equilibrium $q_0 = (\frac{\Lambda}{1-\pi}, 0)$ is always existent.

The Jacobian matrix of the system (4.1) is

$$J = \begin{pmatrix} \pi e^{-\beta I_n} & -\beta\pi S_n e^{-\beta I_n} + \pi_1(\alpha_2 + \alpha_1 e^{-\gamma I_n}) - \gamma\pi_1\alpha_1 I_n e^{-\gamma I_n} \\ \\ \pi(1 - e^{-\beta I_n}) & \beta\pi S_n e^{-\beta I_n} + \pi_1(1 - (\alpha_2 + \alpha_1 e^{-\gamma I_n})) + \gamma\pi_1\alpha_1 I_n e^{-\gamma I_n} \end{pmatrix},$$

The local stability of q_0 can be determined by the Jacobian matrix of system (4.1) evaluated at q_0 which has the following form:

$$J(q_0) = \begin{pmatrix} \pi & -\beta\pi\frac{\Lambda}{1-\pi} + \pi_1(\alpha_2 + \alpha_1) \\ \\ 0 & \beta\pi\frac{\Lambda}{1-\pi} + \pi_1(1 - (\alpha_2 + \alpha_1)) \end{pmatrix},$$

so the eigenvalues $0 < \lambda_1 = \pi < 1$, and $0 < \lambda_2 = \beta\pi\frac{\Lambda}{1-\pi} + \pi_1(1 - (\alpha_2 + \alpha_1) < 1$ if and only if

$$\frac{\beta\pi\frac{\Lambda}{1-\pi}}{1 - \pi_1(1 - (\alpha_2 + \alpha_1)} < 1.$$

We now can obtain the basic reproduction number is

$$R_d = \frac{\beta\pi\Lambda}{(1-\pi)(1-\pi_1+\pi_1(\alpha_1+\alpha_2))}, \tag{4.2}$$

and we have the following theorem:

Theorem 4.1. *For model (4.1), the disease-free equilibrium q_0 is locally asymptotically stable if $R_d^2 < 1$, and is unstable when $R_d^2 > 1$.*

Furthermore, the global stability of e_0 can obtain:

Theorem 4.2. *For model (4.1), if $R_d < 1$, the disease-free equilibrium q_0 is globally stable when $\alpha_1\gamma - \beta \leq 0$.*

Proof. Since $0 < \pi_1 \leq \pi < 1$, so take the sum of the two equations of (4.1), we obtain:

$$\begin{aligned} S_{n+1} + I_{n+1} &= \Lambda + \pi S_n + \pi_1 I_n \\ &= \Lambda + \pi(S_n + I_n) - (\pi - \pi_1)I_n \end{aligned} \tag{4.3}$$

So $N = \frac{\Lambda-(\pi-\pi_1)I}{1-\pi} \leq \frac{\Lambda}{1-\pi}$, so $S_n \leq \frac{\Lambda}{1-\pi} - I_n$, substituting into the last equations of models (4.1). And according to that $\alpha_1\gamma - \beta \leq 0$ and $1 - e^{-\beta I_n} \leq \beta I_n$, we can obtain

that:

$$\begin{aligned} I_{n+1} &\leq \pi(\frac{\Lambda}{1-\pi} - I_n)(1 - e^{-\beta I_n}) + \pi_1 I_n(1 - (\alpha_2 + \alpha_1 e^{-\gamma I_n})) \\ &\leq \pi(\frac{\Lambda}{1-\pi} - I_n)\beta I_n + \pi_1 I_n[1 - (\alpha_1 + \alpha_2) + \alpha_1(1 - e^{-\gamma I_n})] \\ &\leq \frac{\beta\pi\Lambda}{1-\pi} I_n - \pi\beta {I_n}^2 + \pi_1 I_n[1 - (\alpha_1 + \alpha_2)] + \pi_1\alpha_1\gamma {I_n}^2 \\ &= \frac{\beta\pi\Lambda}{1-\pi} I_n + \pi I_n[1 - (\alpha_1 + \alpha_2)] + \pi(\alpha_1\gamma - \beta){I_n}^2 \\ &= \frac{\beta\pi\Lambda}{1-\pi} I_n + \pi_1 I_n[1 - (\alpha_1 + \alpha_2)] \\ &= [R_d + \pi_1(1 - R_d)(1 - (\alpha_1 + \alpha_2))] I_n. \end{aligned} \tag{4.4}$$

Since $R_d < 1$, $0 < \pi_1, \alpha_1 + \alpha_2 < 1$, so

$$0 < \pi_1(1 - R_d)(1 - (\alpha_1 + \alpha_2)) < 1 - R_d,$$

then

$$R_d + \pi(1 - R_d)(1 - (\alpha_1 + \alpha_2)) < R_d + (1 - R_d) = 1, \tag{4.5}$$

which can clearly conclude that $\lim_{n\to\infty} I_n = q_0$, i.e the disease-free equilibrium e_0 is globally stable. □

4.2 Analysis: $\pi = \pi_1$

Assume $\pi = \pi_1$, the probability of an susceptible and infected individual alive via a time step is the same. Then we have $N_{n+1} = \Lambda + \pi N_n$, using limiting equations $\lim_{t\to+\infty} N = \frac{\Lambda}{1-\pi}$. Substituting $S_n = N - I_n$ into the last equations of models (4.1),

gives the following equations:

$$I_{n+1} = \pi(\frac{\Lambda}{1-\pi} - I_n)(1 - e^{-\beta I_n}) + \pi I_n(1 - (\alpha_2 + \alpha_1 e^{-\gamma I_n})), \tag{4.6}$$

Let $x_n = \frac{I_n}{N}$, then (4.6) becomes to:

$$x_{n+1} = \pi(1 - e^{-bx_n} + x_n e^{-bx_n} - x_n(\alpha_2 + \alpha_1 e^{-rx_n})) \stackrel{\triangle}{=} d(x), \tag{4.7}$$

where $b = \beta N$, $r = \gamma N$ and $x_n \in [0, 1]$.

Firstly, we consider the stability of the equilibrium $e_0 = 0$, which is always existent. We have

$$d'(x) = \pi(be^{-bx} - e^{-bx} - bxe^{-bx} - \alpha_2 - \alpha_1 e^{-rx} - r\alpha_1 xe^{-rx}), \tag{4.8}$$

since $d'(0) = \pi(b + 1 - \alpha_2 - \alpha_1)$, the equilibrium $e_0 = 0$ is asymptotically stable if $|d'(0)| < 1$, we can obtain the basic reproduction number is

$$R_d^1 = \frac{\beta\pi\Lambda}{(1-\pi)^2 + \pi(1-\pi)(\alpha_1 + \alpha_2)} = \frac{\beta\pi\Lambda}{(1-\pi)(1-\pi+\pi(\alpha_1+\alpha_2))}, \tag{4.9}$$

and we have the following theorem:

Theorem 4.3. *For model (4.7), the disease-free equilibrium e_0 is locally asymptotically stable if $R_d^1 < 1$, and is unstable when $R_d^1 > 1$.*

Furthermore, the global stability of e_0 can obtain:

Theorem 4.4. *For model (4.7), if $R_d^1 < 1$, the disease-free equilibrium e_0 is globally stable when $\alpha_1\gamma - \beta \leq 0$.*

Proof. According to that $\alpha_1\gamma - \beta \leq 0$ and $1 - e^{-\beta I_n} \leq \beta I_n$, we can obtain that:

$$\begin{aligned} I_{n+1} &= \pi(\frac{\Lambda}{1-\pi} - I_n)(1 - e^{-\beta I_n}) + \pi I_n(1 - (\alpha_2 + \alpha_1 e^{-\gamma I_n})) \\ &\leq \pi(\frac{\Lambda}{1-\pi} - I_n)\beta I_n + \pi I_n[1 - (\alpha_1 + \alpha_2) + \alpha_1(1 - e^{-\gamma I_n})] \\ &\leq \frac{\beta\pi\Lambda}{1-\pi} I_n - \pi\beta {I_n}^2 + \pi I_n[1 - (\alpha_1 + \alpha_2)] + \pi\alpha_1\gamma {I_n}^2 \\ &= \frac{\beta\pi\Lambda}{1-\pi} I_n + \pi I_n[1 - (\alpha_1 + \alpha_2)] + \pi(\alpha_1\gamma - \beta){I_n}^2 \\ &\leq \frac{\beta\pi\Lambda}{1-\pi} I_n + \pi I_n[1 - (\alpha_1 + \alpha_2)] \\ &= [R_d^1 + \pi(1 - R_d^1)(1 - (\alpha_1 + \alpha_2))] I_n. \end{aligned} \tag{4.10}$$

Since $R_d^1 < 1$, $0 < \pi, \alpha_1 + \alpha_2 < 1$, so

$$0 < \pi(1 - R_d^1)(1 - (\alpha_1 + \alpha_2)) < 1 - R_d^1,$$

then

$$R_d^1 + \pi(1 - R_d^1)(1 - (\alpha_1 + \alpha_2)) < R_d^1 + (1 - R_d^1) = 1, \tag{4.11}$$

which can clearly conclude that $\lim_{n\to\infty} I_n = e_0$, i.e the disease-free equilibrium e_0 is globally stable. □

5 Conclusion and discussion

Using density-dependent treatment function $p(I) = \alpha_2 + \alpha_1 e^{-\gamma I}$, this paper show that that the bi-stability and backward bifurcation are not automatically connected theoretically. Firstly, a continuous model with density-dependent treatments are studied to show that when a backward bifurcation occurs, the disease-free equilibrium can be globally stable, and a stable limit cycle exist. We find bifurcation curves in a subset of entire parameter space. When $\hat{R}_0 < R_0 < 1$, there are two endemic equilibria for the system, one of which is an saddle, and the other can have rich dynamics. The endemic equilibria is an stable focus when parameter β is large enough, but as parameter β decreases, it lose stability and becomes an center. The limit cycle can become bigger and bigger as parameter β decreases, when the limit cycle intersect with the orbit of saddle, a homoclinic orbit occur. If β continue to decrease, the limit cycle will break, and the disease-free equilibrium is globally stable. Finally, a corresponding discrete-time model with density-dependent treatment are used to find these type of bifurcations.

Acknowledgments

We would like to thank Dr. Carlos Castillo-Chavez, Executive Director of the Mathematical and Theoretical Biology Institute (MTBI), for giving us the opportunity to participate in this research program. We would also like to thank Co-Executive Summer Directors Dr. Erika T. Camacho and Dr. Stephen Wirkus for their efforts in planning and executing the day to day activities of MTBI. We also want to give special thanks to Yiqiang Zheng, for his kindness help during this project. This research was conducted in MTBI at the Mathematical, Computational and Modeling Sciences Center (MCMSC) at Arizona State University (ASU). This project has been partially supported by grants from the National Science Foundation (NSF - Grant DMPS-1263374), the National Security Agency (NSA - Grant H98230-13-1-0261), the Office of the President of ASU, and the Office of the Provost of ASU. Finally, we would like to thank Professor Zhen Jin for supporting Xiaoguang Zhang's international travelling.

References

[1] J. Zhang, Z. Ma, Global analysis of the SEI epidemic model with constant inflows of different compartments, Journal of Xian Jiaotong University, 6(37): 653-656, 2003.

[2] S. Yuan, L. Han, Z. Ma, A kind of epidemic model h aving infectious f orce in both latent period and infected period, Journal of B iomathematics, 4(16): 392-398, 2001.

[3] G. Li and Z. Jin, Global stability of an SEI epidemic model, Chaos, Solitons and Fractals, 4(21): 925-931, 2004.

[4] C. Castillo-Chavez, A. Yakubu. Intraspecific competition, dispersal and disease dynamic in discrete-time patchy environments.

[5] C. Castillo-Chavez, A. Yakubu. Discrete-time SIS models with simple and complex population dynamics.

[6] C. Castillo-Chavez, A. Yakubu. Dispersal, disease and life-history evolution. Mathematical Biosciences 173(2001),35-53.

[7] C. Castillo-Chavez, A. Yakubu. Discrete-time SIS models with complex dynamics. Nonlinear analysis 47(2001), 4753-4762.

[8] F. Brauer, Z. Feng, C. Castillo-Chavez. Discrete epidemic models. Mathematical Biosciences and Engineering: 1(7),2010.

[9] Abdul-Aziz Yakubu, Roberto Sáenz, Julie Stein, Laura E. Jones. Monarch butterfly spatially discrete advection model, Mathematical Biosciences 190 (2004) 183-202

[10] Jia Li, Baojun Song, Xiaohong Wang. An extended discrete Ricker population model with Allee effects. Journal of Difference Equations and Applications, 4(13):309-321,2007.

[11] H. Cao, Y. Zhou, B. Song, 2011. Complex Dynamics of Discrete SEIS Models with Simple Demography, Discrete Dynamics in Nature and Society, vol. 2011. doi:10.1155/2011/653937.

[12] C. Castillo-Chavez, B. Song, Dynamical models of tuberculosis and their applications, Mathematical Biosciences and Engineering, 2(1):361-404, 2004.

[13] J. Dushoff , W. Huang, C. Castillo-Chavez, Backwards bifurcations and catastrophe in simple models of fatald iseases, Journal of Mathematical Biology, 3(36): 227-248, 1998.

[14] S. Ruan, W. Wang, Dynamical behavior of an epidemic model with a nonlinear incidence rate, Journal of D ifferential Equations, 1(88):135-163, 2003.

[15] J. Cui, X. Mu, H. Wan, 2008. Saturation recovery leads to multiple endemic equilibria and backward bifurcation,Journal of Theoretical Biology, 254:275-283.

[16] Z. Hu, S. Liu, H. Wang, 2008. Backward bifurcation of an epidemic model with standard incidence rate and treatment rate, Nonlinear Analysis, 9:2302-2312.

[17] X. Li, W. Li, Mini Ghosh, 2009. Stability and bifurcation of an epidemic model with nonlinear incidence and treatment, Applied Mathematics and Computation,210:141-150.

[18] W. Wang, 2006. Backward bifurcation of an epidemic model with treatment, Mathematical Biosciences,201, 58-71.

[19] X. Zhang, X. Liu, 2008. Bifurcation of an epidemic model with saturated treatment function, J. Math. Anal. Appl., 348:433-443.

[20] P. van den Driessche and J. Watmough, 2002, Reproduction numbers and subthreshold endemic equilibria for compartmental models of disease transmission, Math. Biosc., 180:29-48.

[21] O. Diekmann, J. A. P. Heesterbeek, J. A. J. Metz, On the definition and the computation of the basic reproduction ratio R_0 in models for infectious diseases in heterogeneous populations, J. Math. Biol., 28: 365-382, 1990.

[22] Rongsong Liu, Zhilan Feng, Huaiping Zhu, Donald L. DeAngelis, Bifurcation analysis of a plant-herbivore model with toxin-determined functional response, J. Differential Equations 245 (2008) 442-467.

[23] Baojun. Song, Wen Du, Jie Lou, Different types of backward bifurcations due to density-dependent treatments, Mathematical Biosciences and Engineering.