# Backward bifurcation analysis for two continuous and discrete epidemiological models

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#### **Abstract**

The bifurcation analysis of a continuous *n*-dimensional nonlinear dynamical system with a nonhyperbolic equilibrium point is done by using the main theorem in [4]. We derive an analogue of this theorem for discrete dynamical systems. We design non-standard finite difference (NSFD) schemes for a Susceptible-Infectious-Susceptible (SIS) epidemiological model with vaccination as well as for a malaria model. For the latter model, we sharpen the interval of the values of the disease induced death rate for which backward bifurcation may occur. Applying the discrete theorem, it is shown that each NSFD scheme replicates the property of the continuous model of having backward bifurcation at the value 1 of the basic reproduction number.

## **Keywords**

Epidemiological Model, Reproductive number, Bifurcation Analysis, Center Manifold Theory

#### 1 Introduction

The motivation for this study is that the local qualitative analysis of a nonlinear continuous time dynamical system at an equilibrium point (fixed point or steady state point) is done by Hartman-Grobman linearisation theorem [16] of the system around the equilibrium point whenever it is hyperbolic, (i.e. the real parts of all the eigenvalues of the associated Jacobian matrix are different from zero). In this case, the equilibrium point is locally asymptotically stable (LAS) if all real parts are negative, whereas it is unstable if at least one of the real parts is positive [16]. The problem arises when the equilibrium point is nonhyperbolic i.e. at least one of the real parts is zero. Then no immediate conclusion can be drawn. However, the Center Manifold Theory guarantees the existence and computation of a center manifold for the system under consideration such that the dynamics of the system restricted to the center manifold determine the dynamics of the system [19].

Regarding discrete dynamical systems, often referred to as maps, the analogues of both the Hartman-Grobman theorem and the Center Manifold results apply to the study of the local stability of hyperbolic and nonhyperbolic fixed-point respectively [16, 19].

In this paper we are interested in the backward bifurcation phenomenon. It occurs when a locally asymptotically stable branch of equilibrium changes its stability at the bifurcation point and becomes unstable, while at the same time a new branch of positive unstable steady state emerges to coexist with the initial steady state. Our focus on backward bifurcation is in the context of epidemiology. In this context, backward bifurcation happens when the basic reproduction number  $R_0$  is less than 1 in the sense that a small positive unstable equilibrium appears while the disease free equilibrium and a large positive equilibrium are locally asymptotically stable [4]. Several authors have studied mathematical models which exhibit the backward bifurcation phenomenon, see for instance ([10, 9, 4, 3, 8]). In particular [8] outlines some of the basic causes of the backward bifurcation

phenomenon, which include imperfect vaccine and disease induced mortality on which the two models under consideration in this paper are based.

There are two major challenges when the backward bifurcation occurs. Firstly, if  $R_0$  gets slightly greater than unity a massive number of infectives emerge in the population and this makes it very difficult to control the epidemic. Secondly, reducing  $R_0$  to less than unity is not sufficient to eradicate the disease as is the case when  $R_0 = 1$  is a forward bifurcation. One needs to reduce  $R_0$  until it becomes less than some critical value below which the disease comes under control.

The above mentioned facts will be discussed in the settings of a SIS model with vaccination [18] and a malaria model with disease induced death rate [5]. More precisely, we show that the SIS model undergoes the backward bifurcation at  $R_0 = 1$ . We sharpen the interval of the values of the disease-induced death rate for which the backward bifurcation phenomenon occurs for the malaria model. We extend the theorem in [4] to the study of backward bifurcation for discrete dynamical system. This result is successfully applied to show that two NSFD schemes that we have contructed are dynamically consistent with the backward bifurcation property of the continuous SIS and malaria models. NSFD schemes that replicate the backward bifurcation property of continuous models were also investigated in the works [13] for SIS-Volterra integral equation models and in [7].

The paper is organised as follows: In section 2 we prove the backward bifurcation theorem for discrete dynamical systems. Section 3 is devoted to the backward bifurcation analysis of the SIS continuous model and its NSFD discretisation. A similar analysis is performed for the malaria model with density dependent force of infection in section 4. Finally section 5 provides concluding remarks and discussions as to how our results fit in the literature.

## 2 Bifurcation of discrete dynamical systems

In this section, we are interested in determining the direction of bifurcation for the discrete dynamical system

$$x_{n+1} = f(x_n, \phi). \tag{1}$$

Here and after, the function  $f: \mathbb{R}^n \times \mathbb{R} \to \mathbb{R}^n$  is of class  $C^2$  and it is assumed that x = 0 is a fixed-point of the system(1) for all values of the parameter  $\phi$ :

$$f(0,\phi) \equiv 0$$
 for all  $\phi$ . (2)

To begin with, we recall Theorem 1 below regarding the continuous dynamical system:

$$\frac{dx}{dt} = f(x, \phi),$$
 where the function  $f$  satisfies eqn (2). That is  $x = 0$  is an equilibrium point of system (3) for all values of the parameter  $\phi$ .

where the function f satisfies eqn (2). That is x = 0 is an equilibrium point of system (3) for all values of the parameter  $\phi$ . In what follows, we denote by

$$A = D_x f(0,0), \tag{4}$$

the Jacobian matrix of the function f(x, 0) at the point x = 0.

**Theorem 1** (Castillo-Chavez & Song,[4]). Assume the following:

A1: Zero is a simple eigenvalue of A and all other eigenvalues of A have negative real parts.

A2: Matrix A has a nonnegative right eigenvector  $\mathbf{w}$  and a left eigenvector  $\mathbf{v}$  corresponding to the zero eigenvalue.

Let  $f_k$  be the  $k^{th}$  component of f and

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j} (0,0)$$
 (5)

$$b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi} (0,0)$$
 (6)

where we assume that b > 0. Then the local dynamics of system (3) around x = 0 are determined by the sign of the number a as follows:

- i) If a > 0, then  $\phi = 0$  is a backward bifurcation. More precisely the equilibrium point x = 0 is locally asymptotically stable and there exists a positive unstable equilibrium when  $\phi < 0$ , whereas the equilibrium point x = 0 is unstable and there exists a negative locally asymptotically stable equilibrium when  $0 < \phi < 1$ .
- ii) If a < 0, then  $\phi = 0$  is a forward bifurcation. More precisely when  $\phi$  changes its sign from negative to positive, x = 0 changes its stability from stable to unstable.

Our main theorem reads as follows:

#### Theorem 2. Assume

- 1.  $A = D_x f(0,0) = \left(\frac{\partial f_i}{\partial x_j}(0,0)\right)$  is the linearisation matrix of system (1) around the equilibrium x = 0 with  $\phi$  evaluated at 0. One is a simple eigenvalue of A and no other eigenvalues of A have modulus greater than 1;
- 2. Matrix A has a nonnegative right eigenvector w and a left eigenvector v corresponding to the eigenvalue 1.

The local dynamics of system (1) around the fixed-point x = 0 are determined by the signs of the numbers a and b given in (5) and (6) as in Theorem 1.

*Proof.* The proof is similar to that of Theorem 1 for the continuous model. It is based on the center manifold theory of [19, Sec. 2]. The space  $\mathbb{R}^n$  can be expressed as the direct sum

$$\mathbb{R}^n = E^c \oplus E^s \,, \tag{7}$$

where  $E^c$  is the one-dimensional center subspace and  $E^s$  is the (n-1)-dimensional stable subspace corresponding to the eigenvalue 1 and to eigenvalues with modulus less than 1, respectively. It follows from [19, Thm. 2.1.5] that there exists a center manifold  $W^c(0)$  for the map in system (1), which after parametrisation by  $c \in \mathbb{R}$  is given by:

$$W^{c}(0) = \{(x, \phi) | x(c, \phi) = c\mathbf{w} + h(c, \phi) : \mathbf{v} \cdot h(c, \phi) = 0, |c| \le \overline{c}, c(0) = 0\},$$
(8)

where  $h(c, \phi) \in E^s$  and is of at least order 2 with respect to both c and  $\phi$ .

Consider a solution of (1) initiated at  $x_0 \in W^c(0)$ . Using that  $W^c(0)$  is an invariant set, while  $x_n$  remains sufficiently close to 0 we have  $x_n \in W^c(0)$  that is, there exists  $c_n \in (-\overline{c}, \overline{c})$  such that

$$x_n = c_n \mathbf{w} + h(c_n, \phi) \tag{9}$$

Using (1) we have

$$c_{n+1} + h(c_{n+1}, \phi) = f(c_n \mathbf{w} + h(c_n, \phi))$$
(10)

Multiplying by the left eigenvector  $\mathbf{v}$  we obtain

$$c_{n+1} = \mathbf{v}f(c_n\mathbf{w} + h(c_n, \phi)) \tag{11}$$

From the center manifold theory [19, Thm. 2.1.5] it follows that 0 is asymptotically stable for eqn (1) iff 0 is asymptotically stable fixed point of eqn (11). Hence, we investigate equation (11). By using Taylor's expansion of f about the point  $(x, \phi)$  =

(0,0), eqn (11) becomes

$$c_{n+1} = \mathbf{v} f(0,0) + \mathbf{v} D_{\phi} f(0,0) \phi$$

$$+ \mathbf{v} D_{x} f(0,0) (c_{n} \mathbf{w} + h) + \frac{1}{2} \mathbf{v} D_{\phi\phi} f(0,0) \phi^{2}$$

$$+ \mathbf{v} D_{x\phi} f(0,0) \phi (c_{n} \mathbf{w} + h) + \frac{1}{2} \mathbf{v} D_{xx} f(0,0) (c_{n} \mathbf{w} + h)^{2} + \cdots$$
(12)

In view of eqn (2), we have  $f(0, \phi) \equiv 0$ , and  $D_{\phi} f(0, 0) = 0$ ,  $D_{\phi\phi} f(0, 0) = 0$ . On the other hand

$$\frac{1}{2}D_{xx}f(0,0)(c_n\mathbf{w}+h)^2 = \frac{1}{2}\left[I_n \otimes (c_n\mathbf{w}+h)^T\right]D_{xx}^2f(0,0)(c_n\mathbf{w}+h) 
= \frac{c_n^2}{2}\left[I_n \otimes \mathbf{w}^T\right]D_{xx}^2f(0,0)\mathbf{w} + \cdots,$$
(13)

where  $I_n$  is the identity matrix of order n;  $\otimes$  is the Kronecker product and

$$D_{xx}^{2}f = \begin{pmatrix} \frac{\partial^{2}f_{1}}{\partial x_{1}\partial x_{1}} & \frac{\partial^{2}f_{1}}{\partial x_{1}\partial x_{2}} & \cdots & \frac{\partial^{2}f_{1}}{\partial x_{1}\partial x_{n}} \\ \vdots & \vdots & \ddots & \vdots \\ \frac{\partial^{2}f_{1}}{\partial x_{n}\partial x_{1}} & \frac{\partial^{2}f_{1}}{\partial x_{n}\partial x_{2}} & \cdots & \frac{\partial^{2}f_{1}}{\partial x_{n}\partial x_{n}} \\ \vdots & \vdots & \ddots & \vdots \\ \frac{\partial^{2}f_{n}}{\partial x_{1}\partial x_{1}} & \frac{\partial^{2}f_{n}}{\partial x_{1}\partial x_{2}} & \cdots & \frac{\partial^{2}f_{n}}{\partial x_{1}\partial x_{n}} \\ \vdots & \vdots & \ddots & \vdots \\ \frac{\partial^{2}f_{n}}{\partial x_{n}\partial x_{1}} & \frac{\partial^{2}f_{n}}{\partial x_{n}\partial x_{2}} & \cdots & \frac{\partial^{2}f_{n}}{\partial x_{n}\partial x_{n}} \end{pmatrix}$$
 is the Hessian matrix.

This simplifies eqn (12) into

$$c_{n+1} = \mathbf{v}c_{n}\mathbf{w} + \mathbf{v}D_{x\phi}f(0,0)\phi c_{n}\mathbf{w} + \mathbf{v}\frac{c_{n}^{2}}{2}\left[I_{n}\otimes\mathbf{w}^{T}\right]D_{xx}^{2}f(0,0)\mathbf{w} + \mathcal{O}(3)$$

$$= c_{n} + \mathbf{v}D_{x\phi}f(0,0)\phi c_{n}\mathbf{w} + \frac{c_{n}^{2}}{2}\mathbf{v}\left[I_{n}\otimes\mathbf{w}^{T}\right]D_{xx}^{2}f(0,0)w + \mathcal{O}(3)$$

$$= (1 + \phi b)c_{n} + ac_{n}^{2}$$
(14)

System (1) has dynamics same as those of the simpler map in eqn (14) in the neighbourhood of the equilibrium point  $(x, \phi) = (0, 0)$ , using [19, Thm. 2.1.5] precisely as described in the theorem.

In the next section we consider an epidemiological model. We carry out bifurcation analysis using Theorem 2.

## 3 SIS model with vaccination

In this section, we consider the SIS model proposed in [18]. The system corresponding to the flow diagram in Fig.1 as well as to the variables and parameters in Table 1 reads as follows:

**Table 1.** Model variables and parameters

S	The number of susceptible humans at time t
I	The number of infectious humans at time t
V	Total number of vaccinated humans at time t
N	Total human population at time t
c	Recovery rate
$\sigma$	Transmission rate inhibitor
β	Transmission rate
$\phi_v$	Vaccination rate of the susceptible

$$\dot{S} = -\beta S \frac{I}{N} - \phi_v S + cI$$

$$\dot{I} = \beta S \frac{I}{N} + \sigma \beta V \frac{I}{N} - cI$$

$$\dot{V} = -\sigma \beta V \frac{I}{N} + \phi_v S$$
(15)

It is clear from eqn (15) that the total population N is constant. We eliminate V by using V = N - I - S to obtain the equivalent system

$$\begin{cases} \dot{S} = -\beta S \frac{I}{N} - \phi_v S + cI \\ \dot{I} = \beta S \frac{I}{N} + \sigma \beta (N - S - I) \frac{I}{N} - cI \end{cases}$$
(16)

The disease free equilibrium (DFE)  $(S^*, I^*)$  and the basic reproduction number  $R_0$  are

$$(S^*, I^*) = (0, 0)$$
 and  $R_0 = \frac{\sigma \beta}{c}$ , (17)

respectively. The following result is established in [18];

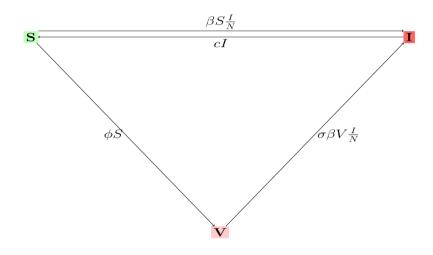


Figure 1. Flow diagram of SIS model

**Theorem 3.** The model (15) undergoes a backward bifurcation at  $R_0 = 1$  if

$$\mathcal{A}(\phi_n) = (1 - \sigma)\beta - \phi_n > 0. \tag{18}$$

Remark 1. There can be no backward bifurcation if the vaccine is absolutely ineffective ( $\sigma = 1$ ). If the vaccine is totally effective ( $\sigma = 0$ ), there is no bifurcation and the DFE is always asymptotically stable.

#### 3.1 A Nonstandard Finite Difference Scheme

We propose the following nonstandard finite difference(NSFD) scheme [14]:

$$\begin{cases}
\frac{S^{n+1} - S^n}{\psi(\Delta t)} = -\beta S^{n+1} \frac{I^n}{N} - \phi_v S^{n+1} + c I^n \\
\frac{I^{n+1} - I^n}{\psi(\Delta t)} = \beta S^{n+1} \frac{I^n}{N} + \sigma \beta (N - S^{n+1} - I^{n+1}) \frac{I^n}{N} - c I^n
\end{cases}$$
(19)

The superscripts in  $S^n$  and  $I^n$  denote the approximations of the exact solution S and I at the discrete time  $t_n = n\Delta t$ ,  $n = 0, 1, 2, \dots, \Delta t$  being the step size. Mickens' rules for the construction of NSFD schemes are fully re-inforced in eqn (19). Instead

of  $\Delta t$  there is a complex denominator function  $\psi(\Delta t)$  of the discrete derivative given by

$$\psi(\Delta t) = 1 - e^{-\Delta t} \tag{20}$$

Also, the nonlinear term SI is approximated in a nonlocal manner see [1, 2, 3, 14]. Upon rearrangement, we obtain

$$S^{n+1} = \frac{S^n + \psi c I^n}{1 + \beta \psi \frac{I^n}{N} + \psi \phi_v} = f_1(x_1, x_2)$$

$$I^{n+1} = \frac{I^n + (1 - \sigma)\psi \beta S^{n+1} \frac{I^n}{N} + \rho c \psi I^n}{1 + \psi \sigma \beta \frac{I^n}{N}} = f_2(x_1, x_2)$$
(21)

where

$$(x_1, x_2) = (S^n, I^n), \quad \rho = R_0 - 1$$
 is the bifurcation parameter, (22)

and the corresponding Jacobian matrix is

$$J = \begin{pmatrix} \frac{1}{\psi\phi_v + 1} & \frac{\psi c}{\psi\phi_v + 1} \\ 0 & \psi\rho c + 1 \end{pmatrix}$$
 (23)

The eigenvalues of J are

$$\lambda_1 = \frac{1}{\psi \phi_{\nu} + 1} < 1$$
 and  $\lambda_2 = \psi c \rho + 1$ 

With  $\rho = 0$ , the right and left eigenvectors associated with  $\lambda_2 = 1$  are

$$\mathbf{w} = \begin{pmatrix} \frac{\sigma \beta}{\phi_v} \\ 1 \end{pmatrix} \text{ and } \mathbf{v} = \begin{pmatrix} 0 \\ 1 \end{pmatrix}. \tag{24}$$

At  $(x_1, x_2, \rho) = (0, 0, 0)$ , we obtain the following result

$$\frac{\partial^2 f_2}{\partial x_1^2} = 0, \quad \frac{\partial^2 f_2}{\partial x_1 \partial x_2} = \frac{\psi \beta (1-\sigma)}{(\psi \phi_v + 1) N}, \quad \frac{\partial^2 f_2}{\partial x_2^2} = \frac{2 \psi^2 \beta^2 \sigma (1-\sigma)}{(\psi \phi_v + 1) N} - \frac{2 \sigma \beta \psi}{N}.$$

If  $\rho = 0$ , i.e.  $R_0 = 1$ , we have  $\lambda_2 = 1$  and so we are in the setting of Theorem 2. Finally, the coefficients a and b in Theorem 2 are given by

$$a = \frac{2\psi\sigma\beta}{N\phi_v}[(1-\sigma)\beta - \phi_v]. \tag{25}$$

and

$$b = \psi c > 0, \tag{26}$$

We have established the following result.

**Theorem 4.** The NSFD scheme (19) is dynamically consistent with respect to the backward bifurcation property of the continuous SIS model with vaccination. That is the discrete SIS model (19) undergoes the backward bifurcation at  $R_0 = 1$  under the condition (18).

The number a in eqn (25) is negative under the condition (27) below. Thus the following result.

Corollary 1. The disease-free equilibrium is globally asymptotically stable if

$$0 < \beta - \phi_v < c. \tag{27}$$

Table	2	Data	set for	SIS	model.
I avic	4.	Data	SCL IOI	. ) [ . )	model.

Parameters	set1	set 2
N	500	500
I(0)	20	0
$\Delta t$	0.5	_
c	0.01	0.02
β	0.02	0.2
$\phi_v$	0.02 & 0.001	0.02
σ	0.02	0.9001 & 0.0999

The diagrams below are plotted using data set in Table 2 above. When  $R_0 = 0.04 < 1$ , Fig. 2 illustrates that the disease may die out or persist depending on wider ( $\phi_v = 0.02$ ) or smaller ( $\phi_v = 0.001$ ) vaccine coverage. Fig. 3 illustrates the results of Theorems 1 and 2. In Fig. 3(a),  $\sigma = 0.9001$  which means that the vaccine is not very effective. The bifurcation in this case is forward. Backward bifurcation phenomenon is shown in Fig. 3(b) with  $\sigma = 0.0999$ ; and the vaccine is very effective. In this scenario we observe the existence of a positive endemic equilibrium even though  $R_0 < 1$ .

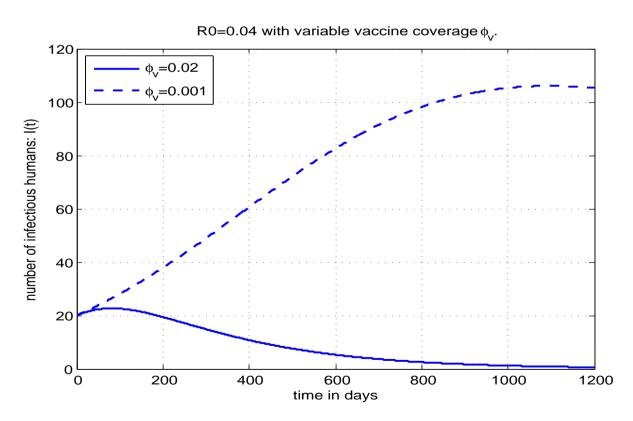
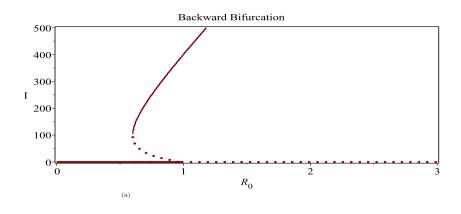
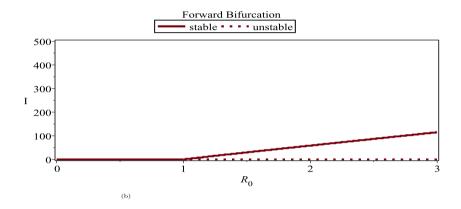


FIGURE 2 Dynamics of the model versus vaccination coverage: Data set 1.





**Figure 3.** Bifurcation direction versus vaccine efficacy (Data set 2):(a) Backward bifurcation (effective vaccine,  $\sigma = 0.0999$ ). (b) Forward bifurcation (ineffective vaccine,  $\sigma = 0.9001$ ).

#### 4 Malaria transmission model

In this section, we apply Theorem 1 to a malaria transmission model developed in [5]. In doing so, we sharpen and prove a conjecture made in this reference, namely that a backward bifurcation occurs for sufficiently large values of the disease-induced death rate. We design a NSFD scheme and show, using Theorem 2, that it is dynamically consistent with the property of backward bifurcation of the continuous model.

#### 4.1 Formulation of the model

The malaria model in [5] is a SEIRS model for the human population and an SI model for the vector population. Human and vector population sizes are assumed to be not constant. The recovered humans develop temporary immunity and become susceptible again. Model parameters are non-negative. All newborns are susceptible to malaria infection. Not all mosquito bites result in infection. Vectors do not die from the infection.  $N_h(t)$  denotes the total human population at time t.  $S_h(t)$  is the number of susceptible humans at time t.  $E_h(t)$  represents the number of exposed humans at time t. The number of infectious humans at time t is denoted by  $I_h(t)$ , and  $R_h(t)$  is the number of recovered humans at time t. The female mosquito population at time t is denoted by  $N_v(t)$ ,  $S_v(t)$  is the number of susceptible mosquitoes at time t,  $E_v(t)$  is the number of exposed mosquitoes at time t, and finally we denote the number of infectious mosquitoes at time t by  $I_v(t)$ . Model parameters are explained in Table 3, and the flow diagram is in Fig. 4. The corresponding model is

$\Lambda_h$	Immigration rate of humans. Humans $\times Time^{-1}$
$ \psi_h $	Per capita birth rate of humans. <i>Time</i> <sup>-1</sup>
$ \psi_v $	Per capita birth rate of mosquitoes. $Time^{-1}$
$\sigma_v$	Number of times one mosquito would want to bite humans per
	unit time, if humans were freely available. $Time^{-1}$
$\sigma_h$	The maximum number of mosquito bites a human can have per
	unit time. $Time^{-1}$
$\beta_{hv}$	Probability of transmission of infection from an infectious mosquito to
	a susceptible human, given that a contact between the two occurs. Dimensionless
$\beta_{vh}$	Probability of transmission of infection from an infectious human to
	a susceptible mosquito, given that a contact between the two occurs. Dimensionless
$ \tilde{eta}_{vh} $	Probability of transmission of infection from a recovered human to
	a susceptible mosquito, given that a contact between the two occurs. Dimensionless
$\nu_h$	Per capita rate of progression of humans from the exposed state to
	the infectious state. $Time^{-1}$
$v_v$	Per capita rate of progression of mosquitoes from the exposed
	state to the infectious state. $Time^{-1}$
$\gamma_h$	Per capita recovery rate for humans from the infectious state to
	the recovered state. $Time^{-1}$
$\delta_h$	Per capita disease induced death rate. $Time^{-1}$
$\rho_h$	Per capita rate of loss of immunity for humans. Time <sup>-1</sup>
$\mu_{1h}$	Density-independent part of the death rate for humans. $Time^{-1}$
$\mu_{2h}$	Density-dependent part of the death rate for humans. $Humans^{-1} \times Time^{-1}$
$\mu_{1v}$	Density-independent part of the death rate for mosquitoes. $Time^{-1}$
$\mu_{2v}$	Density-dependent part of the death rate for mosquitoes.
	$Mosquitoes^{-1} \times Time^{-1}$

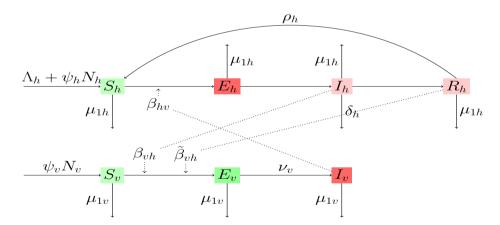


Figure 4. Flow diagram for host-vector dynamics of malaria model

$$\begin{cases} \dot{S}_{h}(t) = \Lambda_{h} + \psi_{h} N_{h}(t) + \rho_{h} R_{h}(t) - c \left(N_{h}(t), N_{v}(t)\right) \beta_{hv} I_{v}(t) S_{h}(t) - f_{h} \left(N_{h}(t)\right) S_{h}(t), \\ \dot{E}_{h}(t) = c \left(N_{h}(t), N_{v}(t)\right) \beta_{hv} I_{v}(t) S_{h}(t) - M_{1} E_{h}(t), \\ \dot{I}_{h}(t) = v_{h} E_{h}(t) - M_{2} I_{h}(t), \\ \dot{R}_{h}(t) = \gamma_{h} I_{h}(t) - M_{3} R_{h}(t), \\ \dot{S}_{v}(t) = \psi_{v} N_{v}(t) - c \left(N_{h}(t), N_{v}(t)\right) \left(\beta_{vh} I_{h}(t) + \tilde{\beta}_{vh} R_{h}(t)\right) S_{v}(t) - f_{v} \left(N_{v}(t)\right) S_{v}(t), \\ \dot{E}_{v}(t) = c \left(N_{h}(t), N_{v}(t)\right) \left(\beta_{vh} I_{h}(t) + \tilde{\beta}_{vh} R_{h}(t)\right) S_{v}(t) - M_{4} E_{v}(t), \\ \dot{I}_{v}(t) = v_{v} E_{v}(t) - f_{v} \left(N_{v}(t)\right) I_{v}(t), \end{cases}$$

$$(28)$$

where

$$M_1 = v_h + f_h$$
,  $M_2 = \gamma_h + \delta_h + f_h$ ,  $M_3 = \rho_h + f_h$ ,  $M_4 = v_v + f_v$ .

The total population sizes  $N_h(t) = S_h(t) + E_h(t) + I_h + R_h(t)$  and  $N_v(t) = S_v(t) + E_v(t) + I_v(t)$ , are such that

$$\dot{N}_h = \Lambda_h + \psi_h N_h - f_h N_h - \delta_h I_h \quad \text{and} \quad \dot{N}_v = \psi_v N_v - f_v N_v$$
 (29)

#### 4.2 Analysis of the Model

It was shown in [5] that model (28) is epidemiologically and mathematically well-posed in the domain:

$$\mathcal{D} = \left\{ (S_h, E_h, I_h, R_h, S_v, E_v, I_v) | (S_h, E_h, I_h, R_h, S_v, E_v, I_v) \ge 0 \right\}, \tag{30}$$

that is for any initial conditions in  $\mathcal{D}$ , the system (28) has a unique solution which remains in  $\mathcal{D}$  for all  $t \ge 0$ . Further, the malaria model (28) has exactly one disease-free equilibrium point,  $x^* = (N_h^*, 0, 0, 0, N_v^*, 0, 0) \in \mathcal{D}$ , where

$$N_h^* = \frac{\psi_h - \mu_{1h} + \left( (\psi_h - \mu_{1h})^2 + 4\mu_{2h}\Lambda_h \right)^{1/2}}{2\mu_{2h}}, \quad N_v^* = \frac{\psi_v - \mu_{1v}}{\mu_{2v}}.$$

Using the notation

$$\begin{split} M_1^* &= \nu_h + f_h^*, \qquad M_2^* = \gamma_h + \delta_h + f_h^*, \qquad M_3^* = \rho_h + f_h^*, \\ M_4^* &= \nu_v + f_v^*, \qquad f_h^* = f_h(N_h^*) = \psi_h + \frac{\Lambda_h}{N_h^*}, \qquad f_v^* = f_v(N_v^*) = \psi_v, \\ c^* &= \frac{\sigma_v \sigma_h}{\sigma_h N_h^* + \sigma_v N_v^*}, \qquad B = \sqrt{(\psi_h - \mu_{1h})^2 + 4\mu_{2h} \Lambda_h}, \end{split}$$

the basic reproduction number derived in [6] may be rewritten as

$$R_{0} = c^{*} \left( \frac{\beta_{hv} \nu_{h} \nu_{v} N_{h}^{*} N_{v}^{*} \left( \beta_{vh} + \frac{\tilde{\beta}_{vh} \gamma_{h}}{M_{3}^{*}} \right)}{f_{v}^{*} M_{1}^{*} M_{2}^{*} M_{4}^{*}} \right)^{1/2}.$$
(31)

The following result regarding the bifurcation at  $R_0 = 1$  was proved in [5].

**Theorem 5.** If there is no disease induced death rate ( $\delta_h = 0$ ), then DFE is globally asymptotically stable for  $R_0 < 1$ . Moreover, the bifurcation at  $R_0 = 1$  is forward bifurcation.

Inspired by the conjecture made in [5], here we extend this result by investigating the properties of the bifurcation at  $R_0 = 1$  when the disease induced death rate is positive. This is done in two scenarios. The first scenario is intuitive. It is considered because the proof is more direct and not based on the center manifold theory. To this end, we observe that the total population  $N_h^{**} \equiv N_h^*(I_h^*)$  of humans at an endemic equilibrium  $I_h^* > 0$  is less than or equal to  $N_h^* \equiv N_h^*(0)$ , which is the total population of humans corresponding to the disease-free equilibrium  $I_h^* = 0$ . For the purpose of investigating directly the existence of backward bifurcation, it is sensible to assume that  $N_h^{**}$  is a linear function in  $I_h^*$  such that

$$N_h^{**} = N_h^* - FI_h^* \quad \text{where} \quad F = \frac{\delta_h}{R}.$$
 (31a)

**Theorem 6.** Suppose that condition (31a) holds and the disease induced death rate satisfies the inequality

$$\delta_h > \frac{\sigma_v \beta_{vh} B}{f_v^*},\tag{32}$$

then system (28) has at least one endemic equilibrium when  $R_0 < 1$ .

*Proof.* Any endemic equilibrium point  $x^{**} = (S_h^{**}, E_h^{**}, I_h^*, R_h^{**}, S_v^{**}, E_v^{**}, I_v^{**})$  satisfies the following equations,

$$\begin{cases}
S_h^{***} = \frac{\Lambda_h + \psi_h N_h^{**} + \rho_h R_h^{**}}{c^{**} \beta_{h\nu} I_v^{**} + f_h^{**}}, \quad E_h^{***} = \frac{c^{**} \beta_{h\nu} I_v^{**} S_h^{**}}{M_1^{**}}, \quad R_h^{**} = \frac{\gamma_h I_h^*}{M_3^{**}}, \\
S_v^{***} = \frac{\psi_v N_v^{**}}{c^{**} (\beta_{\nu h} I_h^{*} + \tilde{\beta}_{\nu h} R_h^{**}) + f_v^{**}}, \quad E_v^{***} = \frac{c^{**} (\beta_{\nu h} I_h^{*} + \tilde{\beta}_{\nu h} R_h^{**}) S_v^{**}}{M_4^{**}}, \quad I_v^{***} = \frac{\nu_v E_v^{**}}{f_v^{**}}.
\end{cases} \tag{33}$$

At the endemic equilibrium we have

$$c^{**} = \frac{c^*}{1 - DI_h^*}, f_h^{**} = f_h^* - AI_h^*, M_1^{**} = M_1^* - AI_h^*, M_2^{**} = M_2^* - AI_h^*, M_3^{**} = M_3^* - AI_h^*, M_4^{**} = M_4^*, \tag{34}$$

where

$$B = \sqrt{(\psi_h - \mu_{1h})^2 + 4\mu_{2h}\Lambda_h}, \quad A = \frac{\mu_{2h}\delta_h}{B}, \quad D = \frac{\sigma_h\delta_h}{B(\sigma_v N_v^* + \sigma_h N_h^*)}, \quad E = \left(\beta_{vh} + \frac{\gamma_h\tilde{\beta}_{vh}}{M_3^*}\right), \quad F = \frac{\delta_h}{B}. \tag{35}$$

Using

$$v_h E_h^{**} - M_2^{**} I_h^* = 0 (36)$$

we obtain that  $I_h^*$  is a root of algebraic equation of the form

$$A_{7}I_{h}^{*7} + A_{6}I_{h}^{*6} + A_{5}I_{h}^{*5} + A_{4}I_{h}^{*4} + A_{3}I_{h}^{*3} + A_{2}I_{h}^{*2} + A_{1}I_{h}^{*} + A_{0} = 0$$

$$(37)$$

Explicit expressions of the coefficients,  $A_0, \dots, A_7$  can be derived in terms of the parameters of the model. Specifically for  $A_0$  and  $A_7$ , which we use in the sequel we have

$$\begin{cases} A_0 = M_1^* M_2^* M_3^{*2} M_4^* f_h^* f_v^* (R_0^2 - 1) \\ A_7 = A^5 f_v^* M_4^* D^2 - c^* M_4^* A^5 D \beta_{vh} = A^5 M_4^* D (D f_v^* - c^* \beta_{vh}) \end{cases}$$
(38)

Using eqn (32) and (35) it is easy to see that  $A_7 > 0$ . Let  $R_0 < 1$ . Then clearly  $A_0 < 0$ . Hence the eqn (37) has at least one positive root. That is, for  $R_0 < 1$  the stable DFE co-exists with an endemic equilibrium which approaches DFE as  $R_0 \to 1$ .

## 4.3 Bifurcation Analysis via Center Manifold Theory

We investigate the nature of the bifurcation by using Theorem 1. Let

$$\zeta_{1} = \left(\frac{M_{1}^{*} M_{2}^{*} M_{4}^{*} f_{v}^{*}}{\beta_{hv} \nu_{h} \nu_{v} N_{h}^{*} N_{v}^{*} \left(\beta_{vh} + \frac{\tilde{\beta}_{vh} \gamma_{h}}{M_{3}^{*}}\right)}\right)^{1/2},$$
(39)

If  $c^* < \zeta_1$  the DFE is locally asymptotically stable and unstable when  $c^* > \zeta_1$ . Thus,  $c^*$  is a bifurcation parameter. In order to illustrate the role of the disease induced death rate  $\delta_h$  we consider eqn (28) in its equivalent form where the equations of the populations of susceptible individuals  $S_h$  and  $S_v$  are replaced by the eqns in (29) of the total populations  $N_h$  and  $N_v$  respectively. For convenience we introduce the following notation

$$\begin{cases} \mathbf{x} = (x_1, x_2, x_3, x_4, x_5, x_6, x_7)^T = (N_h, E_h, I_h, R_h, N_v, E_v, I_v)^T \\ \dot{\mathbf{x}} = (f_1, f_2, f_3, f_4 f_5, f_6, f_7)^T = f(\mathbf{x}, c) \end{cases}$$
(40)

The Jacobian matrix of system (40) evaluated at DFE is given by

$$J = \begin{pmatrix} -B & 0 & -\delta_h & 0 & 0 & 0 & 0 \\ 0 & -M_1^* & 0 & 0 & 0 & 0 & c^*\beta_{hv}N_h^* \\ 0 & v_h & -M_2^* & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma_h & -M_3^* & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -(f_v^* - \mu_{1v}) & 0 & 0 \\ 0 & 0 & c^*\beta_{vh}N_v^* & c^*\tilde{\beta}_{vh}N_v^* & 0 & -M_4^* & 0 \\ 0 & 0 & 0 & 0 & 0 & v_v & -f_v^* \end{pmatrix}$$

$$(41)$$

The characteristic polynomial of the matrix J is given by

$$\begin{cases} |J - \lambda I| = (\lambda + B)(\lambda + (f_v^* - \mu_{1v})) \left\{ \lambda^5 + \lambda^4 (f_v^* + B_0) + \lambda^3 (f_v^* B_0 + B_1) + \lambda^2 (f_v^* B_1 + B_2) + \lambda (f_v^* B_2 + M_1^* M_2^* M_3^* M_4^*) - \lambda c^{*2} \beta_{hv} \beta_{vh} N_h^* N_v^* v_v v_h + M_1^* M_2^* M_3^* M_4^* f_v^* (1 - R_0^2) \right\} = 0, \end{cases}$$

$$(42)$$

where

$$\begin{cases} B_0 = (M_3^* + M_4^*) + (M_2^* + M_1^*), & B_1 = M_1^* M_2^* + (M_1^* + M_2^*)(M_3^* + M_4^*) + M_4^* M_3^*, \\ B_2 = M_4^* M_3^* (M_1^* + M_2^*) + M_1^* M_2^* (M_3^* + M_4^*). \end{cases}$$
(43)

In what follows we show that the coefficient of  $\lambda^1$  is positive when  $R_0 \le 1$ . Let  $R_0 \le 1$ , then

$$c^{*2}\beta_{hv}\nu_{h}\nu_{v}N_{h}^{*}N_{v}^{*}\left(\beta_{vh}+\frac{\tilde{\beta}_{vh}\gamma_{h}}{M_{3}^{*}}\right)\leq M_{1}^{*}M_{2}^{*}M_{4}^{*}f_{v}^{*}\Rightarrow c^{*2}\nu_{h}\nu_{v}N_{h}^{*}N_{v}^{*}\beta_{hv}\beta_{vh} < M_{1}^{*}M_{2}^{*}M_{4}^{*}f_{v}^{*} < f_{v}^{*}B_{2}. \tag{44}$$

Thus,

$$(f_{\nu}^* B_2 + M_1^* M_2^* M_3^* M_4^*) - c^{*2} \nu_h \nu_{\nu} N_h^* N_{\nu}^* \beta_{h\nu} \beta_{\nu h} > 0.$$

$$(45)$$

Since it assumed in [5] that  $f_v^* > \mu_{1v}$ , it is easy to see that  $\lambda = -B$  and  $\lambda = -(f_v^* - \mu_{1v})$  are negative eigenvalues of J. We now use matrix theory [6, 15] to investigate the eigenvalues of the following irreducible Metzler matrix

$$J^* = \begin{pmatrix} -M_1^* & 0 & 0 & 0 & c^* \beta_{hv} N_h^* \\ v_h & -M_2^* & 0 & 0 & 0 \\ 0 & \gamma_h & -M_3^* & 0 & 0 \\ 0 & c^* \beta_{vh} N_v^* & c^* \tilde{\beta}_{vh} N_v^* & -M_4^* & 0 \\ 0 & 0 & 0 & v_v & -f_v^* \end{pmatrix}. \tag{46}$$

When  $c^* = \zeta_1$  we have  $R_0 = 1$ , and the matrix  $J^*$  admits a simple zero eigenvalue and all the other eigenvalues have negative real parts by Lemmas 1 and 2 below.

**Lemma 1** (Mitkowski,[15]). For any Metzler matrix  $M \in \mathbb{R}^{n \times n}$  with spectrum  $\sigma(M)$ , there exists a real number  $\lambda_{\max} \in \sigma(M)$  such that  $\lambda_{\max} = \max Re(\lambda_i) \ \forall \ i = 1, 2, \dots, n$ .

**Lemma 2** (Mitkowski,[15]). Let  $A = [a_{ij}]$  be a Metzler matrix, i.e.  $a_{ij} \ge 0$ ,  $i \ne j$ . Let  $det(\lambda I - A) = \lambda^n + a_{n-1}\lambda^{n-1} + \cdots + a_1\lambda + a_0$ . Then  $\alpha(A) < 0$  or  $(Re(\lambda_i) < 0, i = 1, 2, \cdots, n)$  iff  $a_i > 0$ ,  $i = 0, 1, 2, \cdots, n-1$ .

The coefficients a and b are computed by letting

$$\mathbf{w} = (w_1, w_2, w_3, w_4, w_5, w_6, w_7)^T \text{ and } \mathbf{v} = (v_1, v_2, v_3, v_4, v_5, v_6, v_7)$$
(47)

be the right and left eigenvectors associated with the zero eigenvalue, respectively. Upon computation the following vector components were found

$$\begin{cases} w_{1} = -\frac{\delta_{h} v_{h} c^{*} \beta_{hv} N_{h}^{*}}{B M_{2}^{*} M_{1}^{*}} w_{7}, & w_{2} = \frac{c^{*} \beta_{hv} N_{h}^{*}}{M_{1}^{*}} w_{7}, & w_{3} = \frac{v_{h} c^{*} \beta_{hv} N_{h}^{*}}{M_{2}^{*} M_{1}^{*}} w_{7}, & w_{4} = \frac{\gamma_{h} v_{h} c^{*} \beta_{hv} N_{h}^{*}}{M_{3}^{*} M_{2}^{*} M_{1}^{*}} w_{7}, & w_{5} = 0, \\ w_{6} = \frac{f_{v}^{*}}{v_{v}} w_{7}, & w_{7} = w_{7} > 0. \end{cases}$$

$$(48)$$

$$\begin{cases} v_{1} = 0, \quad v_{2} = \frac{f_{v}^{*}}{c^{*}\beta_{hv}N_{h}^{*}}v_{7}, \quad v_{3} = \frac{M_{1}^{*}f_{v}^{*}}{v_{h}c^{*}\beta_{hv}N_{h}^{*}}v_{7}, \quad v_{4} = \frac{c^{*}\tilde{\beta}_{vh}N_{v}^{*}v_{v}}{M_{3}^{*}M_{4}^{*}}v_{7}, \quad v_{5} = 0, \\ v_{6} = \frac{v_{v}}{M_{4}^{*}}v_{7}, \quad v_{7} = v_{7} > 0. \end{cases}$$

$$(49)$$

We ignore the partial derivatives of  $f_1$  and  $f_5$  because  $v_1 = 0 = v_5$ . The non zero second order partial derivatives are the following

$$\begin{cases}
\frac{\partial^{2} f_{2}}{\partial x_{1} \partial x_{7}} = c^{*} \beta_{hv}, & \frac{\partial^{2} f_{2}}{\partial x_{2} \partial x_{7}} = -c^{*} \beta_{hv}, & \frac{\partial^{2} f_{2}}{\partial x_{3} \partial x_{7}} = -c^{*} \beta_{hv}, & \frac{\partial^{2} f_{2}}{\partial x_{4} \partial x_{7}} = -c^{*} \beta_{hv}, \\
\frac{\partial^{2} f_{6}}{\partial x_{4} \partial x_{6}} = -c^{*} \tilde{\beta}_{vh}, & \frac{\partial^{2} f_{6}}{\partial x_{4} \partial x_{7}} = -c^{*} \tilde{\beta}_{vh}, & \frac{\partial^{2} f_{6}}{\partial x_{3} \partial x_{7}} = -c^{*} \beta_{vh}, & \frac{\partial^{2} f_{6}}{\partial x_{3} \partial x_{6}} = -c^{*} \beta_{vh}, \\
\frac{\partial^{2} f_{2}}{\partial x_{1} \partial x_{2}} = -\mu_{2h}, & \frac{\partial^{2} f_{3}}{\partial x_{1} \partial x_{3}} = -\mu_{2h}, & \frac{\partial^{2} f_{4}}{\partial x_{1} \partial x_{4}} = -\mu_{2h}, \\
\frac{\partial^{2} f_{2}}{\partial c^{*} \partial x_{7}} = N_{h}^{*} \beta_{hv}, & \frac{\partial^{2} f_{6}}{\partial x_{3} \partial c^{*}} = N_{v}^{*} \beta_{vh}, & \frac{\partial^{2} f_{6}}{\partial x_{4} \partial c^{*}} = N_{v}^{*} \tilde{\beta}_{vh},
\end{cases}$$
(50)

From above expressions we obtain

$$a = 2v_{7}w_{7}^{2} \left\{ \frac{\delta_{h}f_{v}^{*}v_{h}c^{*}\beta_{hv}}{BM_{2}^{*}M_{1}^{*}} \left( \frac{\mu_{2h}N_{h}^{*}(M_{1}^{*} + M_{2}^{*})}{M_{1}^{*}M_{2}^{*}} - 1 \right) - \frac{f_{v}^{*}c^{*}\beta_{hv}}{M_{1}^{*}} \left( 1 + \frac{v_{h}}{M_{2}^{*}} + \frac{\gamma_{h}v_{h}}{M_{3}^{*}M_{2}^{*}} \right) - \frac{v_{h}c^{*2}\beta_{hv}N_{h}^{*}}{M_{2}^{*}M_{1}^{*}} \left( \beta_{vh} + \frac{\gamma_{h}\tilde{\beta}_{vh}}{M_{3}^{*}} \right) + \frac{\delta_{h}c^{*3}\tilde{\beta}_{vh}N_{v}^{*}v_{v}v_{h}^{2}\beta_{hv}^{2}N_{h}^{*2}\gamma_{h}\mu_{2h}}{BM_{4}^{*}M_{3}^{*2}M_{2}^{*2}M_{1}^{*2}} \right\}$$

$$= 2v_{7}w_{2}^{2}\Phi^{*} \left( \Pi^{*} - 1 \right).$$

$$(51)$$

and

$$b = \frac{2f_v^*}{c^*} \nu_7 w_7 > 0. \tag{52}$$

$$\begin{cases} \Pi^* = \frac{\frac{\delta_h f_v^* v_h c^* \beta_{hv} \mu_{2h} N_h^* (M_1^* + M_2^*)}{B M_2^{*2} M_1^{*2}} + \frac{\delta_h c^{*3} \tilde{\beta}_{vh} N_v^* v_v v_h^2 \beta_{hv}^2 N_h^{*2} \gamma_h \mu_{2h}}{B M_4^* M_3^{*2} M_2^{*2} M_1^{*2}} \\ \text{and} \\ \Phi^* = \frac{\delta_h f_v^* v_h c^* \beta_{hv}}{B M_2^* M_1^*} + \frac{f_v^* c^* \beta_{hv}}{M_1^*} \left(1 + \frac{v_h}{M_2^*} + \frac{\gamma_h v_h}{M_3^* M_2^*}\right) + \frac{v_h c^{*2} \beta_{hv} N_h^*}{M_2^* M_1^*} \left(\beta_{vh} + \frac{\gamma_h \tilde{\beta}_{vh}}{M_3^*}\right). \end{cases}$$
(53)

Then using Theorem 1, we obtain the following characterization of the bifurcation at  $R_0 = 1$ .

**Theorem 7.** The malaria model (28) exhibits a backward bifurcation at  $R_0 = 1$  if  $\delta_h$  is large enough in the sense that  $\Pi^* > 1$  and a forward bifurcation if  $\Pi^* < 1$ .

Remark 2. It should be noted that when  $\delta_h = 0$ , the bifurcation at  $R_0 = 1$  is supercritical because a < 0.

#### 4.4 NSFD Scheme

In this section, we construct a NSFD scheme that replicates among other things the backward bifurcation of the malaria model (28) in its equivalent form where the equations of the susceptible populations are replaced by equations of total species

populations in eqn (29). The NSFD scheme reads as follows:

$$\begin{cases}
\frac{N_{h}^{n+1} - N_{h}^{n}}{\phi} = \Lambda_{h} + \psi_{h} N_{h}^{n} - f_{h} N_{h}^{n} - \delta_{h} I_{h}^{n} \\
\frac{E_{h}^{n+1} - E_{h}^{n}}{\phi} = c \beta_{hv} I_{v}^{n} S_{h}^{n+1} - M_{1} E_{h}^{n} \\
\frac{I_{h}^{n+1} - I_{h}^{n}}{\phi} = v_{v} E_{h}^{n} - M_{2} I_{h}^{n} \\
\frac{R_{h}^{n+1} - R_{h}^{n}}{\phi} = \gamma_{h} I_{h}^{n} - M_{3} R_{h}^{n} \\
\frac{N_{v}^{n+1} - N_{v}^{n}}{\phi} = \psi_{v} N_{v}^{n} - f_{v} N_{v}^{n} \\
\frac{E_{v}^{n+1} - E_{v}^{n}}{\phi} = c (\beta_{vh} I_{h}^{n} + \tilde{\beta}_{vh} R_{h}^{n}) S_{v}^{n+1} - M_{4} E_{v}^{n} \\
\frac{I_{v}^{n+1} - I_{v}^{n}}{\phi} = v_{v} E_{v}^{n} - f_{v} I_{v}^{n}
\end{cases}$$
(54)

The standard denominator  $\Delta t$  is replaced by the function

$$\phi = \phi(\Delta t) = \frac{1 - e^{-q\Delta t}}{q} \quad \text{where} \quad q \ge \max\left\{f_h, f_v, M_1, M_2, M_3, M_4, \frac{|\lambda|^2}{2|Re\lambda|}\right\},\tag{55}$$

and  $\lambda$  represents all the eigenvalues of the Jacobian matrix in (41) with negative real parts. For an alternative NSFD scheme for the model eqn (28), we refer the reader to [3]. The scheme (54) can be written in the following explicit form

$$\begin{cases} N_{h}^{n+1} = \phi \Lambda_{h} + \phi \psi_{h} N_{h}^{n} + (1 - \phi f_{h}) N_{h}^{n} - \phi \delta_{h} I_{h}^{n} \\ E_{h}^{n+1} = \frac{\phi c \beta_{hv} I_{v}^{n} (N_{h}^{n+1} - I_{h}^{n+1} - R_{h}^{n+1}) + (1 - \phi M_{1}) E_{h}^{n}}{1 + c \phi \beta_{hv} I_{v}^{n}} \\ I_{h}^{n+1} = \phi v_{h} E_{h}^{n} + (1 - \phi M_{2}) I_{h}^{n} \\ R_{h}^{n+1} = \phi \gamma_{h} I_{h}^{n} + (1 - \phi M_{3}) R_{h}^{n} \\ N_{v}^{n+1} = \phi \psi_{v} N_{v}^{n} + (1 - \phi f_{v}) N_{v}^{n} \\ E_{v}^{n+1} = \frac{\phi c (\beta_{vh} I_{h}^{n} + \tilde{\beta}_{vh} R_{h}^{n}) (N_{v}^{n+1} - I_{v}^{n+1}) + (1 - \phi M_{4}) E_{v}^{n}}{1 + \phi c (\beta_{vh} I_{h}^{n} + \tilde{\beta}_{vh} R_{h}^{n})} \\ I_{h}^{n+1} = \phi v_{v} E_{v}^{n} + (1 - \phi f_{v}) I_{h}^{n} \end{cases}$$

The Jacobian matrix of the map in (56) evaluated at the DFE is given by

$$J_{NS} = \begin{pmatrix} 1 - \phi B & 0 & -\phi \delta_h & 0 & 0 & 0 & 0 \\ 0 & 1 - \phi M_1^* & 0 & 0 & 0 & 0 & \phi c^* \beta_{hv} N_h^* \\ 0 & \phi \nu_h & 1 - \phi M_2^* & 0 & 0 & 0 & 0 \\ 0 & 0 & \phi \gamma_h & 1 - \phi M_3^* & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 - \phi (f_v^* - \mu_{1v}) & 0 & 0 \\ 0 & 0 & \phi c^* \beta_{vh} N_v^* & \phi c^* \tilde{\beta}_{vh} N_v^* & 0 & 1 - \phi M_4^* & 0 \\ 0 & 0 & 0 & 0 & 0 & \phi \nu_v & 1 - \phi f_v^* \end{pmatrix} = I + \phi J, \tag{57}$$

where I is the identity matrix. Taking into consideration the fact that by eqn (55)  $\phi < \frac{2|Re\lambda|}{|\lambda|^2}$ , for  $\lambda \neq 0$ , we therefore conclude that  $\lambda_{NS} = 1$  is an eigenvalue of  $J_{NS}$  and all the other eigenvalues  $\lambda_{NS} = 1 + \phi\lambda$  are such that  $|\lambda_{NS}| < 1$ . We are therefore in the setting of Theorem 2.

The right and the left eigenvectors associated with eigenvalue 1 are the same as those in eqns (48) and (49) respectively. The

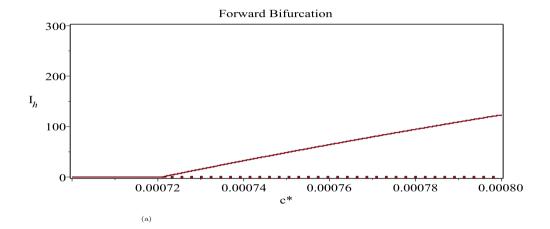
coefficients that determine the direction of the bifurcation at  $R_0 = 1$  are

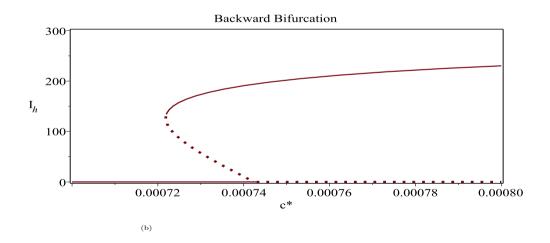
$$\tilde{a} = \phi a$$
 and  $\tilde{b} = \phi b > 0$  (58)

where a and b are defined in eqns (51) and (52). Then using Theorem 2, we obtain the following result which shows that the numerical scheme (54) replicates correctly the properties of the bifurcation for model (28) as stated in Theorem 8.

**Theorem 8.** The discrete scheme (54) exhibits a backward bifurcation at  $R_0 = 1$  if  $\Pi^* > 1$  and a forward bifurcation if  $\Pi^* < 1$ .

Bifurcations diagrams are plotted using the data set in [5]; for convenience we reproduce it in Table. 4 below. The diagram in Fig. 5(a) depicts a forward bifurcation when the disease-induced death rate  $\delta_h = 0.3419 \times 10^{-4}$  which is much less than the threshold in Theorem 6 and  $R_0 = 1.0289$ . In this case there is a unique stable endemic equilibrium. The coexistence of the asymptotically stable disease free equilibrium with a small unstable endemic equilibrium and a larger stable endemic equilibrium is illustrated in Fig. 5(b) with  $\delta_h = 2.7 \times 10^{-4}$  and  $R_0 = 0.9988$ . The latter is known as the backward bifurcation phenomenon.





**Figure 5.** Bifurcation direction versus disease induced death rate: (a) Forward bifurcation ( $\delta_h = 0.3419 \times 10^{-4}$ ). (b) Backward bifurcation ( $\delta_h = 2.7 \times 10^{-4}$ ).

Table 4. Data set for Malaria model.

Parameters	set1
$\Lambda_h$	$3.285 \times 10^{-2}$
$\psi_h$	$7.666 \times 10^{-4}$
$\psi_v$	0.4
$eta_{vh}$	0.8333
$ ilde{eta}_{vh}$	$8.333 \times 10^{-2}$
$\sigma_v$	0.6
$\sigma_h$	18
$v_{1h}$	$8.333 \times 10^{-2}$
$\nu_v$	0.1
$\gamma_h$	$3.704 \times 10^{-3}$
$ ho_h$	$1.460 \times 10^{-2}$
$\mu_h$	$4.212 \times 10^{-5}$
$\mu_{2h}$	$10^{-7}$
$\mu_{1v}$	0.1429
$\mu_{2v}$	$2.279 \times 10^{-4}$
$oldsymbol{eta}_{hv}$	$2 \times 10^{-2}$

## 5 Discussion and conclusion

This paper is motivated by the three facts below.

- (i) A conjecture in [5] regarding the value of the disease induced death rate for their malaria model to undergo the backward bifurcation phenomenon.
- (ii) The need to determine as for the continuous dynamical systems the direction of bifurcation in the discrete dynamical systems for which the linearisation process does not apply.
- (iii) The implementation of item (ii) above for the Nonstandard finite difference schemes such as the one proposed in [3] for the malaria model.

To address item (i), we determined a threshold for the disease induced death rate above which the malaria model undergoes the backward bifurcation phenomenon. With regards to item (ii) and (iii), we derived a discrete analogue (Theorem 2) of the theorem in [4] for the bifurcation analysis by Center Manifold Theory. Two NSFD schemes were contructed for the SIS model in [18] as well as for the malaria model in [5]. We proved that Theorem 2 works for the NSFD schemes as does Theorem 1 for the continuous models.

Our plan for future research is to apply Theorem 2 to some discrete models for infectious diseases, which are not numerical approximations of continuous models (for instance see [11]).

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