Centre Manifold Theory for some Continuous and Discrete Epidemiological Models

by

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DEDICATION

This thesis is dedicated to the memory of my late mother, Victress Thandeka Dukuza, who was so fond of education. The main reason for dedicating the thesis to her memory is that she gave me money to buy my first year university mathematics textbook: *George B. Thomas, Ross L. Finney, Calculus and Analytic Geometry (9th Edition)*, which was the prescribed textbook, just days before she passed on. Little did I know that was to be my final interaction with her still alive, and of course her last and everlasting gift for me. May her soul rest in peace as this work sits at the pinnacle of the National Qualifications Framework levels of the Republic of South Africa.

DECLARATION

I, the undersigned, declare that this thesis, which is submitted for the degree Philosophiae Doctor at the University of Pretoria, is my own independent research. This work has not been submitted, in whole or in part, at any other university.

Signature: _____

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Date: 24 July 2019

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LIST OF ABBREVIATIONS

$A.D.$	Anno Domini
$AIDS$	Acquired Immunodeficiency Syndrome
$B.C.$	Before Christ
CDC	Centre for Disease Control and Prevention
CMT	Centre Manifold Theory
DFE	Disease-free Equilibrium
$DHET$	Department of Higher Education and Training
DST	Department of Science and Technology
EE	Endemic Equilibrium
GAS	Globally Asymptotically Stable
HIV	Human Immunodeficiency Virus
LAS	Locally Asymptotically Stable
$nGAP$	New Generation of Academics Programme
$NICD$	National Institute of Communicable Diseases
NRF	National Research Foundation
$NSFD$	Nonstandard Finite Difference
$SARChI$	South African Research Chairs Initiative
$SEIS$	Susceptible-Exposed-Infected-Susceptible
SIS	Susceptible-Infected-Susceptible
STD	Sexually Transmitted Disease
TB	Tuberculosis
$U.N.$	United Nations
WHO	World Health Organization

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ABSTRACT

In mathematical epidemiology, the threshold theory introduced by W.O. Kermack and A.G. McKendrick (1927) can be expressed in terms of the basic reproduction number R_0. This is defined as the average number of secondary infections that occur when one infective is introduced into a susceptible host population. In this setting and for many diseases, the prediction of the likelihood of persistence or dying out of the disease within the population reads as follows: the disease-free equilibrium is locally asymptotically stable (LAS) when $R_0 < 1$, it is unstable when $R_0 > 1$ and at least one endemic equilibrium (EE) which is LAS is born in this case. In other words, at $R_0 = 1$, a forward bifurcation occurs.

However, some diseases undergo the backward bifurcation phenomenon whereby, for $R_0 < 1$, the LAS disease-free equilibrium coexists with a small positive unstable EE and a large positive LAS EE.

In this thesis, we study theoretically, numerically, and computationally the existence of the backward bifurcation phenomenon for dynamical systems, with emphasis on a "simple" SIS model with vaccination and a "complex" malaria model. We re-centre the reduction theorem in C. Castillo-Chavez and B. Song (2004) and highlight its advantage over the legendary power series approximations in the use of the Centre Manifold Theory (CMT). We propose and prove a Centre Manifold-based theorem for the existence of a backward bifurcation for discrete dynamical systems. We construct nonstandard finite difference (NSFD) schemes and prove that they preserve the backward bifurcation property of the continuous models.

We make the results more specific for the SIS and malaria models for which we also pro-

vide numerical simulations that support the theory. In particular we prove for the malaria model a conjecture by Chitnis et al. (2006) for the existence of the backward bifurcation.

CHAPTER 1. INTRODUCTION

1.1 Brief history of diseases and mathematical epidemiology

Since time immemorial, the development of mankind has often been curtailed by severe diseases. According to Hays [45], diseases have a long history dating as far back as 430 B.C. during the Peloponnesian war. Since then, several pandemics have been recorded with the following timelines (see [45]): Antonine Plague (165 A.D.), Cyprian Plague (250 A.D.), Justinian Plague (541 A.D.), Leprosy (11th century), The Black Death (1350), Fiji Measles (1875), Yellow fever and malaria (1881), Russian Flu (1889), Spanish Flu (1918), Asian Flu (1957) etc. Fevers related to malaria were reported as early as the fifth century B.C. in Greece and Rome. The most common categories of known diseases are the communicable (infectious) and noncommunicable (noninfectious) diseases. The list presented above is endless. As reported in the edited book by Castillo-Chavez et al. [18] on emerging and re-emerging infectious diseases, there is a recurrence of new diseases and old forms of new diseases, such as cancer, HIV/AIDS, malaria, ebola, typhoid fever, cholera etc. that pose a massive threat to the development of Africa and beyond.

It is clear that diseases have survived and will continue to survive the test of time. For this reason, the control and management of diseases became such an important priority after the World War 2 that the United Nations (UN) created the World Health Organization (WHO) on 7 April 1948. Its first mandate was to assume an advisory role and compile reliable statistics on the transmission, coverage, and morbidity rate of the following then prioritised matters: Tuberculosis (TB), malaria, venereal diseases (also known as STDs), maternal and child health, nutrition and environmental hygiene (see for instance WHO [81]). This initial

mandate has expanded significantly to the extent that WHO is nowadays the compelling reference regarding health issues such as public health policies, outbreaks of diseases etc. A report issued by the WHO [82] indicates that TB disease accounted for an estimated 1.3 million deaths in 2017 among HIV negative people alone. On the other hand, malaria remains a burden on many countries as it is reported by the WHO [83] that in 2017, a total of 45.6 million cases of infection were confirmed in the Eastern and Southern Africa region alone. It is not astonishing that the situation is worse in developing parts of the world such as Africa, where new diseases such as ebola, with a very high morbidity rate, emerge from time to time WHO [84].

The discovery of more efficient disease control strategies by means of clinical research through Government Departments of Health and Institutes or Centres for Control of Diseases (e.g. South Africa's National Institute for Communicable Diseases (NICD), United States' Centres for Disease Control and Prevention (CDC)) remains imperative. However, these efforts alone are far from sufficient given the complex nature of the dynamics of diseases such as HIV/AIDS, malaria, cancer to mention just a few. These complexities have led to a need for much more multidisciplinary approaches to scientific research, a strategy that was adopted in the works of Malthus [58], Ross [74], Hamer [43], and Kermack and McKendrick [51] who were rather Physicians and Biologists. As part of this endeavour, mathematicians have joined in and as a result the field of mathematical epidemiology came into being.

Mathematical epidemiology has a long history dating far back to as early as the 18th century in the pioneering work of Bernoulli [11]. Since then, there has been a surge in the development of mathematical models with the aim of giving insight into the critical dynamics of infectious diseases (see Brauer [12], Murray [67], Smith et al. [76], Bailey [10], Malthus [58], Anderson and May [2]). Mathematical models seek to strike a balance between effective disease control measures and their optimal cost given the fact that disease control and prevention mechanisms are typically associated with huge demands for budget. Hence,

mathematical models play an important role in influencing the policy direction of governments and institutions concerned. Depending on the nature and dynamics of the disease, mathematical models often give insight into cost effective strategies. A typical example, is the conclusion which came from a relatively simple malaria prevention model by Ross [74]. The main deduction from the model was that, *reducing the population of mosquitoes below a certain threshold could bring malaria under control.* Even nowadays, the threshold concept, as stated in Principles 1.1.3 and 1.1.4 below, still plays a major role in optimal disease control.

The pioneering works of non-mathematicians mentioned above need to be pointed out again because they layout the three main principles of mathematical modelling in epidemiology as outlined below (see for instance Anderson and May [2]).

Principle 1.1.1. *(Malthus law, Malthus [58])*
For a closed population, the rate of change of its size is proportional to the size of the population.

Principle 1.1.2. *(Mass-Action, Hamer [43] and Ross [74])*
The spread of infection is proportional to the product of the susceptible individuals and the infectious individuals. It should be noted that the mass-action principle (Principle 1.1.2) is often replaced by the standard incidence law, which for several diseases, reflects better the reality of the spread of diseases (Hethcote [46],[47]).

Principle 1.1.3. *(Threshold theory, Ross [74] and Kermack and McKendrick [51])*
The introduction of a few infectious individuals into a community of susceptibles will not give rise to an epidemic outbreak unless the number of susceptibles is above a certain critical value.

In mathematical epidemiology, the threshold theory is stated in terms of the basic reproduction number R_0 as follows:

Principle 1.1.4. *(i.e. Principle 1.1.3 via R_0)*

If $R_0 < 1$, then the disease dies out in the sense that the disease-free equilibrium is locally asymptotically stable. If $R_0 > 1$, then the disease-free equilibrium is unstable and the disease is endemic.

Note that the basic reproduction number R_0 is defined as the average number of secondary infections arising from a single infectious individual during his or her entire infectious period in a population of susceptibles (see Anderson and May [2], Hethcote [47]).

It has been observed that the threshold theory as stated above does not hold for some diseases. More precisely, the backward bifurcation phenomenon can happen. This phenomenon means the following: *Though the basic reproduction number is less than 1, there exist a small positive unstable equilibrium and a large positive locally asymptotically stable (LAS) equilibrium, while the disease-free equilibrium is LAS* (see Huang et al. [48], Castillo-Chavez et al. [17], Gumel [39]). 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 (Dushoff et al. [30]). Secondly, reducing R_0 to less than unity is, although necessary, but not sufficient to eradicate the disease as is the case in the stated Principle 1.1.4 above.

1.2 Purpose of the thesis

The general setting of this thesis is constituted by n-dimensional dynamical systems that undergo the backward bifurcation phenomenon. It is known that the theorem by Castillo-Chavez and Song [16] is one of the leading and widely used mathematical tools to determine the existence of a backward bifurcation in continuous epidemiological models. The problem statement of this thesis is broadly as follows:

- What is the analogue of the theorem for discrete dynamical systems?

- How to construct reliable numerical methods that capture the backward bifurcation phenomenon for continuous epidemiological models?

The ultimate goal is to investigate the dynamics of a malaria model and a simple model for a disease without permanent immunity, that exhibit a backward bifurcation. The specific purpose of the thesis is as follows:

(i) To get a better understanding of the theorem in Castillo-Chavez and Song [16] regarding the conditions under which a backward bifurcation occurs.

(ii) To highlight the advantage of reducing the dimension of the dynamical system in Castillo-Chavez and Song [16] compared to the power series approximation in the implementation of the Centre Manifold Theory (CMT) in Wiggins [85].

(iii) To state and prove, for discrete dynamical systems, a discrete analogue of the main theorem in Castillo-Chavez and Song [16].

(iv) To prove the conjecture formulated in Chitnis et al. [23] for a malaria model by determining the critical threshold of the disease-induced death rate above which the model undergoes a backward bifurcation.

(v) To construct Nonstandard Finite Difference Schemes (NSFD) which are dynamically consistent with respect to the backward bifurcation property of both the malaria and SIS models.

1.3 Literature review

As mentioned earlier, the general setting of this thesis is the qualitative analysis of n-dimensional dynamical systems. Classically, when the equilibrium point under consideration is hyperbolic, the local analysis of the system is readily obtained by the linearisation technique through the Hartman-Grobman theorem (Stuart and Humphries [77]), which is widely used in the literature (see Wiggins [85] for general systems, Brauer and Castillo-Chavez [13]).

When the equilibrium point is nonhyperbolic, the linearisation technique does not apply. This situation is well known. For instance, in epidemiology, when a forward bifurcation occurs at the value $R_0 = 1$ of the basic reproduction number, the global asymptotic stability of the disease-free equilibrium cannot be obtained by linearisation. One has to use deeper tools such as the LaSalle Invariance Principle and Lyapunov functions (see LaSalle [56], [55]). The context of this thesis is more difficult because we are dealing with the situation where the system undergoes a backward bifurcation. The investigation of the backward bifurcation phenomenon is a challenging assignment which is not new in epidemiology (see for instance Castillo-Chavez et al. [17] and Hadeler and Van den Driessche [41]). It is worth mentioning the paper by Gumel [39] in which some of the causes of the backward bifurcation phenomenon are outlined. These include:

(a) Imperfect vaccine.

(b) Exogenous re-infection (e.g. Tuberculosis).

(c) Vaccine-derived immunity waning at a slower rate than natural immunity.

(d) Disease-induced mortality in vector borne diseases.

(e) Differential susceptibility in risk-structured models.

In this thesis, we determine the local dynamics of systems by using the Centre Manifold Theory in [85]. The theory amounts to considering the system on a manifold of reduced dimension. We first follow the approach proposed by Carr [15] and presented in Wiggins [85] for the construction of the Centre Manifold. In this approach, the dynamics of the system of the reduced manifold is eventually obtained by power series approximations. The latter tool is one of the reasons why this approach is not easily applicable to complex systems. As a result, this approach is not popular in epidemiology. Among the few authors that have used it, we can mention Kribs-Zaleta and Velasco-Hernández [52], Zhonghua and Yaohong [88] and Cui et al. [25].

The second approach is due to Castillo-Chavez and Song [16]. It is based on a simple structure of the Centre Manifold that makes it possible to reduce the original system to a scalar equation. It is therefore not surprising that this approach is extensively used in epidemiology (see Garba et al. [37], Buonomo and Vargas-De-León [14] and Feng et al. [35]). For a better understanding of the main theorem in Castillo-Chavez and Song [16], we have re-centred its context and provided more details. This enabled us to state and prove a new result on the discrete analogue of the theorem for discrete dynamical systems. Our effort to better understand the above-outlined approaches is taken one step further by considering a malaria model due to Chitnis et al. [23] and SIS model by Villavicencio-Pulido et al. [79]. Despite the complex nature of the models under consideration, we managed to analytically construct the centre manifold, instead of resorting to computer software codes which are abundantly used in the literature. We theoretically proved and demonstrated by means of numerical simulations the conjecture in Chitnis et al. [23] which reads as follows: *if the disease-induced death rate is large enough, the malaria model undergoes a backward bifurcation at* $R_0 = 1$.

Whether the considered continuous models are complex or simple, in general, they cannot be completely solved by analytic techniques. Consequently, numerical methods are of fundamental importance in gaining more useful insights from the solution of the differential equation.

In this thesis, we use the nonstandard finite difference (NSFD) method. This method was founded almost three decades ago by Mickens and has shown great potential in replicating the dynamics of the solution of a wide range of continuous models, ranging from differential equations (Anguelov and Lubuma [3], Anguelov and Lubuma [4], Anguelov et al. [5], Anguelov et al. [8], Anguelov et al. [7], Dimitrov and Kojouharov [27], Dimitrov and Kojouharov [26], Wood et al. [87], Alexander et al. [1]); integral equations (Roeger [72], Lubuma and Terefe [57], [70]); delay differential equations (Ding et al. [28], Garba et al.

[36]); (advection) reaction diffusion equations (Kama [50], Anguelov et al. [6]); cross-diffusion equations (Chapwanya et al. [20], Chapwanya et al. [21]); models satisfying conservation laws (Mickens and Washington [65]) and pharmacokinetics models (Egbelowo [32], Egbelowo et al. [33]).

NSFD schemes have not been sufficiently developed for epidemiological models with a backward bifurcation. In fact, in the few available works, the focus is on illustrations using numerical simulations (see for instance Anguelov et al. [7] and Garba et al. [37]). In this thesis, we construct NSFD schemes for the two epidemiological models. We perform their full analyses, including computational aspects and numerical simulations, which confirm that they preserve the backward bifurcation property.

1.4 Outline of the thesis

In Chapter 2, we give preliminaries on continuous and discrete dynamical systems. Emphasis is placed on elementary bifurcation theory in the setting where the involved Jacobian matrix of the dynamical system has a simple zero eigenvalue.

Chapter 3 deals with the Centre Manifold Theory (CMT). The focus is on three aspects, namely the existence, the approximation, and the computation of the Centre Manifold. Regarding the latter aspect, two approaches are discussed. The first approach involves the use of power series expansions in the reduction process. The second approach, due to Castillo-Chavez and Song [16], is more practical and is presented in the form of the necessary and sufficient conditions for the existence of a backward bifurcation. An analogue of this result for discrete dynamical systems is stated and proved in this Chapter.

The applications of the theorems discussed in Chapter 3, to continuous epidemiological models, are given in Chapter 4. More precisely, for a complex malaria model and SIS model with vaccination, we establish results which show that at the value 1 of the basic reproduc-

tion number R_0, a backward bifurcation occurs.

Chapter 5 is devoted to the construction and analysis of NSFD schemes which replicate the dynamics, including the backward bifurcation property, of the two epidemic models.

In Chapter 6, we conclude our work by giving a summary of our results, discuss how they fit into the literature, make some remarks, and give suggestions for future research.

CHAPTER 2. PRELIMINARIES ON DYNAMICAL SYSTEMS

In this Chapter, we recall a number of concepts and results on dynamical systems, which will be useful in this thesis. Section 2.1 deals with continuous dynamical systems, while Section 2.2 is devoted to discrete dynamical systems. Elementary bifurcation theory is presented in Section 2.3. Our preferred standard reference is Stuart and Humphries [77]. Other references will be cited when necessary.

2.1 Continuous dynamical systems

Consider the autonomous system of ordinary differential equations (ODEs)

$$\frac{dx}{dt} := \dot{x} = f(x) \tag{2.1.1}$$

with initial condition

$$x(0) = x_0 \in \mathbb{R}^n, \tag{2.1.2}$$

where $f : U \subseteq \mathbb{R}^n \to \mathbb{R}^n$ is sufficiently smooth on an open set U.

By the Fundamental Theorem of Calculus, the initial value problem (2.1.1)-(2.1.2) is equivalent to the integral equation

$$x(t) = x(0) + \int_0^t f(x(r))dr. \tag{2.1.3}$$

Definition 2.1.1. *Eq. (2.1.1) defines a dynamical system on a set $U \subseteq \mathbb{R}^n$ if, for every $x_0 \in U$, there exists a unique solution of Eq. (2.1.1) which is defined for all $t \in [0, \infty)$ and satisfies $x(t) \in U$.*

Definition 2.1.2. *The evolution semigroup operator for a dynamical system on a set U is the map*

$$\varphi(t) : U \to U, \;\; t \geq 0, \tag{2.1.4}$$

defined by

$$\varphi(t)x_0 = x(t), \tag{2.1.5}$$

where $x(t)$ is the unique global solution of the initial value problem (2.1.1)-(2.1.2).

Remark 2.1.3. *The operator φ satisfies the following semigroup properties:*

(i) $\varphi(0)x_0 = x_0$, $x_0 \in U$.

(ii) $\varphi(t+s)x_0 = \varphi(t)\varphi(s)x_0 = \varphi(s)\varphi(t)x_0$.

Item (i) is obvious from the definition in Eq. (2.1.5). Item (ii) follows from (2.1.3). Indeed,

$$\begin{aligned}
\varphi(t+s)x_0 &= x_0 + \int_0^{t+s} f(x(r))dr \\
&= x_0 + \int_0^{s} f(x(r))dr + \int_s^{t+s} f(x(r))dr \\
&= \varphi(s)x_0 + \int_0^{t} f(x(u+s))du \\
&= \varphi(t)\varphi(s)x_0.
\end{aligned}$$

The map $\varphi(t)x_0$ represents the state of the system, after some time t, which started at a state $x_0 = \varphi(0)x_0$ when $t = 0$.

Definition 2.1.4. *A point $x^* \in \mathbb{R}^n$ such that $f(x^*) = 0$ is called an equilibrium point of Eq. (2.1.1).*

Definition 2.1.5. *Let x^* be an equilibrium point of a dynamical system on U defined by Eq. (2.1.1). Then x^* is called:*

(i) *stable if for any* $\epsilon > 0$, *there exists a* $\delta > 0$ *such that* $|x_0 - x^*| < \delta$ *implies* $|x(t) - x^*| < \epsilon$ *for* $t \geq 0$.

(ii) *asymptotically stable if it is stable and there exists* $\eta > 0$ *such that*

$$\lim_{t\to\infty} x(t) = x^* \ \forall \ x_0 \in B_\eta(x^*)$$

where $B_\eta(x^*) \equiv \{x_0 \in \mathbb{R}^n : |x_0 - x^*| < \eta\}$.

(iii) *globally asymptotically stable (GAS), if it is stable and*

$$\lim_{t\to\infty} x(t) = x^* \ \forall \ x_0 \in U.$$

(iv) *unstable whenever it is not stable.*

In order to determine the stability of an equilibrium point x^*, we consider a solution $x(t)$ of Eq. (2.1.1) and make the change of dependent variable $x(t) = x^* + y(t)$. Differentiating with respect to time, then substituting into Eq. (2.1.1) and performing Taylor's expansion of the right hand side about x^*, we obtain

$$\dot{x} = \dot{y}(t) = D_x f(x^*) y(t) + \mathcal{O}(|y|^2)$$

where $D_x f(x^*) = J$ is the Jacobian matrix of f at x^*. By retaining the linear part, we thus obtain

$$\dot{y} = Jy. \tag{2.1.6}$$

Eq. (2.1.6) is known as the linearised system of Eq. (2.1.1) about the equilibrium point x^*. The initial value problem associated with Eq. (2.1.6) has a unique solution

$$y(t) = e^{Jt} y_0, \ \ y_0 = x_0 - x^*, \tag{2.1.7}$$

where $t \rightsquigarrow e^{Jt}$ is the evolution semigroup operator of system (2.1.6). The following definition can be found in Wiggins [85].

Definition 2.1.6. *The equilibrium point x^* of Eq. (2.1.1) is said to be hyperbolic if none of the eigenvalues of J have zero real parts, and nonhyperbolic if at least one of the eigenvalues has a zero real part.*

We now have the necessary background to address the following question: can the qualitative behaviour of solutions of Eq. (2.1.1) near x^* be obtained from the qualitative behaviour of the solution near the origin of Eq. (2.1.6) as given in Eq. (2.1.7)?
The answer to this question is addressed via the Hartman-Grobman Theorem which requires that the diagram in Fig. 2.1 commutes, where h is a homeomorphism.

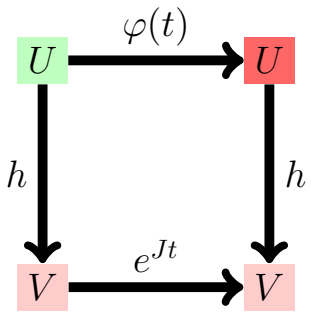


Figure 2.1: Hartman-Grobman Theorem for flows.

The Hartman-Grobman Theorem can be found in most standard textbooks on dynamical systems, (see for instance Chicone [22], Robinson [69], Hale and Koçak [42], and Simon [75]). Theorem 2.1.7 below can be found in Crawford [24].

Theorem 2.1.7. *Assume that Eq. (2.1.1) defines a dynamical system on $\mathbb{R}^n$ and x^* is a hyperbolic equilibrium point. There exists a homeomorphism $h : U \to V$ between a neighbourhood $U \subset \mathbb{R}^n$ of x^* and a neighbourhood $V \subset \mathbb{R}^n$ of the origin, such that $h(\varphi(t)x_0) = e^{\wedge t}h(x_0)$, where $\wedge$ is a matrix similar to the matrix J.*

Remark 2.1.8. *Theorem 2.1.7 implies that the evolution semigroup operators $t \rightsquigarrow e^{Jt}$ and $t \rightsquigarrow \varphi(t)$ are topologically equivalent (see Crawford [24]). Consequently, any topological*

property of the linearised Eq. (2.1.7) is transferred to System (2.1.1). In particular the local stability property of the equilibrium point x^* is similar to that of the origin for System (2.1.7). In view of Eq. (2.1.7), this in turn is characterised by the sign of the real parts of the eigenvalues λ of J i.e. the origin is locally asymptotically stable if and only if $Re(\lambda) < 0$ for all λ.

2.2 Discrete dynamical systems

Discrete dynamical systems refer to processes which evolve with time in discrete time steps. Such systems are modelled using difference equations or sequences. Instead of using the term flow which is used in continuous models, we will use the term maps to refer to solutions of difference equations. Consider

$$x^{k+1} = g(x^k), \;\; k = 0, 1, 2, \cdots, \tag{2.2.1}$$

with initial condition

$$x^0 \in \mathbb{R}^n \tag{2.2.2}$$

where $g : U \subseteq \mathbb{R}^n \to \mathbb{R}^n$ is sufficiently smooth on an open set U.

Definition 2.2.1. *Eq. (2.2.1) defines a discrete dynamical system on a set $U \subseteq \mathbb{R}^n$ if, for every $x^0 \in U$, the sequence generated by Eq. (2.2.1) is well defined and remains in U for all integers $k \geq 0$.*

Definition 2.2.2. *The evolution semigroup operator for Eq. (2.2.1) on the set U is defined by the map*

$$\varphi^k : U \to U \tag{2.2.3}$$

with

$$\varphi^{k+1}(x^0) = x^{k+1} = g(x^k) = g^k(x^0). \tag{2.2.4}$$

Remark 2.2.3. *The sequence φ^k satisfies the following properties for all $x^0 \in U$*

(i) $\varphi^0 x^0 = x^0$.

(ii) $\varphi^{k+s} x^0 = \varphi^k \varphi^s x^0$.

Definition 2.2.4. *A point $x^* \in U$ such that $x^* = g(x^*)$ is called a fixed-point of Eq. (2.2.1).*

Definition 2.2.5. *Let x^* be a fixed-point of a dynamical system on U defined by Eq. (2.2.1). The fixed-point x^* is called:*

(i) stable if for any $\epsilon > 0$,there exists a $\delta > 0$ such that $|x^0 - x^*| < \delta$ implies $|x^k - x^*| < \epsilon$ for all $k \in \mathbb{N}$.

(ii) asymptotically stable if it is stable and there exists $\eta > 0$ such that

$$\lim_{k\to\infty} x^k = x^* \ \forall \ x^0 \in B_\eta(x^*)$$

(iii) globally asymptotically stable if it is stable and

$$\lim_{k\to\infty} x^k = x^* \ \forall \ x^0 \in U.$$

(iv) unstable if it is not stable.

If we expand the right hand side of Eq. (2.2.1) in a Taylor series about the fixed-point x^*, and drop higher order terms we obtain

$$x^{k+1} = g(x^*) + A(x^k - x^*) \tag{2.2.5}$$

where $D_x g(x^*) = A$ is the Jacobian matrix of g evaluated at x^*. By letting $x^k - x^* = y^k$ and using $g(x^*) = x^*$, we obtain the linearised equation

$$y^{k+1} = Ay^k. \tag{2.2.6}$$

where $k \rightsquigarrow A^k$ is the evolution semigroup operator.

Definition 2.2.6. *The fixed-point x^* is said to be hyperbolic if none of the eigenvalues of the matrix A lie on the unit circle, and nonhyperbolic if at least one of the eigenvalues lies on the unit circle.*

The discrete analogue of the Hartman-Grobman theorem and the associated commutative diagram read as follows (Fig. 2.2):

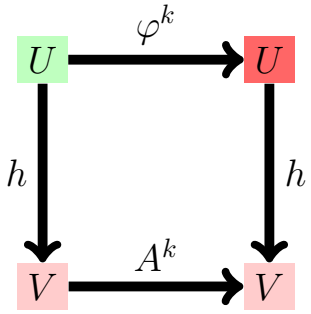


Figure 2.2: Hartman-Grobman Theorem for maps.

Theorem 2.2.7. *Let $x^* \in \mathbb{R}^n$ be a hyperbolic fixed-point of the dynamical system (2.2.1). There exists a homeomorphism $h : U \to V$ between a neighbourhood $U \subset \mathbb{R}^n$ of x^* and a neighbourhood $V \subset \mathbb{R}^n$ of the origin such that*

$$h\left(\varphi^k x^0\right) = A^k h(x^0). \tag{2.2.7}$$

2.3 Elementary bifurcation

Consider a dynamical system

$$\dot{z} = H\left(z, \phi\right), \quad z \in \mathbb{R}^n, \quad \phi \in \mathbb{R}, \tag{2.3.1}$$

depending on a parameter ϕ, where H is sufficiently smooth.

Definition 2.3.1. *A point (z^*, ϕ^*) such that*

$$H(z^*, \phi^*) = 0 \tag{2.3.2}$$

is called an equilibrium point of system (2.3.1).

In many applications of dynamical systems it happens that the asymptotic stability of an equilibrium point changes due to bifurcation, but the equilibrium solution remains (see for instance Crawford [24]). To refer to this situation, we give the following definition.

Definition 2.3.2. *A point $z^* \in \mathbb{R}^n$ is called a permanent equilibrium point if (z^*, ϕ) is an equilibrium point for all ϕ.*

Remark 2.3.3. *Typically in epidemiology, the permanent equilibrium point is the disease-free equilibrium.*

The local bifurcation theory seeks to address the following question: *What is most likely to happen in the phase space $\phi - z$ near an equilibrium point (z^*, ϕ^*) after a small perturbation of ϕ?*

In this thesis, we are interested in a specific value ϕ_c of the ϕ coordinate of the equilibrium instead of a general ϕ^*. Let the eigenvalues λ of the Jacobian matrix $D_z H(z^*, \phi)$ of H evaluated at (z^*, ϕ) depend on the parameter ϕ: $\lambda = \lambda(\phi)$. As ϕ is varies, it may happen that the eigenvalue crosses the imaginary axis. The point $\phi = \phi_c$ at which the real part of the eigenvalue is equal to zero (i.e. $Re(\lambda(\phi_c)) = 0$) is called the critical value of the parameter. Note that the equilibrium point (z^*, ϕ_c) is nonhyperbolic, z^* being a permanent equilibrium point.

In order to address the question stated above about the local bifurcation theory, we consider the following two restrictions of the system (2.3.1):

$$\dot{z} = H(z, \phi_c) \quad on \quad B_\delta(z^*) \tag{2.3.3}$$

and

$$\dot{z} = H(z, \phi) \quad on \quad B_\delta(z^*) \times (\phi_c - \epsilon, \phi_c + \epsilon) =: U_{\delta,\epsilon}(z^*, \phi_c). \tag{2.3.4}$$

Definition 2.3.4 (Wiggins [85]). *An equilibrium point (z^*, ϕ) is said to undergo a bifurcation at $\phi = \phi_c$ if there exist $\delta > 0$ and $\epsilon > 0$ such that the qualitative features of the equilibrium point (z^*, ϕ) of system (2.3.4) are not the same as those of the equilibrium point (z^*, ϕ_c) of system (2.3.3). Alternatively, the number $\phi = \phi_c$ is said to be a bifurcation value for the system (2.3.1) provided that there exists an equilibrium point (z^*, ϕ_c) of the system that satisfies the properties in the previous statement.*

The general class of bifurcation points of interest in this thesis is described in the following definition:

Definition 2.3.5. *A bifurcation occurring at the equilibrium point (z^*, ϕ_c) is said to be a transcritical bifurcation if the following conditions hold:*

(i) at least two curves $z = z(\phi)$ of equilibrium points exist in the $\phi - z$ phase space for both $\phi < \phi_c$ and $\phi_c < \phi$.

(ii) the curves of the equilibrium point branch at $(\phi = \phi_c)$ or intersect at the point (z^*, ϕ_c).

(iii) the stability of an equilibrium point along a given curve changes on passing through (z^*, ϕ_c).

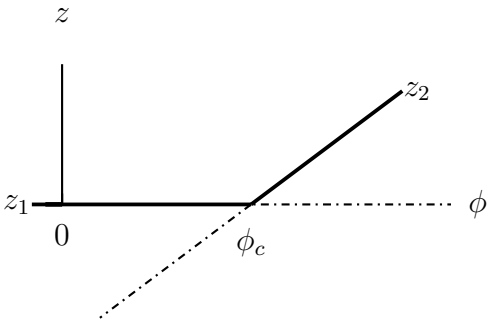


Figure 2.3: Transcritical bifurcation at $\phi = \phi_c$.

Remark 2.3.6. *Fig. 2.3 illustrates Definition 2.3.5. It gives the bifurcation diagram for the scalar equation* $\dot{z} = z(-z - \phi_c + \phi)$ *that has permanent equilibrium point* $z^* = 0$. *Here and after, the dotted lines represent unstable equilibrium points, whereas solid lines represent locally asymptotically stable equilibria.*

In the context of epidemiology, which is the main setting of this thesis, Definition 2.3.5 is made more specific by considering a permanent equilibrium point z^* and nonnegative solutions of the underlying system as follows:

Definition 2.3.7. *Let* $z^* \geq 0$ *be a permanent equilibrium point of the system (2.3.1).*

(i) The system (2.3.1) is said to undergo a forward bifurcation at $\phi = \phi_c$ *(or* $\phi = \phi_c$ *is a forward bifurcation point) provided that the permanent equilibrium point* z^**:*

(a) is locally asymptotically stable for $\phi < \phi_c$ *and unstable for* $\phi > \phi_c$.

(b) for $\phi > \phi_c$, *there exists a curve* $z = z(\phi) > 0$ *of locally asymptotically stable equilibrium points.*

(ii) The system (2.3.1) is said to undergo a backward bifurcation provided that condition i(a) above holds true while for $\phi < \phi_c$, *there exists a curve* $z = z(\phi) > 0$ *of unstable equilibrium points.*

Remark 2.3.8. *Regarding part (ii) of Definition 2.3.7, we will shortly show that both small unstable equilibrium* $z = z(\phi) > 0$ *and a large LAS equilibrium point* $z = z(\phi) > 0$ *can exist. This explains why the definition of backward bifurcation phenomenon in epidemiology makes explicit mention of two positive endemic equilibria (see Castillo-Chavez and Song [16] for instance).*

There are some similarities between the bifurcation theory of continuous and discrete dynamical systems. In what follows we briefly point out some important aspects of the

discrete case. Consider a discrete dynamical system

$$z_{n+1} = F(z_n, \phi), \quad z_n \in \mathbb{R}^n, \quad \phi \in \mathbb{R}, \tag{2.3.5}$$

depending on a parameter ϕ, and F is sufficiently smooth.

Definition 2.3.9. *A point (z^*, ϕ^*) such that*

$$F(z^*, \phi^*) - z^* = 0 \tag{2.3.6}$$

is called a fixed-point of system (2.3.5).

Remark 2.3.10. *According to Guckenheimer and Holmes [38], page 157, "The bifurcation theory for fixed-points of the system (2.3.5) with eigenvalue 1 is completely analogous to the bifurcation theory for equilibria of the system (2.3.1) with eigenvalue 0". Indeed if we let*

$$H(z_n, \phi) = F(z_n, \phi) - z_n, \tag{2.3.7}$$

then

$$H(z^*, \phi) = 0 \text{ and } D_z H(z^*, \phi) = D_z F(z^*, \phi) - I_n. \tag{2.3.8}$$

It is clear from Eq. (2.3.8) that $D_z H(z^*, \phi)$ will have a zero eigenvalue if $D_z F(z^*, \phi)$ has eigenvalue equal to one.

2.4 Simple zero eigenvalue and normal forms

Because of Remark 2.3.10, we shall deal with a case of a simple zero eigenvalue only. Assume that $z^* = 0$ is a permanent equilibrium point of the system (2.3.1) and $\phi = 0$ is a bifurcation point. It can happen that the study of the local behaviour of System (2.3.1) about $(0, 0)$ is reduced to the study of the following one dimensional system

$$\dot{\xi} = \tilde{H}(\xi, \phi), \quad \phi \in \mathbb{R}, \quad \xi \in \mathbb{R}. \tag{2.4.1}$$

This is the case when $\lambda = 0$ is a simple eigenvalue of $D_z H(0,0)$ and all the other eigenvalues have real parts that are less than zero; as we will see in the next Chapter on the Centre Manifold Theory. Without loss of generality, we assume that Eq. (2.4.1) exists for System (2.3.1) and $(0,0)$ is an equilibrium point of Eq. (2.4.1). We expand the right hand side of Eq. (2.4.1) in Taylor series about the point $(\xi,\phi) = (0,0)$ to obtain

$$\tilde{H}(\xi,\phi) = \sum_{i,j=0}^{\infty} d_{i,j}\xi^i\phi^j \quad \text{where} \quad d_{i,j} = \frac{1}{i!j!}\frac{\partial^{i+j}\tilde{H}}{\partial\xi^i\partial\phi^j}\Big|_{(\xi,\phi)=(0,0)}. \tag{2.4.2}$$

Several normal forms may be obtained from Eq. (2.4.1-2.4.2) but we restrict ourselves to what is relevant to our work. If

$$\tilde{H}_\xi(0,0) = 0, \;\; \tilde{H}_\phi(0,0) = 0, \;\; \tilde{H}_{\phi\phi}(0,0) = 0, \;\; \tilde{H}_{\xi\xi}(0,0)\tilde{H}_{\xi\phi}(0,0) \neq 0 \tag{2.4.3}$$

and we drop terms of order higher than two, Eq. (2.4.1) becomes

$$\dot{\xi} = \tilde{H}_{\phi\xi}(0,0)\xi\phi + \frac{1}{2}\tilde{H}_{\xi\xi}(0,0)\xi^2 = b\xi\phi + a\xi^2. \tag{2.4.4}$$

Eq. (2.4.4) is known as the normal form of a transcritical bifurcation.

Remark 2.4.1. *It will becaome clear from the Centre Manifold Theory in the next Chapter why the full Eq. (2.4.1) and the truncated Eq. (2.4.4) have the same bifurcation property at the equilibrium point* $(0,0)$.

We will give specific examples which lead to forward and backward bifurcations. In Fig. 2.4 we illustrate a backward bifurcation by using the following equation.

$$\dot{\xi} = (R_0 - 1)\xi + 2.09\xi^2 \quad \text{where} \quad \phi = R_0 - 1. \tag{2.4.5}$$

Remark 2.4.2. *The choice of* $\phi = R_0 - 1$ *is motivated by the nature of bifurcation parameters found in dynamical systems arising in epidemiology. The equivalence of the bifurcation at* $\phi = 0$ *is the bifurcation value* $R_0 = 1$ *in this case.*

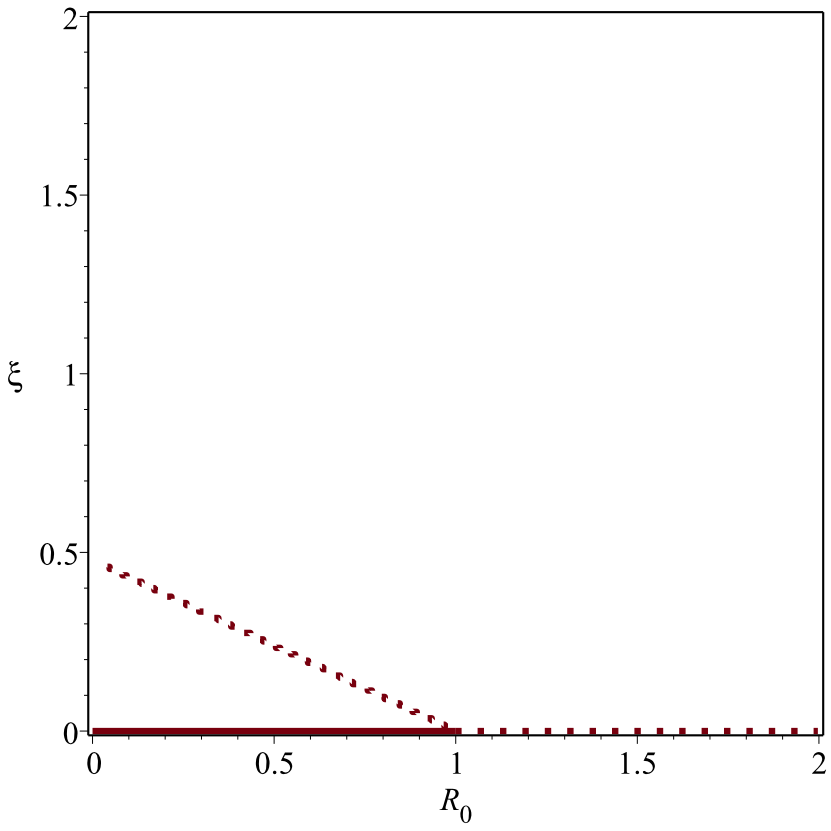


Figure 2.4: Eq. (2.4.5) undergoes a backward bifurcation at $R_0 = 1$.

By changing the sign of the coefficient of ξ^2 in Eq. (2.4.5), which is equivalent to changing the sign of a in Eq. (2.4.4), we obtain Eq. (2.4.6). This is a forward bifurcation as illustrated in Fig. 2.5.

$$\dot{\xi} = (R_0 - 1)\xi - 2.09\xi^2 \quad \text{where} \quad \phi = R_0 - 1. \tag{2.4.6}$$

Remark 2.4.3. *If we drop terms of order higher than five in Eq. (2.4.2), we may obtain*

$$\dot{\xi} = b\xi\phi + a\xi^2 + a_1\xi^3 + a_2\xi^4 + a_3\xi^5. \tag{2.4.7}$$

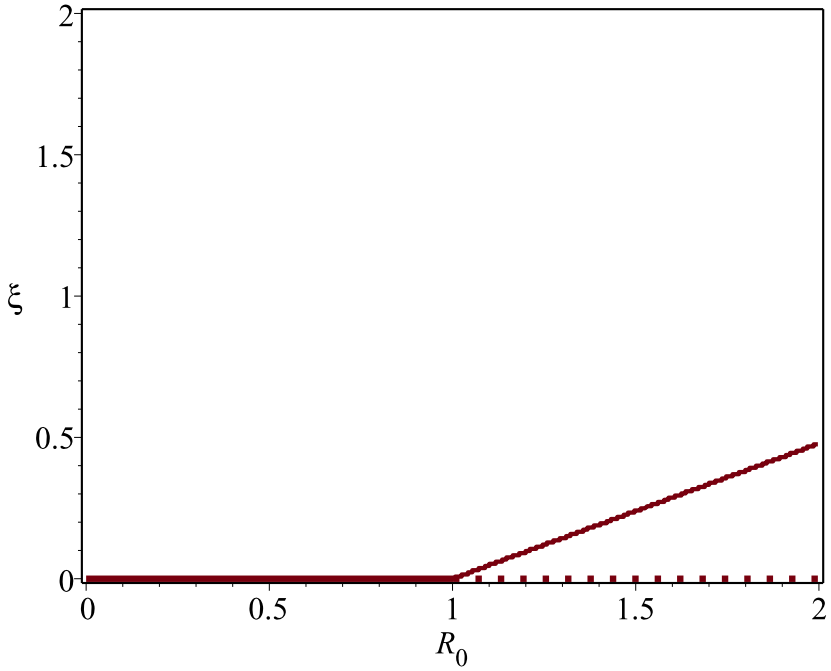


Figure 2.5: Eq. (2.4.6) undergoes a forward bifurcation at $R_0 = 1$.

From Eq. (2.4.7), some kind of a complicated bifurcation called hysteresis (Kuznetsov [53]) bifurcation may arise depending on the sign of the coefficients. Take for instance the equation

$$\dot{\xi} = (R_0 - 1)\xi + 2.09\xi^2 - \xi^3 - 0.001\xi^4 - \xi^5 \tag{2.4.8}$$

which is a special case of Eq. (2.4.7) and a direct extension of Eq. (2.4.5). The associated bifurcation diagram is given in Fig. 2.6. It displays the backward bifurcation phenomenon at $R_0 = 1$ in the more precise form, that for $R_0 < 1$ but close to one (i.e. for $\phi \in (\phi_c - \epsilon, \phi_c)$), there exist both a small unstable positive equilibrium point and a large LAS positive equilibrium which coexists with the LAS permanent equilibrium zero.

Similarly, if we extend Eq. (2.4.6) we may obtain

$$\dot{\xi} = (R_0 - 1)\xi - 2.09\xi^2 - \xi^3 - 0.001\xi^4 - \xi^5 \tag{2.4.9}$$

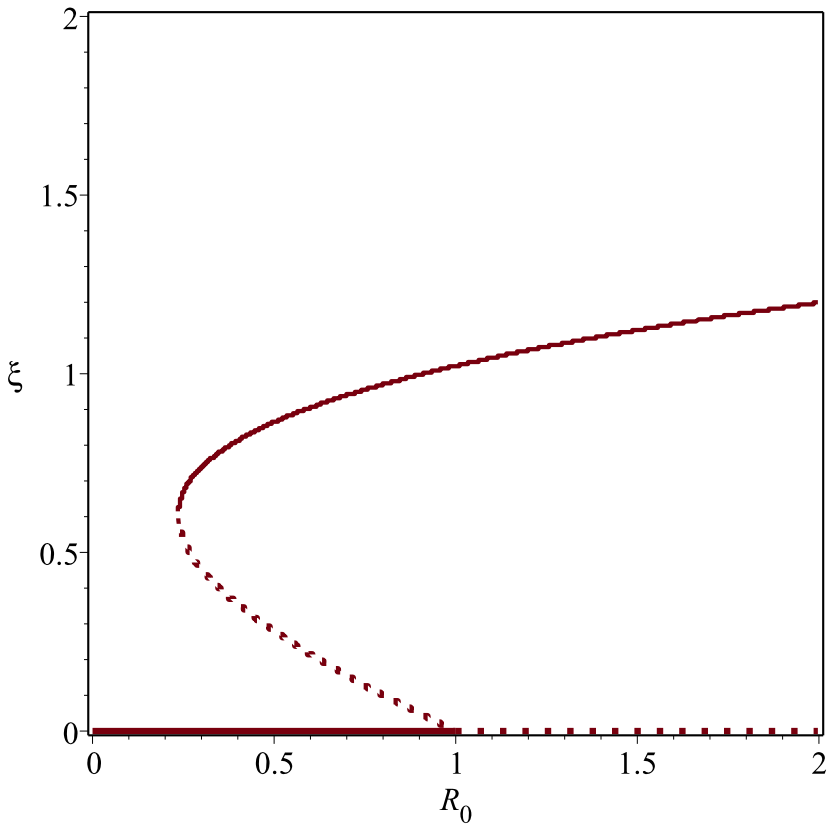


Figure 2.6: Eq. (2.4.8) undergoes a backward bifurcation at $R_0 = 1$.

which is a special case of Eq. (2.4.7). A forward bifurcation occurs at $R_0 = 1$ *as illustrated in Fig. 2.7. The dynamics of Eq. (2.4.9) and Eq. (2.4.6) around the bifurcation point* $R_0 = 1$ *do not differ much, which is consistent with Remark 2.4.1. Their phase portraits are topologically the same to be precise. We further explore a scenario where* R_0 *becomes much larger than* 1. *The local dynamics in the neighbourhood of* $R_0 = 1$ *do not change as we can see in Figs 2.8 and 2.9. However, if we consider Eq. 2.4.10 below*

$$\dot{\xi} = (R_0 - 1)\xi - 2.09\xi^2 - \xi^3 - 0.001\xi^4 + \xi^5, \tag{2.4.10}$$

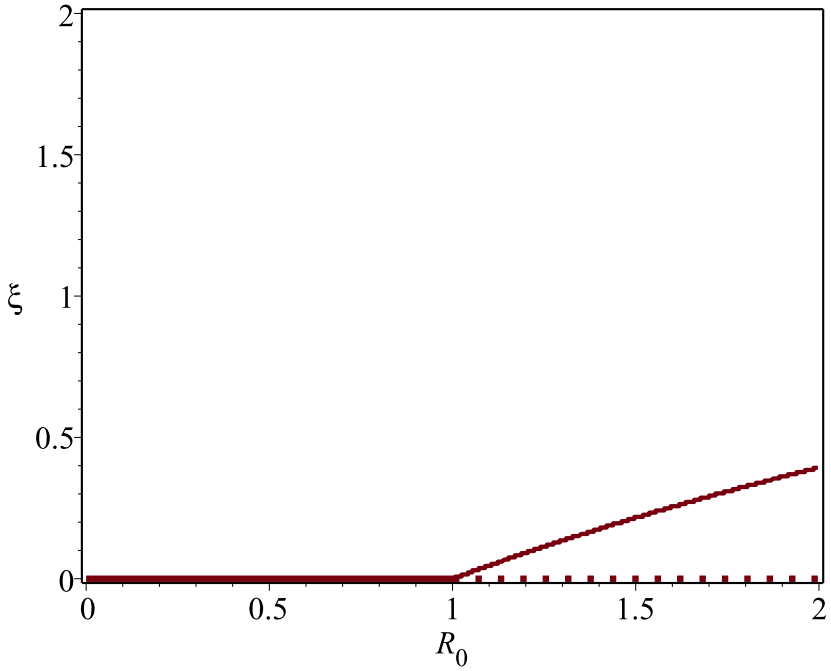


Figure 2.7: Eq. (2.4.9) undergoes a forward bifurcation at $R_0 = 1$.

it becomes clear that the dynamics in the neighbourhood of $R_0 = 1$ *should not be used in global asymptotic stability analysis as it is shown in Fig. 2.10.*

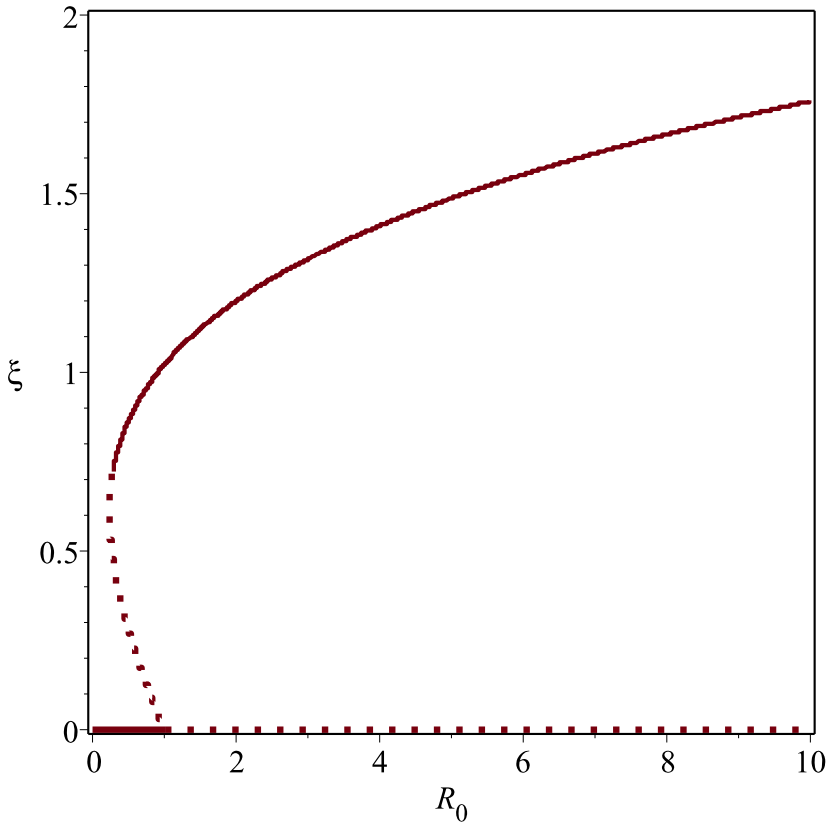


Figure 2.8: Eq. (2.4.8) undergoes a backward bifurcation at $R_0 = 1$.

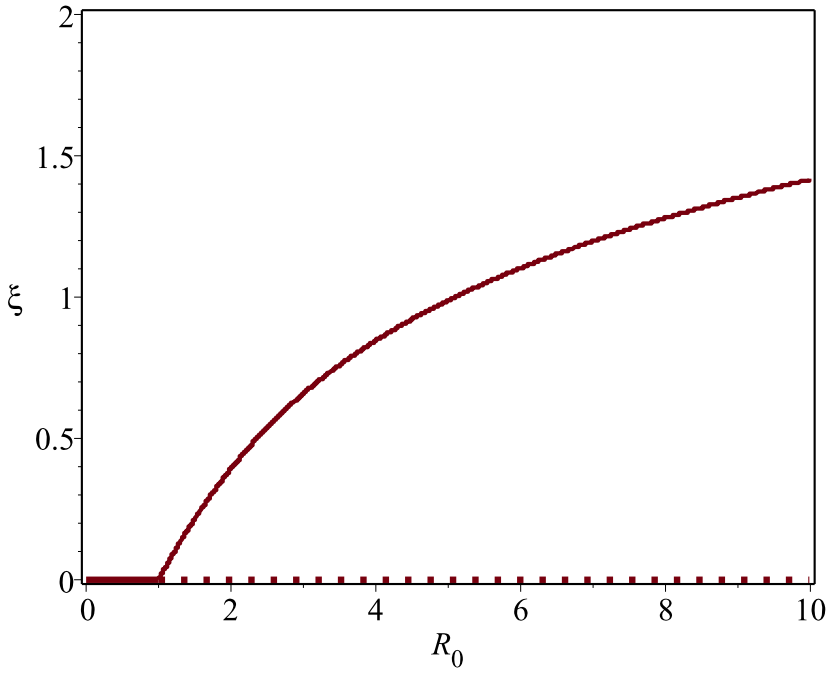


Figure 2.9: Eq. (2.4.9) undergoes a forward bifurcation at $R_0 = 1$.

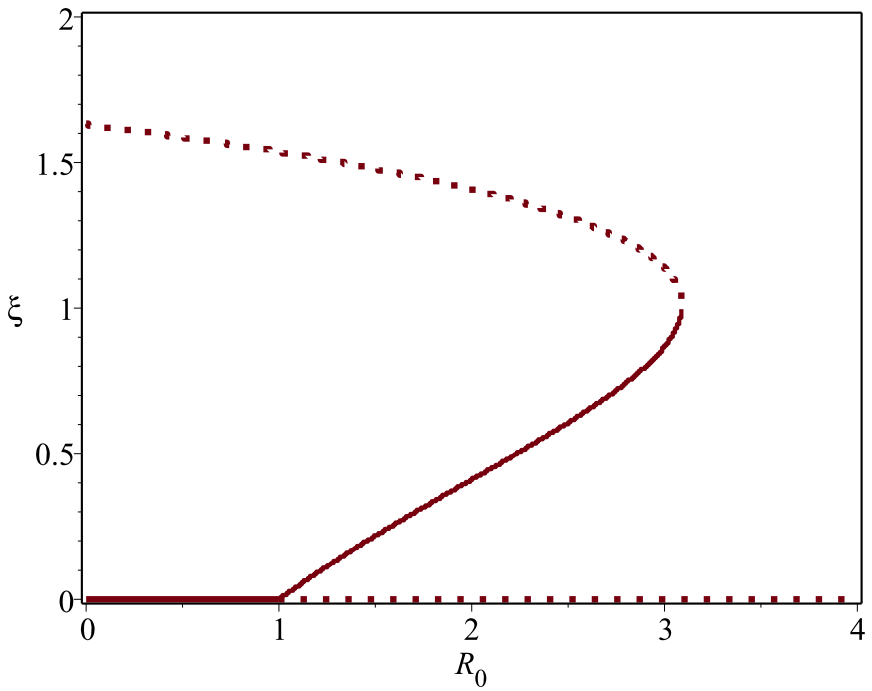


Figure 2.10: Eq. (2.4.10) undergoes a forward bifurcation at $R_0 = 1$. However, the branch of the positive equilibrium when $R_0 > 1$ is not unique. Moreover, there exists a positive branch of an unstable equilibrium when $R_0 < 1$.

CHAPTER 3. THE CENTRE MANIFOLD THEORY

3.1 Introduction

Linearisation about equilibrium points is a powerful approach, which is widely used for the stability analysis of nonlinear dynamical systems. Whenever it works, this approach is relatively simple to implement because the conclusion is based on the sign of the real parts (for continuous systems) and the moduli (for discrete systems) of the eigenvalues of the associated Jacobian matrices. This technique works only when the system on the targeted equilibrium point is hyperbolic as captured in Hartman-Grobman Theorems 2.1.7 and 2.2.7. The centre manifold theory is applied to nonlinear systems which are nonhyperbolic. This is precisely the setting of this Chapter in which various reductions of the systems are investigated. Most of the theory in this Chapter is taken from Wiggins [85] and Castillo-Chavez and Song [16].

3.2 The Setting

The dynamical system (2.3.1) is now considered with $\phi \in \mathbb{R}^p$, $p \geq 1$, and is for convenience reproduced here:

$$\dot{z} = H(z, \phi), \quad z \in \mathbb{R}^n, \quad \phi \in \mathbb{R}^p. \tag{3.2.1}$$

We assume that $z^* = 0$ is a permanent equilibrium point and $(0, 0)$ is a bifurcation point such that the Jacobian matrix $D_z H(0, 0)$ has c eigenvalues with zero real parts, and all the other eigenvalues have negative real parts; thus $c + s = n$. There exists then an invertible

$n \times n$ matrix such that

$$Q^{-1}\left(D_z H(0,0)\right) Q = \begin{pmatrix} A & 0 \\ 0 & B \end{pmatrix} =: C, \tag{3.2.2}$$

where all the eigenvalues of the $c \times c$ matrix A have zero real parts, and the $s \times s$ matrix B has eigenvalues with negative real parts. If we use the change of dependent variables

$$z = Q \begin{pmatrix} x \\ y \end{pmatrix}, \quad \text{where} \quad x \in \mathbb{R}^c \ \text{ and } \ y \in \mathbb{R}^s, \tag{3.2.3}$$

then Eq. (3.2.1) is transformed into

$$\begin{cases} \dot{x} = Ax + f(x, y, \phi), \\ \dot{y} = By + g(x, y, \phi), \\ \dot{\phi} = 0, \end{cases} \tag{3.2.4}$$

where

$$\begin{aligned} f(0,0,0) = 0, \quad Df(0,0,0) = 0, \\ g(0,0,0) = 0, \quad Dg(0,0,0) = 0, \end{aligned} \tag{3.2.5}$$

and f and g are sufficiently smooth.

To conclude this section let

$$z_{n+1} = H\left(z_n, \phi\right), \ \ z \in \mathbb{R}^n, \ \ \phi \in \mathbb{R}^p \tag{3.2.6}$$

be a discrete dynamical system where $z^* = 0$ is a permanent fixed-point, ϕ is a parameter, and $(0,0)$ is a bifurcation point such that the Jacobian matrix $D_z H(0,0)$ has c eigenvalues with modulus equal to one, and all the other eigenvalues have modulus less than one; thus $n = c + s$. Similar to the continuous case, Eq. (3.2.6) can be transformed into

$$\begin{cases} x_{n+1} = Ax_n + f(x_n, y_n, \phi), \\ y_{n+1} = By_n + g(x_n, y_n, \phi), \\ \phi_{n+1} = \phi_n, \end{cases} \tag{3.2.7}$$

where the conditions in (3.2.5) are satisfied. All the theorems which will be discussed for the continuous case (3.2.1) also apply to the discrete case (3.2.6), with the necessary adjustment regarding the location of the eigenvalues of the involved Jacobian matrices (see Remark 2.3.10).

In what follows, our aim is to determine the direction of the bifurcation at the point $(0, 0)$.

3.3 Reduction based on a power series approximation

This section is taken from Wiggins [85], without providing any proofs which however can be found in Carr [15].

Definition 3.3.1 (Wiggins [85])**.** *An invariant manifold for system (3.2.4)-(3.2.5) denoted by $W^c(0)$ is said be a (local) centre manifold for the equilibrium at the origin if it can locally be represented by*

$$\begin{aligned} W^c(0) \quad = \quad & \{(x, y, \phi) \in \mathbb{R}^c \times \mathbb{R}^s \times \mathbb{R}^p | y = h(x, \phi), |x| < \delta, |\phi| < \overline{\delta}, \\ & h(0, 0) = 0, Dh(0, 0) = 0\}, \end{aligned}$$

for δ and $\overline{\delta}$ sufficiently small.

The existence of a centre manifold is stated in the next result.

Theorem 3.3.2. *There exists a smooth centre manifold for Eq. (3.2.4). The dynamics of Eq. (3.2.4) restricted to the centre manifold is, for u sufficiently small, given by the following c-dimensional system of ODEs*

$$\begin{cases} \dot{u} = Au + f(u, h(u, \phi), \phi), & (u, h(u, \phi), \phi) \in \mathbb{R}^c \times \mathbb{R}^s \times \mathbb{R}^p, \\ \dot{\phi} = 0. \end{cases} \tag{3.3.1}$$

The dynamics of the system (3.2.4) near the origin is determined by the dynamics of (3.3.1) near $u = 0$ as a consequence of the following theorem.

Theorem 3.3.3. *(Wiggins [85]) i) If the equilibrium point zero in Theorem 3.3.2 is stable, asymptotically stable or unstable, then the equilibrium point zero of Eq. (3.2.4) is also stable, asymptotically stable or unstable, respectively.*
ii) Suppose that the equilibrium point zero in Theorem 3.3.2 for System 3.3.1 is stable. Then if $(x(t), y(t), \phi)$ is a solution of Eq. (3.2.4) with initial conditions $(x(0), y(0), \phi(0))$ sufficiently small, there is a solution $u(t)$ of Eq. (3.3.1) such that as $t \to \infty$

$$x(t) = u(t) + \mathcal{O}(e^{-\gamma t}), \tag{3.3.2}$$

$$y(t) = h(u(t), \phi) + \mathcal{O}(e^{-\gamma t}), \quad \gamma > 0. \tag{3.3.3}$$

The computation of the centre manifold in Definition 3.3.1 is as difficult as solving the original equation (3.2.4) because by the chain rule the defining function $y = h(x, \phi)$ must be a solution of the following quasi linear partial differential equation:

$$\mathcal{N}(h(x,\phi)) := D_x h(x,\phi)[Ax + f(x, h(x,\phi), \phi)] - Bh(x,\phi) - g(x, h(x,\phi), \phi) = 0. \tag{3.3.4}$$

Fortunately, we have the following result that provides an approximation of h to any degree of accuracy, specifically in terms of a power series expansion.

Theorem 3.3.4. *(Wiggins [85]) Let $\psi : \mathbb{R}^c \times \mathbb{R}^{\prime} \to \mathbb{R}^s$ be a C^1 mapping with $\psi(0,0) = D\psi(0,0) = 0$ such that $\mathcal{N}(\psi(x,\phi)) = \mathcal{O}(|x|^q + |\phi|^q)$ as $x, \phi \to 0$ for some $q > 1$. Then*

$$|h(x,\phi) - \psi(x,\phi)| = \mathcal{O}(|x|^q + |\phi|^q), \quad as \quad x, \phi \to 0. \tag{3.3.5}$$

Consequently, h can be computed in the form of a power series

$$h(x,\phi) = \sum_{(\alpha,\beta)\in\mathbb{N}^c\times\mathbb{N}^p} a_{\alpha,\beta} x^\alpha \phi^\beta, \tag{3.3.6}$$

in which the coefficients $a_{\alpha,\beta}{}'s$ are determined by matching powers of dependent variables in Eq. (3.3.4).

Remark 3.3.5. *The representation in Theorem 3.3.4 of the centre manifold by power series speaks for itself regarding the difficulty of this approach, particularly when the system (3.2.4) is large. We will illustrate this in the next Chapter. In the meantime, we consider an alternative approach which is relatively user friendly.*

3.4 Reduction to a scalar ODE

In this section we deal with the general dynamical system (3.2.1) in the particular case where $p = 1$ but without making use of the decomposition (3.2.4). The strategy to determine the direction of the bifurcation at the equilibrium point $(0, 0)$ consists of reducing the system (3.2.1) to the scalar equation

$$\dot{c} = \phi bc + ac^2, \tag{3.4.1}$$

where a and b are defined in Eq. (3.4.2) below. The main result due to Castillo-Chavez and Song [16] reads as follows:

Theorem 3.4.1 (Castillo-Chavez and Song [16])**.** *Assume the following:*

(i) Zero is a simple eigenvalue of $D_zH(0, 0)$ and all other eigenvalues have negative real parts.

(ii) The matrix $D_zH(0, 0)$ has a nonnegative right eigenvector $\mathbf{w}$ and a left eigenvector $\mathbf{v}$ corresponding to the zero eigenvalue.

Let H_k be the k^{th} component of H and

$$\begin{aligned} a &= \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 H_k}{\partial z_i \partial z_j}(0, 0) = \mathbf{v}\left[I_n \otimes \mathbf{w}^T\right] D^2_{zz}H(0, 0)\mathbf{w} \\ b &= \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 H_k}{\partial z_i \partial \phi}(0, 0) = \mathbf{v}D_{z\phi}H(0, 0)\mathbf{w} \end{aligned} \tag{3.4.2}$$

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

$$D^2_{zz}H = \begin{pmatrix} \frac{\partial^2 H_1}{\partial z_1 \partial z_1} & \frac{\partial^2 H_1}{\partial z_1 \partial z_2} & \cdots & \frac{\partial^2 H_1}{\partial z_1 \partial z_n} \\ \vdots & \vdots & \ddots & \vdots \\ \frac{\partial^2 H_1}{\partial z_n \partial z_1} & \frac{\partial^2 H_1}{\partial z_n \partial z_2} & \cdots & \frac{\partial^2 H_1}{\partial z_n \partial z_n} \\ \vdots & \vdots & \ddots & \vdots \\ \frac{\partial^2 H_n}{\partial z_1 \partial z_1} & \frac{\partial^2 H_n}{\partial z_1 \partial z_2} & \cdots & \frac{\partial^2 H_n}{\partial z_1 \partial z_n} \\ \vdots & \vdots & \ddots & \vdots \\ \frac{\partial^2 H_n}{\partial z_n \partial z_1} & \frac{\partial^2 H_n}{\partial z_n \partial z_2} & \cdots & \frac{\partial^2 H_n}{\partial z_n \partial z_n} \end{pmatrix} \quad \textit{is the Hessian matrix.}$$

If we assume that $b > 0$, then the local dynamics of system (3.2.1) around $z = 0$ are determined by the sign of the number a as follows:

(i) If $a > 0$, then $\phi = 0$ is a backward bifurcation in the sense of Definition 2.3.7.

(ii) If $a < 0$, then $\phi = 0$ is a forward bifurcation in the sense of Definition 2.3.7.

Remark 3.4.2. *Eq. (3.4.1) is actually a truncated Taylor series in which terms of order higher than two have been dropped. This equation takes the form*

$$\dot{c} = \phi bc + ac^2 + \sum_{\substack{i+j\geq 3 \\ i,j=0}}^{m} d_{i,j}\phi^j c^i, \tag{3.4.3}$$

if we include higher order terms. This could be essential in epidemiology to highlight the "full" meaning of the backward bifurcation phenomenon, namely the existence of a small unstable equilibrium $c^u > 0$ and a large locally asymptotically stable equilibrium $c^s > 0$ branching at $(c, \phi) \equiv (0, 0)$ for ϕ less than the critical value ϕ_c of the parameter (see Definition 2.3.7 and

Remark 2.3.8). For instance a fifth order expansion together with an appropriate choice of coefficients $d_{i,j}$'s could lead to a hysteresis as shown in Fig. 2.6.

3.5 Reduction to a scalar difference equation

We are interested in determining the direction of a bifurcation for the general discrete dynamical system (2.3.5) with $z^* = 0$ which is a permanent fixed-point. We obtain the following new result:

Theorem 3.5.1. *Assume that*

(i) The matrix $D = D_z F(0,0)$, has 1 as a simple eigenvalue of D, and all the other eigenvalues of D have modulus less than 1.

(ii) The matrix D has a nonnegative right eigenvector $\mathbf{w}$ and a left eigenvector $\mathbf{v}$ corresponding to the eigenvalue 1.

Then the local dynamics of system (2.3.5) around the fixed-point $z^* = 0$ are determined by the signs of the numbers a and b given in Eq. (3.4.2), as in Theorem 3.4.1 but with H replaced by F.

Proof. For convenience we write explicitly the analogue of formula (3.4.2)

$$\begin{aligned} a &= \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 F_k}{\partial z_i \partial z_j}(0,0) = \mathbf{v}\left[I_n \otimes \mathbf{w}^T\right] D^2_{zz} F(0,0)\mathbf{w} \\ b &= \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 F_k}{\partial z_i \partial \phi}(0,0) = \mathbf{v} D_{z\phi} F(0,0)\mathbf{w} \end{aligned} \tag{3.5.1}$$

The proof is similar to that of Theorem 3.4.1 in Castillo-Chavez and Song [16] for the continuous model. The space $\mathbb{R}^n$ can be expressed as the direct sum

$$\mathbb{R}^n = E^c \oplus E^s \,, \tag{3.5.2}$$

where E^c is the one-dimensional centre subspace and E^s is the $(n-1)$-dimensional stable subspace corresponding to the eigenvalue 1 and to all the eigenvalues that lie inside a unit circle, respectively. Note that from the decomposition (3.5.2), we have (see for instance Kuznetsov [53]) $\langle \mathbf{v}, h\rangle = 0 \ \forall \ h \in E^s$. Since one is a simple eigenvalue of $D_zF(0,0)$, the system (3.2.6) can be written in the form (3.2.7) where $c = 1$. It follows from Theorem 3.3.2 and Definition 3.3.1 that there exists a smooth centre manifold $W^c(0)$ consisting of points $(z,\phi) \equiv \left(c, \tilde{h}(c,\phi), \phi\right) \in \mathbb{R}\times\mathbb{R}^{n-1}\times\mathbb{R}$ such that $|c| < \delta$, $|\phi| < \bar{\delta}$, $\tilde{h}(0,0) = 0$, and $D_z\tilde{h}(0,0) = 0$. In view of the decomposition (3.5.2), we have

$$z_n = c_n\mathbf{w} + h(c_n,\phi). \tag{3.5.3}$$

Therefore the centre manifold is represented by

$$W^c(0) = \{(z,\phi)|z(c,\phi) = c\mathbf{w} + h(c,\phi) : \mathbf{v}\cdot h(c,\phi) = 0, |c| \le \bar{c}, c(0) = 0\}\ , \tag{3.5.4}$$

where $h(c,\phi) \in E^s$ and is of at least order 2 with respect to both c and ϕ. Using Eq. (2.3.5) together with Eq. (3.5.3) we have

$$\begin{aligned} F(c_n\mathbf{w} + h(c_n,\phi),\phi) &= z_{n+1} \\ &= c_{n+1}\mathbf{w} + h(c_{n+1},\phi). \end{aligned} \tag{3.5.5}$$

Multiplying by the left eigenvector $\mathbf{v}$ on both sides of Eq. (3.5.5), we obtain

$$c_{n+1} = \mathbf{v}F(c_n\mathbf{w} + h(c_n,\phi),\phi). \tag{3.5.6}$$

From Theorem 3.3.3 it follows that 0 is asymptotically stable for Eq. (2.3.5) if and only if 0 is an asymptotically stable fixed-point of Eq. (3.5.6). Hence, we investigate Eq. (3.5.6). By using Taylor's expansion of F about the point $(z^*,\phi) = (0,0)$, Eq. (3.5.6) becomes

$$\begin{aligned} c_{n+1} &= \mathbf{v}F(0,0) + \mathbf{v}D_\phi F(0,0)\phi \\ &\quad + \mathbf{v}D_zF(0,0)(c_n\mathbf{w}+h) + \frac{1}{2}\mathbf{v}D_{\phi\phi}F(0,0)\phi^2 \\ &\quad + \mathbf{v}D_{z\phi}F(0,0)\phi(c_n\mathbf{w}+h) + \frac{1}{2}\mathbf{v}D_{zz}F(0,0)(c_n\mathbf{w}+h)^2 + \cdots . \end{aligned} \tag{3.5.7}$$

In view of fact that $z^* = 0$ is a permanent equilibrium, 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

$$\begin{aligned}\frac{1}{2}D_{zz}F(0,0)(c_n\mathbf{w}+h)^2 &= \frac{1}{2}\left[I_n \otimes (c_n\mathbf{w}+h)^T\right] D^2_{zz}F(0,0)(c_n\mathbf{w}+h)\\ &= \frac{c_n^2}{2}\left[I_n \otimes \mathbf{w}^T\right] D^2_{zz}F(0,0)\mathbf{w} + \cdots .\end{aligned} \tag{3.5.8}$$

This simplifies Eq. (3.5.7) into

$$\begin{aligned}c_{n+1} &= \mathbf{v}c_n\mathbf{w} + \mathbf{v}D_{z\phi}F(0,0)\phi c_n\mathbf{w} + \mathbf{v}\frac{c_n^2}{2}\left[I_n \otimes \mathbf{w}^T\right] D^2_{zz}F(0,0)\mathbf{w} + \mathcal{O}(3)\\ &= c_n + \mathbf{v}D_{z\phi}F(0,0)\phi c_n\mathbf{w} + \frac{c_n^2}{2}\mathbf{v}\left[I_n \otimes \mathbf{w}^T\right] D^2_{zz}F(0,0)\mathbf{w} + \mathcal{O}(3)\\ &= (1+\phi b)c_n + ac_n^2 + \mathcal{O}(3).\end{aligned}$$

Using Theorem 3.3.3, in the neighbourhood of the equilibrium point $(z^*,\phi) = (0,0)$, System (2.3.5) has dynamics that are the same as those of the simpler map

$$c_{n+1} = (1+\phi b)c_n + ac_n^2. \tag{3.5.9}$$

This is precisely the manner in which it is stated in the theorem. □

Remark 3.5.2. *In line with the normal form in Eq. (2.4.4) for a transcritical bifurcation for a continuous dynamical system, Eq. (3.5.9) is the normal form of a transcritical bifurcation for a discrete dynamical system. The bifurcation diagrams of the discrete analogues of Eqs (2.4.5), (2.4.6), (2.4.8), and (2.4.9) are given in the following figures which are similar to the continuous cases for* $a = \pm 2.09$ *and* $b = 1$*:*

$$c_{n+1} = R_0c_n + 2.09c_n^2 \quad \textit{where} \quad \phi = R_0 - 1. \tag{3.5.10}$$

$$c_{n+1} = R_0c_n - 2.09c_n^2. \tag{3.5.11}$$

$$c_{n+1} = R_0c_n + 2.09c_n^2 - c_n^3 - 0.001c_n^4 - c_n^5 \tag{3.5.12}$$

$$c_{n+1} = R_0c_n - 2.09c_n^2 - c_n^3 - 0.001c_n^4 - c_n^5 \tag{3.5.13}$$

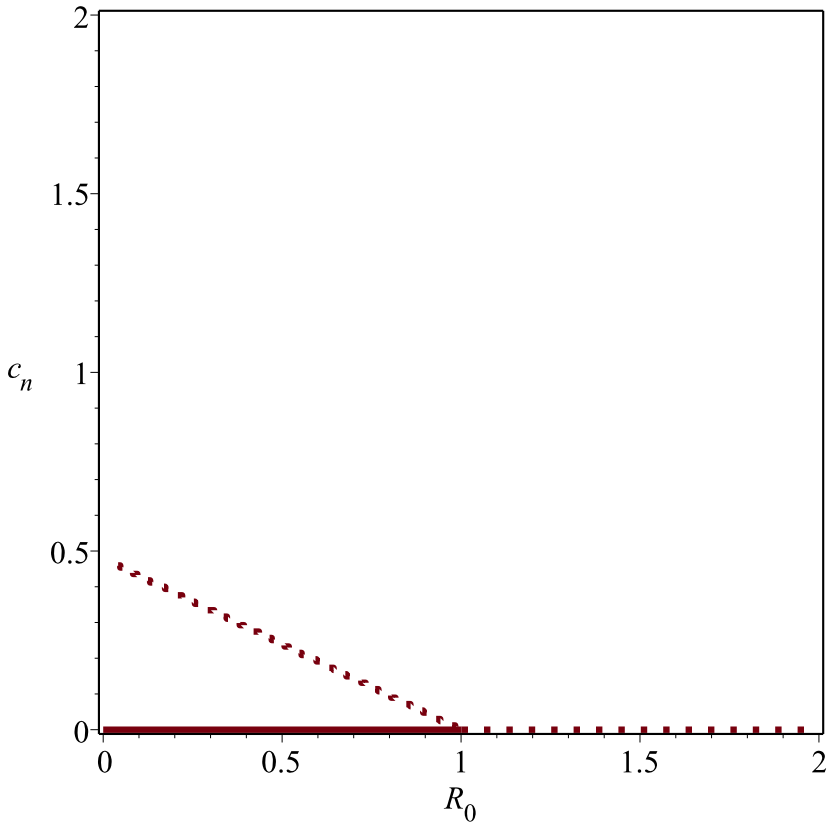


Figure 3.1: Eq. (3.5.10) undergoes a backward bifurcation at $R_0 = 1$.

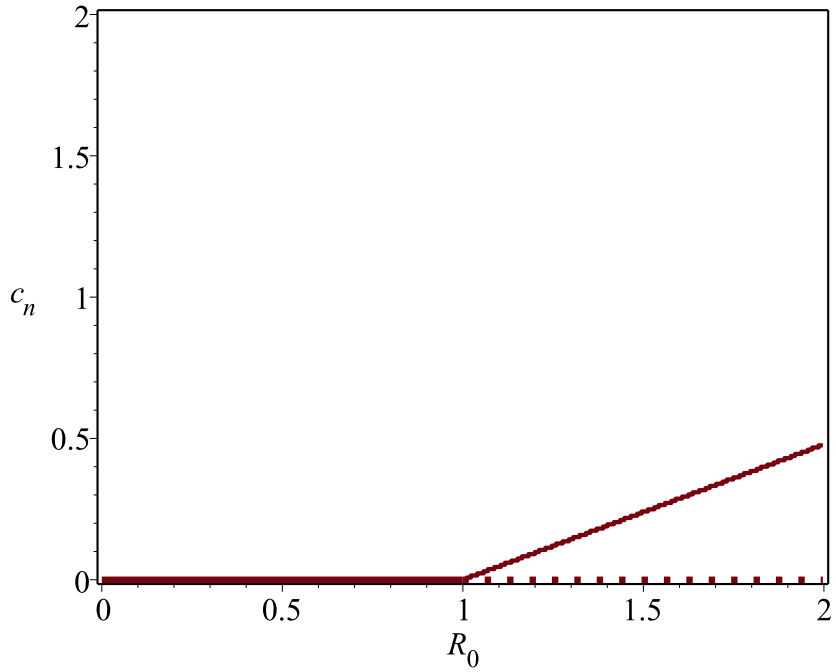


Figure 3.2: Eq. (3.5.11) undergoes a forward bifurcation at $R_0 = 1$.

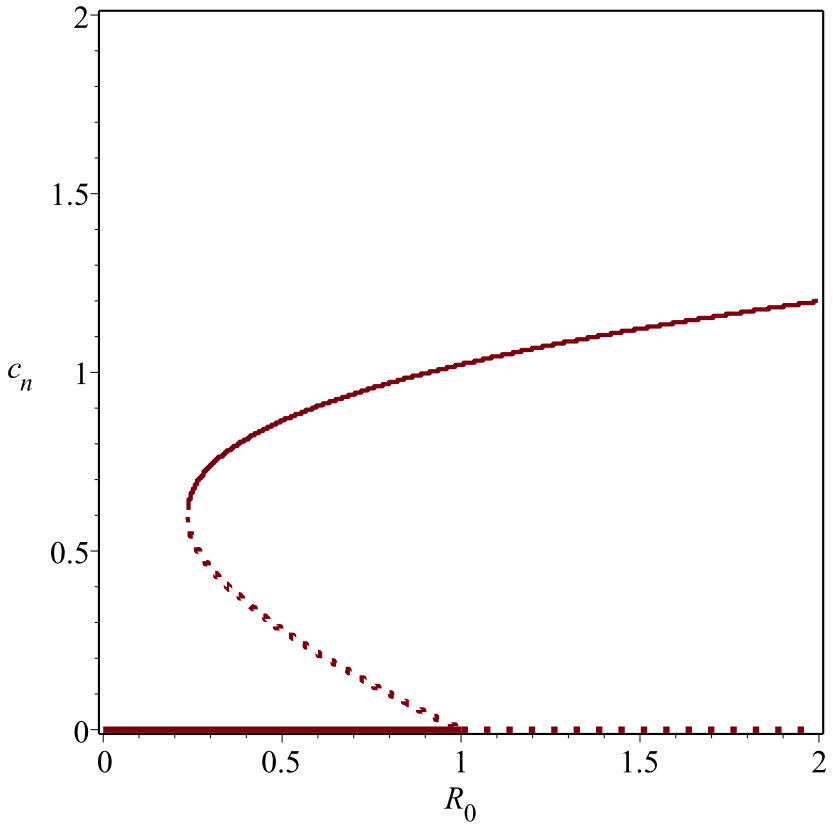


Figure 3.3: Eq. (3.5.12) undergoes a backward bifurcation at $R_0 = 1$.

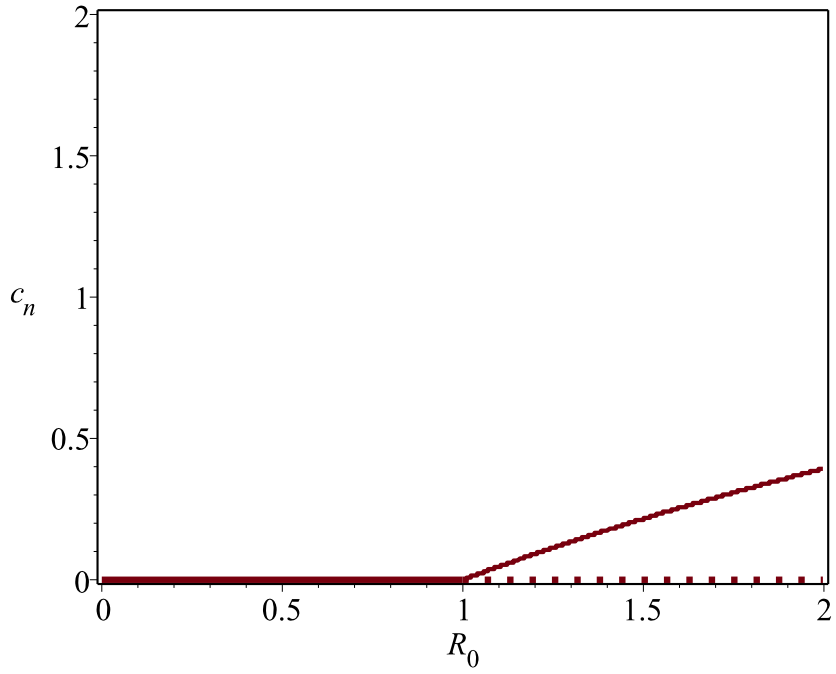


Figure 3.4: Eq. (3.5.13) undergoes a forward bifurcation at $R_0 = 1$.

CHAPTER 4. APPLICATIONS TO EPIDEMIOLOGICAL MODELS

4.1 Introduction

Vaccination is effective at preventing diseases from spreading throughout the entire population. However, there are challenges associated with vaccine administration. Many mathematical models are designed to give insight into what could possibly go wrong when rolling out a vaccination strategy if critical factors are ignored (see for instance Dushoff et al. [31]). The SIS model (Villavicencio-Pulido et al. [79]) considered in this Chapter is relatively simple. This choice is made deliberately in order to illustrate in a simple manner, by using the centre manifold theory, the challenge of the existence of backward bifurcation in terms of vaccine coverage and efficacy. The simple structure of the SIS model enables us to implement the two reduction methods that are presented in Chapter 3 to demonstrate the existence of the backward bifurcation. As a second step, a malaria model proposed by Chitnis et al. [23] is considered in this Chapter. Apart from the high number of equations involved, the complexity of this system is apparent from the strong nonlinearity. As a result of this, reduction via power series approximations is not a viable option. We establish the existence of backward bifurcation by the approach proposed in Castillo-Chavez and Song [16]. The results of this Chapter as well as their nonstandard discretisations presented in Chapter 5 are published in Anguelov et al. [9].

4.2 SIS model with vaccination

4.2.1 The model

In this subsection, we consider the SIS model proposed in Villavicencio-Pulido et al. [79]. The system corresponding to the flow diagram in Fig. 4.1 as well as to the variables and parameters in Table 4.1 reads as follows:

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*
σ	*Transmission rate inhibitor*
β	*Transmission rate*
ϕ_v	*Vaccination rate of the susceptible*

Table 4.1: Model variables and parameters for the SIS model

$$\begin{aligned} \dot{S} &= -\beta S\frac{I}{N} - \phi_v S + cI, \\ \dot{I} &= \beta S\frac{I}{N} + \sigma\beta V\frac{I}{N} - cI, \\ \dot{V} &= -\sigma\beta V\frac{I}{N} + \phi_v S. \end{aligned} \tag{4.2.1}$$

It is clear that the total population $N = S + I + V$ is constant . The disease-free equilibrium (DFE) of System (4.2.1) is found by setting the right side of the system and I equal zero and solving for S and V. Consequently, we obtain

$$DFE = (S^*, I^*, V^*) = (0, 0, N). \tag{4.2.2}$$

The basic reproduction number denoted by R_0 is given by

$$R_0 = \frac{\sigma\beta}{c}. \tag{4.2.3}$$

Indeed, we use the next generation matrix method and let

$$\dot{I} = \mathcal{F}(S, I, V) - \mathcal{V}(S, I, V), \tag{4.2.4}$$

where $\mathcal{F}$ is the rate of appearance of new infections in the Infectious class and $\mathcal{V}$ is the transfer rate of individuals out of the Infectious class (see Van den Driessche and Watmough [78]). Then

$$R_0 = \left.\frac{\frac{\partial \mathcal{F}}{\partial I}}{\frac{\partial \mathcal{V}}{\partial I}}\right|_{DFE} = \frac{\sigma\beta}{c}.$$

Since N is constant, the system can be reduced to a two-dimensional system. We consider two lower dimensional systems that we will use in this Chapter. The first system is obtained by eliminating $V = N - I - S$. It reads as follows:

$$\begin{aligned} \dot{S} &= -\beta S\frac{I}{N} - \phi_v S + cI, \\ \dot{I} &= \beta, S\frac{I}{N} + \sigma\beta(N - S - I)\frac{I}{N} - cI. \end{aligned} \tag{4.2.5}$$

Similarly, we eliminate $S = N - I - V$ from System (4.2.1) so that

$$\begin{aligned} \dot{I} &= \beta(N - I - V)\frac{I}{N} + \sigma\beta V\frac{I}{N} - cI, \\ \dot{V} &= -\sigma\beta V\frac{I}{N} + \phi_v(N - I - V). \end{aligned} \tag{4.2.6}$$

The second reduction comes from Kribs-Zaleta and Velasco-Hernández [52]. We introduce a new state variable $\overline{V}$ and parameter ρ defined as follows:

$$\overline{V} = V - N \quad \text{and} \quad \rho = R_0 - 1. \tag{4.2.7}$$

Upon substitution of the transformations (4.2.7) into (4.2.6), we obtain

$$\begin{aligned} \dot{I} &= \beta(N - I - \overline{V})\frac{I}{N} - \beta I + \sigma\beta\overline{V}\frac{I}{N} + \sigma\beta I - cI, \\ \dot{\overline{V}} &= -\sigma\beta V\frac{I}{N} + \phi_v(N - I - V). \end{aligned} \tag{4.2.8}$$

As a consequence of equations (4.2.3) and (4.2.7), our second system is given by

$$\begin{aligned} \dot{I} &= -\beta\frac{I^2}{N} + (\sigma - 1)\beta\overline{V}\frac{I}{N} + \rho cI, \\ \dot{\overline{V}} &= -\sigma\beta\overline{V}\frac{I}{N} - \sigma\beta I - \phi_v I - \phi_v\overline{V}. \end{aligned} \tag{4.2.9}$$

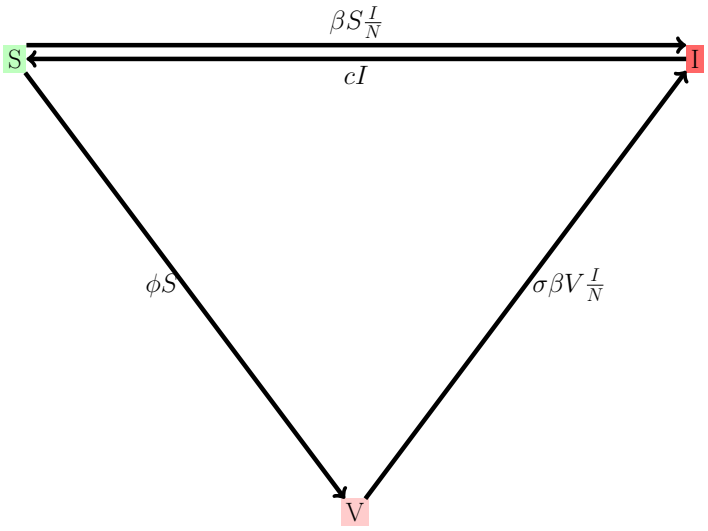


Figure 4.1: Flow diagram of the SIS model

[1]

4.2.2 Application of a power series approximation

In this Subsection, we apply Theorem 3.3.4 to the system (4.2.9). To this end, we need to modify it into an equivalent system that has the required form as per (3.2.4). We linearise System (4.2.9) at the DFE $(I^*, \overline{V}^*) = (0, 0)$ to obtain

$$\dot{X} = JX + F(X) \tag{4.2.10}$$

where

$$\dot{X} = \begin{pmatrix} \dot{I} \\ \dot{\overline{V}} \end{pmatrix}, J = \begin{pmatrix} 0 & 0 \\ -(\sigma\beta + \phi_v) & -\phi_v \end{pmatrix}, F = \begin{pmatrix} -\beta\frac{I^2}{N} + (\sigma - 1)\beta\overline{V}\frac{I}{N} + \rho cI \\ -\sigma\beta\overline{V}\frac{I}{N} \end{pmatrix}. \tag{4.2.11}$$

The eigenvalues of the matrix J are $\lambda_1 = 0$ and $\lambda_2 = -\phi_v$ with eigenvectors

$$\begin{pmatrix} -\dfrac{\phi_v}{(\sigma\beta+\phi_v)} \\ 1 \end{pmatrix}, \begin{pmatrix} 0 \\ 1 \end{pmatrix}, \quad \text{respectively.} \tag{4.2.12}$$

The transition matrix T and its inverse are given by

$$T = \begin{pmatrix} -\dfrac{\phi_v}{(\sigma\beta+\phi_v)} & 0 \\ 1 & 1 \end{pmatrix}, \quad \text{and} \quad T^{-1} = \begin{pmatrix} -\dfrac{(\sigma\beta+\phi_v)}{\phi_v} & 0 \\ \dfrac{(\sigma\beta+\phi_v)}{\phi_v} & 1 \end{pmatrix}. \tag{4.2.13}$$

By using the transformation matrix we obtain

$$\begin{pmatrix} I \\ \overline{V} \end{pmatrix} = T \begin{pmatrix} x \\ y \end{pmatrix} = \begin{pmatrix} -\dfrac{\phi_v}{(\sigma\beta+\phi_v)}x \\ x+y \end{pmatrix}. \tag{4.2.14}$$

Consequently,

$$F(x,y) = \begin{pmatrix} F_1 \\ F_2 \end{pmatrix}, \tag{4.2.15}$$

where

$$\begin{aligned} F_1 &= -\left(\left(\frac{\phi_v}{\sigma\beta+\phi_v}\right)+(\sigma-1)\right)\frac{\beta}{N}\left(\frac{\phi_v}{\sigma\beta+\phi_v}\right)x^2 \\ &\quad -(\sigma-1)\frac{\beta}{N}\left(\frac{\phi_v}{\sigma\beta+\phi_v}\right)xy - \left(\frac{\phi_v}{\sigma\beta+\phi_v}\right)\rho c x, \\ F_2 &= \frac{\sigma\beta}{N}\left(\frac{\phi_v}{\sigma\beta+\phi_v}\right)x^2 + \frac{\sigma\beta}{N}\left(\frac{\phi_v}{\sigma\beta+\phi_v}\right)xy. \end{aligned} \tag{4.2.16}$$

The transformed system reads as follows:

$$\begin{pmatrix} \dot{x} \\ \dot{y} \end{pmatrix} = T^{-1}JT \begin{pmatrix} x \\ y \end{pmatrix} + T^{-1}F(x,y). \tag{4.2.17}$$

Upon simplification we express the transformed system in the desired form of Eq. (3.2.4) as

$$\begin{aligned} \dot{x} &= \rho c x + \frac{\beta}{N}\left(\frac{\phi_v}{\sigma\beta+\phi_v}+(\sigma-1)\right)x^2 + (\sigma-1)\frac{\beta}{N}xy, \\ \dot{y} &= -\phi_v y - \rho c x - (\sigma-1)\frac{\beta}{N}x^2 + \left(\frac{\sigma\phi_v}{\sigma\beta+\phi_v}-(\sigma-1)\right)\frac{\beta}{N}xy, \\ \dot{\rho} &= 0, \end{aligned} \tag{4.2.18}$$

where

$$\begin{cases} A=0, \quad f=\rho cx+\dfrac{\beta}{N}\left(\dfrac{\phi_v}{\sigma\beta+\phi_v}+(\sigma-1)\right)x^2+(\sigma-1)\dfrac{\beta}{N}xy, \\ B=-\phi_v, \quad g=-\rho cx-(\sigma-1)\dfrac{\beta}{N}x^2+\left(\dfrac{\sigma\phi_v}{\sigma\beta+\phi_v}-(\sigma-1)\right)\dfrac{\beta}{N}xy. \end{cases} \tag{4.2.19}$$

In view of Theorem 3.3.4, the centre manifold as per Definition 3.3.1 is computed through a power series of the form

$$y=h(x,\rho)=a_1x^2+a_2x\rho+a_3\rho^2+\cdots \tag{4.2.20}$$

such that

$$\begin{aligned} \mathcal{N}(h(x,\rho)) &= \frac{\partial h}{\partial x}\left(Ax+f(x,h(x,\rho),\rho)-Bh(x,\rho)-g(x,h(x,\rho),\rho)\right) \\ &= (2a_1x+a_2\rho+\cdots)\left[\rho cx+\frac{\beta}{N}\left(\frac{\phi_v}{\sigma\beta+\phi_v}+(\sigma-1)\right)x^2\right. \\ &+ \left.(\sigma-1)\frac{\beta}{N}x(a_1x^2+a_2x\rho+a_3\rho^2+\cdots)\right] \\ &+ \phi_v(a_1x^2+a_2x\rho+a_3\rho^2+\cdots)+c\rho x+(\sigma-1)\frac{\beta}{N}x^2 \\ &+ \left(\frac{\sigma\phi_v}{\sigma\beta+\phi_v}-(\sigma-1)\right)\frac{\beta}{N}x(a_1x^2+a_2x\rho+a_3\rho^2+\cdots)=0. \end{aligned} \tag{4.2.21}$$

Upon substitution of equations (4.2.19) into Eq. (4.2.21) and matching of powers as suggested in Theorem 3.3.4 we obtain the following:

$$\begin{cases} \text{For} \quad x^2, \quad \phi_v a_1+(\sigma-1)\dfrac{\beta}{N}=0 \quad \text{so that} \quad a_1=\dfrac{(1-\sigma)\beta}{\phi_v N}, \\ \text{For} \quad x\rho, \quad \phi_v a_2+c=0 \quad \text{so that} \quad a_2=-\dfrac{c}{\phi_v}. \end{cases} \tag{4.2.22}$$

Therefore, Eq. (4.2.20) is precisely

$$y=h(x,\rho)=\frac{(1-\sigma)\beta}{\phi_v N}x^2-\frac{c}{\phi_v}x\rho+\mathcal{O}(3), \tag{4.2.23}$$

from which, in view of Eq. (4.2.18), we obtain

$$\dot{x}=c\rho x-\frac{\sigma\beta[(1-\sigma)\beta-\phi_v]}{N(\sigma\beta+\phi_v)}x^2+\mathcal{O}(3). \tag{4.2.24}$$

Using Eq. (4.2.14) in Eq. (4.2.24), we obtain

$$\dot{I} = c\rho I + \frac{\sigma\beta[(1-\sigma)\beta - \phi_v]}{N\phi_v} I^2 + \mathcal{O}(3). \tag{4.2.25}$$

The structure of Eq. (4.2.25) is that of the normal form of a transcritical bifurcation as in Eq. (2.4.4). The stability of equilibrium solution of System (4.2.1) is determined by the stability of Eq. (4.2.25) by Theorem 3.3.2 in the more specific manner stated in the next result.

Theorem 4.2.1. *Model (4.2.1) undergoes a backward bifurcation at* $R_0 = 1$ *if*

$$\mathcal{A}(\phi_v) = (1-\sigma)\beta - \phi_v > 0. \tag{4.2.26}$$

Remark 4.2.2. *There can be no backward bifurcation if the vaccine is absolutely ineffective* ($\sigma = 1$). *If the vaccine is totally effective* ($\sigma = 0$), *i.e.* $R_0 = 0$ *and* $\rho = -1$), *there is no bifurcation and the DFE is always asymptotically stable.*

4.2.3 Application of Theorem 3.4.1

In this subsection, we perform bifurcation analysis of Model (4.2.1) by applying Theorem 3.4.1 to the system (4.2.5). Let $(z_1, z_2) = (S, I)$ and $\rho = R_0 - 1$. The Jacobian matrix of equation (4.2.5) evaluated at the DFE $(S^*, I^*) = (0, 0)$ is

$$J = \begin{pmatrix} -\phi_v & c \\ \\ 0 & \rho c \end{pmatrix}, \tag{4.2.27}$$

with eigenvalues $\lambda_1 = -\phi_v$ and $\lambda_2 = \rho c$.

Note that λ_1 is always less than zero, but the sign of λ_2 depends on the parameter ρ. If $\rho \neq 0$, then we may apply the Hartman-Grobman theorem (i.e. Theorem 2.1.7) to determine the local stability of the DFE because the matrix J is hyperbolic.

Our interest is in the local stability of the DFE at $\rho = 0$ $(i.e.\ R_0 = 1)$ which makes $\lambda_2 = 0$. In this case J is nonhyperbolic and the Hartman-Grobman Theorem does not apply. Hence we apply Theorem 3.4.1 . For $\rho = 0$, the simple eigenvalue $\lambda_2 = 0$ has the following associated right and left eigenvectors

$$\mathbf{w} = \begin{pmatrix} \frac{c}{\phi_v} \\ 1 \end{pmatrix} \text{ and } \mathbf{v^T} = \begin{pmatrix} 0 \\ 1 \end{pmatrix}, \tag{4.2.28}$$

respectively. We are therefore in the setting of Theorem 3.4.1. Since $v_1 = 0$ there is no need to evaluate the partial derivatives of H_1. On the contrary partial derivatives of H_2 evaluated at $(z_1, z_2, \rho) = (0, 0, 0)$ are needed and they are equal to

$$\frac{\partial^2 H_2}{\partial x_1^2} = 0, \quad \frac{\partial^2 H_2}{\partial x_1 \partial x_2} = (1-\sigma)\frac{\beta}{N}, \quad \frac{\partial^2 H_2}{\partial x_2^2} = \frac{-2\beta\sigma}{N}, \quad \frac{\partial^2 H_2}{\partial x_1 \partial \rho} = 0, \quad \frac{\partial^2 H_2}{\partial x_2 \partial \rho} = c. \tag{4.2.29}$$

Upon substitution into Eq. (3.4.2) we obtain

$$a = \frac{2\sigma\beta}{\phi_v N}((1-\sigma)\beta - \phi_v) = \frac{2\sigma\beta}{\phi_v N}\mathcal{A}(\phi_v) \tag{4.2.30}$$

and

$$b = c > 0. \tag{4.2.31}$$

The conclusion of Theorem 3.4.1 is stated in the following result, which is a rephrasing of Theorem 4.2.1.

Theorem 4.2.3. *The value of $R_0 = 1$ is a backward or forward bifurcation depending on whether $a > 0$ or $a < 0$.*

Remark 4.2.4. *If $a > 0$ then the disease-free equilibrium is globally asymptotically stable if*

$$0 < \beta - \phi_v < c \quad \textit{and} \quad R_0 < R_0^c := \frac{4\beta\phi_v}{[\beta - \phi_v - c]^2 + 4\beta\phi_v}. \tag{4.2.32}$$

Indeed, to prove this, we consider Eq. (4.2.6) at the endemic equilibrium. We set the right hand-side of this equation to zero to obtain

$$\begin{aligned} \beta(N-I-V)\frac{I}{N}+\sigma\beta V\frac{I}{N}-cI &= 0, \\ -\sigma\beta V\frac{I}{N}+\phi_v(N-I-V) &= 0. \end{aligned} \tag{4.2.33}$$

Solving for V in the second equation we obtain

$$\begin{aligned} V &= \frac{\phi_v(N-I)}{\sigma\beta\dfrac{I}{N}+\phi_v} \\ &= \frac{N\phi_v(N-I)}{cR_0I+\phi_vN}. \end{aligned} \tag{4.2.34}$$

Simplification of the first equation in (4.2.33) and the expression of R_0 in (4.2.3) leads to

$$\begin{aligned} 0 &= \left[\beta N-\beta I-\beta(1-\sigma)V\right]\frac{I}{N}-cI \\ &= \beta I-\frac{\beta}{N}I^2-\frac{\beta}{N}VI+\frac{\sigma\beta}{N}VI-cI \\ &= \beta I-\frac{\beta}{N}I^2-\frac{\beta}{N}VI+\frac{cR_0}{N}VI-cI. \end{aligned} \tag{4.2.35}$$

Substitution of Eq. (4.2.34) into Eq. (4.2.35) leads to

$$-\frac{\beta cR_0}{N}I^3+\left[\beta-\phi_v-c\right]cR_0I^2+(R_0-1)c\phi_vNI=0. \tag{4.2.36}$$

We are not interested in the trivial root $I=0$ of Eq. (4.2.36). Its nonzero roots are obtained from the quadratic equation

$$AI^2+BI+C=0 \tag{4.2.37}$$

where

$$\begin{aligned} A &= -\frac{\beta cR_0}{N}, \\ B &= \left[\beta-\phi_v-c\right]cR_0, \\ C &= N(R_0-1)c\phi_v. \end{aligned} \tag{4.2.38}$$

Global asymptotic stability of the DFE will be achieved if there is no positive real root of Eq. (4.2.36). To investigate the values of R_0 for which the DFE is globally asymptotically

stable we set $B^2 - 4AC < 0$. *After some algebraic manipulation we obtain*

$$R_0 < \frac{4\beta\phi_v}{[\beta - \phi_v - c]^2 + 4\beta\phi_v} =: R_0^c \tag{4.2.39}$$

where R_0^c *is the value of* R_0 *for which* $B^2 - 4AC = 0$. *The proof is complete.*

4.3 A malaria model

Malaria is defined as a specific protozoal infection transmitted by certain mosquitoes. In humans the causal parasite passes through separate phases of development in the liver and red blood cells (see Richardson and Woodruff [68] for instance). This disease can be fatal in some cases as reported in WHO [83]. In the East and Southern Africa region alone, a total of 13.5 million (2010), 34.0 million (2015), 45.6 million (2017) malaria cases were confirmed. This shows an increase of 238% for the period 2010-2017. A total of 70, 700 (2010), 38, 300 (2015), 20, 100 (2017) malaria related deaths were reported. Even more concerning is that children under 5 years of age constitute a huge portion of the totals reported by these statistics. Indeed, this shows that malaria still poses a challenge. From the pioneering work of Ross [74], improved by McDonald [59] in yet another seminal work, there has been a great deal of progress in the mathematical modeling of malaria (see Smith et al. [76] for the timeline). In this section, we focus on one of the relatively recent models proposed by Chitnis et al. [23] as described in Subsection 4.3.1. We sharpen the conjecture made in Chitnis et al. [23], namely that a backward bifurcation occurs for sufficiently large values of the disease-induced death rate. This is done by the centre manifold theory through a theorem due to Castillo-Chavez and Song [16].

4.3.1 The model and basic results

The malaria model in Chitnis et al. [23] is a $SEIRS$ model for the human population and a SEI model for the vector population.

- Human and vector population sizes are assumed not to be 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.
- Mosquitoes 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 4.1, and the flow diagram is in Fig. 4.1. The corresponding model is

The corresponding model is

$$\begin{cases} \dot{S}_h(t) = \Lambda_h + \psi_h N_h(t) + \rho_h R_h(t) - c\left(N_h(t), N_v(t)\right)\beta_{hv} I_v(t) S_h(t) - f_h\left(N_h(t)\right) S_h(t), \\ \dot{E}_h(t) = c\left(N_h(t), N_v(t)\right)\beta_{hv} I_v(t) S_h(t) - M_1 E_h(t), \\ \dot{I}_h(t) = \nu_h E_h(t) - M_2 I_h(t), \\ \dot{R}_h(t) = \gamma_h I_h(t) - M_3 R_h(t), \\ \dot{S}_v(t) = \psi_v N_v(t) - c\left(N_h(t), N_v(t)\right)\left(\beta_{vh} I_h(t) + \tilde{\beta}_{vh} R_h(t)\right) S_v(t) - f_v\left(N_v(t)\right) S_v(t), \\ \dot{E}_v(t) = c(N_h(t), N_v(t))\left(\beta_{vh} I_h(t) + \tilde{\beta}_{vh} R_h(t)\right) S_v(t) - M_4 E_v(t), \\ \dot{I}_v(t) = \nu_v E_v(t) - f_v\left(N_v(t)\right) I_v(t), \end{cases} \tag{4.3.1}$$

Λ_h	*Immigration rate of humans.* $Humans \times Days^{-1}$
ψ_h	*Per capita birth rate of humans.* $Days^{-1}$
ψ_v	*Per capita birth rate of mosquitoes.* $Days^{-1}$
σ_v	*Number of times one mosquito would want to bite humans per unit time, if humans were freely available.* $Days^{-1}$
σ_h	*The maximum number of mosquito bites a human can have per unit time.* $Days^{-1}$
β_{hv}	*Probability of transmission of infection from an infectious mosquito to a susceptible human, given that a contact between the two occurs. Dimensionless*
β_{vh}	*Probability of transmission of infection from an infectious human to a susceptible mosquito, given that a contact between the two occurs. Dimensionless*
$\tilde{\beta}_{vh}$	*Probability of transmission of infection from a recovered human to a susceptible mosquito, given that a contact between the two occurs. Dimensionless*
ν_h	*Per capita rate of progression of humans from the exposed state to the infectious state.* $Days^{-1}$
ν_v	*Per capita rate of progression of mosquitoes from the exposed state to the infectious state.* $Days^{-1}$
γ_h	*Per capita recovery rate for humans from the infectious state to the recovered state.* $Days^{-1}$
δ_h	*Per capita disease-induced death rate.* $Days^{-1}$
ρ_h	*Per capita rate of loss of immunity for humans.* $Days^{-1}$
μ_{1h}	*Density-independent part of the death rate for humans.* $Days^{-1}$
μ_{2h}	*Density-dependent part of the death rate for humans.* $Humans^{-1} \times Days^{-1}$
μ_{1v}	*Density-independent part of the death rate for mosquitoes.* $Days^{-1}$
μ_{2v}	*Density-dependent part of the death rate for mosquitoes.* $Mosquitoes^{-1} \times Days^{-1}$

Table 4.2: Parameters for the malaria model

where:

$$N_h(t) := S_h(t) + E_h(t) + I_h(t) + R_h(t)$$
$$\text{and} \tag{4.3.2}$$
$$N_v(t) := S_v(t) + E_v(t) + I_v(t)$$

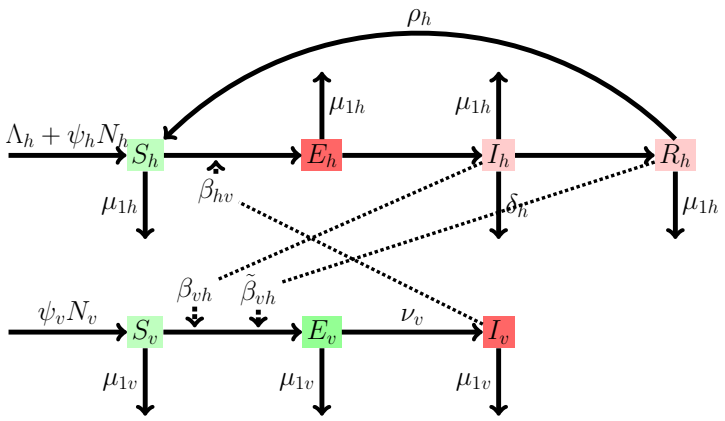


Figure 4.2: Flow diagram for host-vector dynamics of the malaria model

are total populations of humans and mosquitoes, respectively;

$$\begin{cases} c(N_h(t), N_v(t)) = \dfrac{\sigma_v \sigma_h}{\sigma_h N_h(t) + \sigma_v N_v(t)}, \\ f_h(N_h(t)) = \mu_{1h} + \mu_{2h} N_h(t), \\ f_v(N_v(t)) = \mu_{1v} + \mu_{2v} N_v(t), \\ M_1 = \nu_h + f_h, \;\; M_2 = \gamma_h + \delta_h + f_h, \;\; M_3 = \rho_h + f_h, \;\; M_4 = \nu_v + f_v. \end{cases} \tag{4.3.3}$$

By adding from Eq. (4.3.1) human-related equations alone and mosquito-related equations separately, we obtain the conservation laws in the form:

$$\dot{N}_h = \Lambda_h + \psi_h N_h - f_h N_h - \delta_h I_h \quad \text{and} \quad \dot{N}_v = \psi_v N_v - f_v N_v. \tag{4.3.4}$$

It is shown in Chitnis et al. [23] that model (4.3.1) is epidemiologically and mathematically well-posed in the domain:

$$\mathcal{D} = \{z = (S_h, E_h, I_h, R_h, S_v, E_v, I_v) | (S_h, E_h, I_h, R_h, S_v, E_v, I_v) \geq 0\}. \tag{4.3.5}$$

That is for any initial conditions in $\mathcal{D}$, the system (4.3.1) has a unique solution which remains in $\mathcal{D}$ for all $t \geq 0$. Further, the malaria model (4.3.1) has exactly one disease-free equilibrium (DFE) point, $z^* = (N_h^*, 0, 0, 0, N_v^*, 0, 0) \in \mathcal{D}$, where

$$N_h^* = \frac{\psi_h - \mu_{1h} + \sqrt{(\psi_h - \mu_{1h})^2 + 4\mu_{2h}\Lambda_h}}{2\mu_{2h}} \quad \text{and} \quad N_v^* = \frac{\psi_v - \mu_{1v}}{\mu_{2v}}. \tag{4.3.6}$$

The basic reproduction number derived in Chitnis et al. [23] may be rewritten as

$$R_0 = c^* \left(\frac{\beta_{hv}\nu_h\nu_v N_h^* N_v^* \left(\beta_{vh} + \dfrac{\tilde{\beta}_{vh}\gamma_h}{M_3^*}\right)}{f_v^* M_1^* M_2^* M_4^*} \right)^{1/2} \tag{4.3.7}$$

where the symbol $(^*)$ represents the variables in Eq. (4.3.3) evaluated at the DFE z^*. Indeed, we use the next generation method to derive Eq. (4.3.7) by considering the diseased compartments $\tilde{z} = \{E_h, I_h, R_h, E_v, I_v\}$. Let

$$\mathcal{F} = \begin{pmatrix} c\beta_{hv} I_v S_h \\ 0 \\ 0 \\ c\left(\beta_{vh} I_h + \tilde{\beta}_{vh} R_h\right) S_v \\ 0 \end{pmatrix} \quad \text{and} \quad \mathcal{V} = \begin{pmatrix} M_1 E_h \\ -\nu_h E_h + M_2 I_h \\ -\gamma_h I_h + M_3 R_h \\ M_4 E_v \\ -\nu_v E_v + f_v I_v \end{pmatrix}, \tag{4.3.8}$$

Eq. (4.3.8) leads to

$$\left.\frac{\partial \mathcal{F}}{\partial \tilde{z}}\right|_{DFE} = F = \begin{pmatrix} 0 & 0 & 0 & 0 & c^*\beta_{hv}N_h^* \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & c^*\beta_{vh}N_v^* & c^*\tilde{\beta}_{vh}N_v^* & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

$$\left.\frac{\partial \mathcal{V}}{\partial \tilde{z}}\right|_{DFE} = V = \begin{pmatrix} M_1^* & 0 & 0 & 0 & 0 \\ -\nu_h & M_2^* & 0 & 0 & 0 \\ 0 & -\gamma_h & M_3^* & 0 & 0 \\ 0 & 0 & 0 & M_4^* & 0 \\ 0 & 0 & 0 & -\nu_v & f_v^* \end{pmatrix}.$$

By using the elementary row operations for augmented matrices and the equivalence relation $[V|I] \sim [I|V^{-1}]$ we obtain the following inverse of the matrix V

$$V^{-1} = \begin{pmatrix} \frac{1}{M_1^*} & 0 & 0 & 0 & 0 \\ \frac{\nu_h}{M_1^* M_2^*} & \frac{1}{M_2^*} & 0 & 0 & 0 \\ \frac{\gamma_h \nu_h}{M_1^* M_2^* M_3^*} & \frac{\gamma_h}{M_2^* M_3^*} & \frac{1}{M_3^*} & 0 & 0 \\ 0 & 0 & 0 & \frac{1}{M_4^*} & 0 \\ 0 & 0 & 0 & \frac{\nu_v}{M_4^* f_v^*} & \frac{1}{f_v^*} \end{pmatrix}.$$

After performing standard matrix multiplication we obtain

$$FV^{-1} = \begin{pmatrix} 0 & 0 & 0 & F_{14} & F_{15} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ F_{41} & F_{42} & F_{43} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix} \tag{4.3.9}$$

where

$$\begin{aligned} F_{14} &= \frac{c^*\beta_{hv}N_h^*\nu_v}{f_v^*M_4^*}, \\ F_{15} &= \frac{c^*\beta_{hv}N_h^*}{f_v^*}, \\ F_{41} &= \frac{c^*\beta_{vh}N_v^*\nu_h}{M_1^*M_2^*} + \frac{c^*\gamma_h\nu_h\tilde{\beta}_{vh}N_v^*}{M_1^*M_2^*M_3^*}, \\ F_{42} &= \frac{c^*\beta_{vh}N_v^*}{M_2^*} + \frac{c^*\gamma_h\tilde{\beta}_{vh}N_v^*}{M_2^*M_3^*}, \\ F_{43} &= \frac{c^*\tilde{\beta}_{vh}N_v^*}{M_3^*}. \end{aligned} \tag{4.3.10}$$

The dominant eigenvalue of the matrix in Eq. (4.3.9) is

$$\rho\left(FV^{-1}\right) = c^*\left(\frac{\beta_{hv}\nu_h\nu_v N_h^*N_v^*\left(\beta_{vh} + \dfrac{\tilde{\beta}_{vh}\gamma_h}{M_3^*}\right)}{f_v^*M_1^*M_2^*M_4^*}\right)^{1/2} = R_0. \tag{4.3.11}$$

The following result regarding the bifurcation at $R_0 = 1$ is proven in Chitnis et al. [23].

Theorem 4.3.1. *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 a forward bifurcation.*

Furthermore, Chitnis et al. [23] stated the following conjecture, which is the motivation for our research.

Conjecture 4.3.2. *If the disease-induced death rate δ_h is large enough, the model (4.3.1) undergoes a backward bifurcation at $R_0 = 1$.*

4.3.2 Towards the existence of a backward bifurcation

Inspired by the Conjecture 4.3.2, here we investigate the properties of the bifurcation at $R_0 = 1$ when the disease-induced death rate is positive and thus extend Theorem 4.3.1. 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 centre manifold theory. By definition, any endemic equilibrium point $z^{**} = (S_h^{**}, E_h^{**}, I_h^*, R_h^{**}, S_v^{**}, E_v^{**}, I_v^{**})$ is obtained by setting the right-hand side of Eq. (4.3.1) equal to zero. Algebraic manipulations show that

$$\begin{cases} S_h^{**} = \dfrac{\Lambda_h + \psi_h N_h^{**} + \rho_h R_h^{**}}{c^{**}\beta_{hv} I_v^{**} + f_h^{**}}, \\ E_h^{**} = \dfrac{c^{**}\beta_{hv} I_v^{**} S_h^{**}}{M_1^{**}}, \\ R_h^{**} = \dfrac{\gamma_h I_h^*}{M_3^{**}}, \\ S_v^{**} = \dfrac{\psi_v N_v^*}{c^{**}(\beta_{vh} I_h^* + \tilde{\beta}_{vh} R_h^{**}) + f_v^*}, \\ E_v^{**} = \dfrac{c^{**}(\beta_{vh} I_h^* + \tilde{\beta}_{vh} R_h^{**}) S_v^{**}}{M_4^*}, \\ I_v^{**} = \dfrac{\nu_v E_v^{**}}{f_v^*}. \end{cases} \tag{4.3.12}$$

Moreover, from the human component of the conservation law Eq. (4.3.4) we have

$$\Lambda_h + \psi_n N_h^{**} - f_h^{**}(N_h^{**}) - \delta_h I_h^* = 0. \tag{4.3.13}$$

Since

$$f_h^{**} = \mu_{1h} + \mu_{2h} N_h^{**}, \tag{4.3.14}$$

Eq. (4.3.13) becomes the following quadratic equation in N_h^{**}

$$-\mu_{2h}\left(N_h^{**}\right)^2 + \left(\psi_h - \mu_{1h}\right) N_h^{**} + \Lambda_h - \delta_h I_h^* = 0. \tag{4.3.15}$$

Therefore,

$$N_h^{**} \equiv N_h^{**}(I_h^*) = \frac{\psi_h - \mu_{1h} \pm \sqrt{(\psi_h - \mu_{1h})^2 + 4\mu_{2h}(\Lambda_h - \delta_h I_h^*)}}{2\mu_{2h}}. \tag{4.3.16}$$

A Taylor's expansion of Eq. (4.3.16) about $I_h^* = 0$ leads to

$$N_h^{**}\left(I_h^*\right) = N_h^{**}(0) - \frac{\delta_h}{B} I_h^* + \mathcal{O}\left(I_h^{*^2}\right) \text{ where } B = \sqrt{(\psi_h - \mu_{1h})^2 + 4\mu_{2h}\Lambda_h}. \tag{4.3.17}$$

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

$$N_h^{**} = N_h^* - FI_h^* \quad \text{where} \quad F = \frac{\delta_h}{B}. \tag{4.3.18}$$

Theorem 4.3.3. *Suppose that condition (4.3.18) holds and the disease-induced death rate δ_h is large enough in the sense that the rate at which people die due to the disease satisfies the inequality*

$$\delta_h > \delta_h^{crit.} = \frac{\sigma_v \beta_{vh} B}{f_v^*}, \tag{4.3.19}$$

then system (4.3.1) has at least one endemic equilibrium when $R_0 < 1$.

Proof. At the endemic equilibrium we have

$$\begin{cases} c^{**} = \dfrac{c^*}{1 - DI_h^*}, \\ f_h^{**} = f_h^* - AI_h^*, \\ M_1^{**} = M_1^* - AI_h^*, \\ M_2^{**} = M_2^* - AI_h^*, \\ M_3^{**} = M_3^* - AI_h^*, \\ M_4^{**} = M_4^*, \end{cases} \tag{4.3.20}$$

where

$$A = \frac{\mu_{2h}\delta_h}{B}, \quad D = \frac{\sigma_h \delta_h}{B(\sigma_v N_v^* + \sigma_h N_h^*)}, \quad E = \left(\beta_{vh} + \frac{\gamma_h \tilde{\beta}_{vh}}{M_3^*}\right). \tag{4.3.21}$$

Using

$$\nu_h E_h^{**} - M_2^{**} I_h^* = 0 \tag{4.3.22}$$

we obtain I_h^* as a root of the algebraic equation of the form

$$\mathcal{A}_7 I_h^{*7} + \mathcal{A}_6 I_h^{*6} + \mathcal{A}_5 I_h^{*5} + \mathcal{A}_4 I_h^{*4} + \mathcal{A}_3 I_h^{*3} + \mathcal{A}_2 I_h^{*2} + \mathcal{A}_1 I_h^* + \mathcal{A}_0 = 0. \tag{4.3.23}$$

Explicit expressions of the coefficients, $\mathcal{A}_0, \cdots, \mathcal{A}_7$ can be derived in terms of the parameters of the model. Specifically for $\mathcal{A}_0$ and $\mathcal{A}_7$, we have

$$\begin{cases} \mathcal{A}_0 = M_1^* M_2^* M_3^{*2} M_4^* f_h^* f_v^* (R_0^2 - 1) \\ \mathcal{A}_7 = A^5 f_v^* M_4^* D^2 - c^* M_4^* A^5 D \beta_{vh} = A^5 M_4^* D (D f_v^* - c^* \beta_{vh}) \end{cases} \tag{4.3.24}$$

Using Eq. (4.3.19) and Eq. (4.3.21) it is easy to see that $\mathcal{A}_7 > 0$. Let $R_0 < 1$. Then clearly $\mathcal{A}_0 < 0$. Hence by Descartes' rule of signs Eq. (4.3.23) has at least one positive root (see Murray [67], Wang [80]). That is, for $R_0 < 1$ the stable DFE co-exists with an endemic equilibrium which approaches DFE as $R_0 \to 1$. □

Remark 4.3.4. *The biological meaning of the inequality (4.3.19) can be obtained by addressing the research question no.(i) in the concluding Chapter. That is to find a critical value R_0^c of the basic reproduction number R_0 such that the disease-free equilibrium coexists with an endemic equilibrium whenever $R_0 \in (R_0^c, 1)$ for $\delta_h > 0$.*

4.3.3 Application of Theorem 3.4.1

We investigate the nature of the bifurcation by using Theorem 3.4.1. We consider $c(N_h, N_v)$ in Eq. (4.3.3) and evaluate its equation at the disease-free equilibrium to obtain

$$c^* = c(N_h^*, N_v^*) = \frac{\sigma_v \sigma_h}{\sigma_h N_h^* + \sigma_v N_v^*} \tag{4.3.25}$$

as defined in Eq. (4.3.7). Let

$$\zeta_1 = \left(\frac{M_1^* M_2^* M_4^* f_v^*}{\beta_{hv} \nu_h \nu_v N_h^* N_v^* \left(\beta_{vh} + \dfrac{\tilde{\beta}_{vh} \gamma_h}{M_3^*} \right)} \right)^{1/2}. \tag{4.3.26}$$

If $c^* < \zeta_1$ the DFE is locally asymptotically stable and unstable when $c^* > \zeta_1$. Thus, c^* is a bifurcation parameter. The parameter c^* is introduced in the system (4.3.1) as follows:

$$c(N_h, N_v) = \frac{\sigma_v \sigma_h}{\sigma_h N_h + \sigma_v N_v} \times \frac{\sigma_h N_h^* + \sigma_v N_v^*}{\sigma_h N_h^* + \sigma_v N_v^*} = c^* \frac{\sigma_h N_h^* + \sigma_v N_v^*}{\sigma_h N_h + \sigma_v N_v}. \tag{4.3.27}$$

Remark 4.3.5. *In the sequel, the constant ratio of* $\frac{\sigma_h}{\sigma_v} = 30$ *in Chitnis et al. [23] will be used.*

In order to illustrate the role of the disease-induced death rate δ_h we consider Eq. (4.3.1) in its equivalent form where the equations of the populations of susceptible individuals S_h and S_v are replaced by the equations in (4.3.4) of the total populations N_h and N_v respectively. Henceforth, our calculations will involve a lot of second-order partial derivatives. For convenience we introduce the following notation

$$\begin{cases} \mathbf{z} = (z_1, z_2, z_3, z_4, z_5, z_6, z_7)^T = (N_h, E_h, I_h, R_h, N_v, E_v, I_v)^T \\ \dot{\mathbf{z}} = (H_1, H_2, H_3, H_4, H_5, H_6, H_7)^T = H(\mathbf{z}, c^*) \end{cases} \tag{4.3.28}$$

The Jacobian matrix of system (4.3.28) evaluated at DFE is given by

$$J = \begin{pmatrix} -B & 0 & -\delta_h & 0 & 0 & 0 & 0 \\ 0 & -M_1^* & 0 & 0 & 0 & 0 & c^*\beta_{hv}N_h^* \\ 0 & \nu_h & -M_2^* & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma_h & -M_3^* & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -(f_v^* - \mu_{1v}) & 0 & 0 \\ 0 & 0 & c^*\beta_{vh}N_v^* & c^*\tilde{\beta}_{vh}N_v^* & 0 & -M_4^* & 0 \\ 0 & 0 & 0 & 0 & 0 & \nu_v & -f_v^* \end{pmatrix} \tag{4.3.29}$$

The characteristic polynomial of the matrix J is given by

$$\begin{aligned} |J - \lambda I| \;=\; & (\lambda + B)(\lambda + (f_v^* - \mu_{1v}))\Big\{\lambda^5 + \lambda^4(f_v^* + B_0) + \lambda^3(f_v^*B_0 + B_1) + \lambda^2(f_v^*B_1 + B_2) \\ & + \lambda^1(f_v^*B_2 + M_1^*M_2^*M_3^*M_4^*) - \lambda^1 c^{*2}\beta_{hv}\beta_{vh}N_h^*N_v^*\nu_v\nu_h + M_1^*M_2^*M_3^*M_4^*f_v^*(1 - R_0^2)\Big\} = 0, \end{aligned} \tag{4.3.30}$$

where

$$\begin{cases} B_0 = (M_3^* + M_4^*) + (M_2^* + M_1^*), \;\; B_1 = M_1^* M_2^* + (M_1^* + M_2^*)(M_3^* + M_4^*) + M_4^* M_3^*, \\ B_2 = M_4^* M_3^* (M_1^* + M_2^*) + M_1^* M_2^* (M_3^* + M_4^*). \end{cases} \tag{4.3.31}$$

We write λ^1 with a superscript to distinguish it in Eq. (4.3.30) for referencing purposes. In what follows we show that the coefficient of λ^1 is positive when $R_0 \leq 1$. Let $R_0 \leq 1$, then

$$c^{*2}\beta_{hv}\nu_h\nu_v N_h^* N_v^* \left(\beta_{vh} + \frac{\tilde{\beta}_{vh}\gamma_h}{M_3^*}\right) \leq M_1^* M_2^* M_4^* f_v^* \Rightarrow c^{*2}\nu_h\nu_v N_h^* N_v^* \beta_{hv}\beta_{vh} < M_1^* M_2^* M_4^* f_v^* < f_v^* B_2. \tag{4.3.32}$$

Thus,

$$(f_v^* B_2 + M_1^* M_2^* M_3^* M_4^*) - c^{*2}\nu_h\nu_v N_h^* N_v^* \beta_{hv}\beta_{vh} > 0. \tag{4.3.33}$$

Since it assumed in Chitnis et al. [23] that in the absence of the disease, the density independent death rate of the mosquitoes μ_{1v} is less than the per capita birth rate f_v^*, i.e. $f_v^* > \mu_{1v}$, it is easy to see that $\lambda = -B$ and $\lambda = -(f_v^* - \mu_{1v})$ are negative eigenvalues of J. We now use matrix theory in Farina and Rinaldi [34], Mitkowski [66] to investigate the eigenvalues of the following irreducible Metzler matrix

$$J^* = \begin{pmatrix} -M_1^* & 0 & 0 & 0 & c^*\beta_{hv}N_h^* \\ \nu_h & -M_2^* & 0 & 0 & 0 \\ 0 & \gamma_h & -M_3^* & 0 & 0 \\ 0 & c^*\beta_{vh}N_v^* & c^*\tilde{\beta}_{vh}N_v^* & -M_4^* & 0 \\ 0 & 0 & 0 & \nu_v & -f_v^* \end{pmatrix}. \tag{4.3.34}$$

When $c^* = \zeta_1$ we have $R_0 = 1$, which means that the disease progression makes a transition or bifurcates, from a non-invasive state to an invasive one, in the sense that one infectious individual can infect exactly one susceptible individual. At this point, the matrix J^* admits

a simple zero eigenvalue and all the other eigenvalues have negative real parts by Lemmas 4.3.6 and 4.3.7 below as stated in Mitkowski [66].

Lemma 4.3.6. *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 $\lambda_{\max} = \max Re(\lambda_i)\ \forall\ i = 1, 2, \cdots, n$.*

Lemma 4.3.7. *Let $A = [a_{ij}]$ be a Metzler matrix, i.e. $a_{ij} \geq 0$, $i \neq j$. 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 of Eq. (3.4.2) in Theorem 3.4.1 are computed by letting

$$\mathbf{w} = (w_1, w_2, w_3, w_4, w_5, w_6, w_7)^T \text{ and } \mathbf{v} = (v_1, v_2, v_3, v_4, v_5, v_6, v_7) \tag{4.3.35}$$

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

$$\begin{cases} w_1 = -\dfrac{\delta_h \nu_h c^* \beta_{hv} N_h^*}{B M_2^* M_1^*} w_7, \quad w_2 = \dfrac{c^* \beta_{hv} N_h^*}{M_1^*} w_7, \quad w_3 = \dfrac{\nu_h c^* \beta_{hv} N_h^*}{M_2^* M_1^*} w_7, \quad w_4 = \dfrac{\gamma_h \nu_h c^* \beta_{hv} N_h^*}{M_3^* M_2^* M_1^*} w_7, \\ w_5 = 0, \quad w_6 = \dfrac{f_v^*}{\nu_v} w_7, \quad w_7 = w_7 > 0. \end{cases} \tag{4.3.36}$$

$$\begin{cases} v_1 = 0, \quad v_2 = \dfrac{f_v^*}{c^* \beta_{hv} N_h^*} v_7, \quad v_3 = \dfrac{M_1^* f_v^*}{\nu_h c^* \beta_{hv} N_h^*} v_7, \quad v_4 = \dfrac{c^* \tilde{\beta}_{vh} N_v^* \nu_v}{M_3^* M_4^*} v_7, \quad v_5 = 0, \\ v_6 = \dfrac{\nu_v}{M_4^*} v_7, \quad v_7 = v_7 > 0. \end{cases} \tag{4.3.37}$$

Remark 4.3.8. *In the literature (see for instance Castillo-Chavez and Song [16] and Hassan et al. [44]), the eigenvectors $\mathbf{w}$ and $\mathbf{v}$ are normalised such that $\mathbf{w^T} \cdot \mathbf{v} = \mathbf{1}$. However, this requirement has no impact on the calculations done above.*

We ignore the partial derivatives of H_1 and H_5 because $v_1 = 0 = v_5$. The non-zero second-order partial derivatives are the following:

$$\begin{cases} \frac{\partial^2 H_2}{\partial z_1 \partial z_7} = -\frac{30c^*\beta_{hv}N_h^*}{30N_h^* + N_v^*} + c^*\beta_{hv}, \quad \frac{\partial^2 H_2}{\partial z_2 \partial z_7} = -c^*\beta_{hv}, \quad \frac{\partial^2 H_2}{\partial z_3 \partial z_7} = -c^*\beta_{hv}, \\ \frac{\partial^2 H_2}{\partial z_4 \partial z_7} = -c^*\beta_{hv}, \quad \frac{\partial^2 H_2}{\partial z_1 \partial z_2} = -\mu_{2h}, \quad \frac{\partial^2 H_3}{\partial z_1 \partial z_3} = -\mu_{2h}, \quad \frac{\partial^2 H_4}{\partial z_1 \partial z_4} = -\mu_{2h}, \\ \frac{\partial^2 H_6}{\partial z_4 \partial z_7} = -c^*\tilde{\beta}_{vh}, \quad \frac{\partial^2 H_6}{\partial z_3 \partial z_1} = -\frac{30c^*\beta_{vh}N_v^*}{30N_h^* + Nv*}, \quad \frac{\partial^2 H_6}{\partial z_3 \partial z_6} = -c^*\beta_{vh}, \\ \frac{\partial^2 H_6}{\partial z_4 \partial z_1} = -\frac{30c^*\tilde{\beta}_{vh}N_v^*}{30N_h^* + N_v^*}, \quad \frac{\partial^2 H_6}{\partial z_3 \partial z_7} = -c^*\beta_{vh}, \quad \frac{\partial^2 H_6}{\partial z_4 \partial z_6} = -c^*\tilde{\beta}_{vh}, \\ \frac{\partial^2 H_2}{\partial c^* \partial z_7} = N_h^*\beta_{hv}, \quad \frac{\partial^2 H_6}{\partial z_3 \partial c^*} = N_v^*\beta_{vh}, \quad \frac{\partial^2 H_6}{\partial z_4 \partial c^*} = N_v^*\tilde{\beta}_{vh}. \end{cases} \tag{4.3.38}$$

From the expressions in (4.3.36)-(4.3.38) we obtain

$$\begin{aligned} a &= 2v_7 w_7^2 \Bigg\{ \frac{30c^*\beta_{hv}N_h^* f_v^* \delta_h \nu_h}{(30N_h^* + N_v^*)BM_2^* M_1^*}(R_0^2 + 1) + \frac{\mu_{2h} f_v^* \delta_h \nu_h c^* \beta_{hv} N_h^*}{BM_2^{*2} M_1^{*2}}(M_1^* + M_2^*) \\ &+ \frac{\delta_h c^{*3} \tilde{\beta}_{vh} N_v^* \nu_v \nu_h^2 \beta_{hv}^2 N_h^{*2} \gamma_h \mu_{2h}}{BM_4^* M_3^{*2} M_2^{*2} M_1^{*2}} - \frac{f_v^* c^* \beta_{hv} \delta_h \nu_h}{BM_2^* M_1^*} - \frac{f_v^* c^* \beta_{hv}}{M_1^*}\left(1 + \frac{\nu_h}{M_2^*} + \frac{\gamma_h \nu_h}{M_3^* M_2^*}\right) \\ &- \frac{\nu_h c^{*2} \beta_{hv} N_h^*}{M_2^* M_1^*}\left(\beta_{vh} + \frac{\gamma_h \tilde{\beta}_{vh}}{M_3^*}\right) \Bigg\} \\ &= 2v_7 w_7^2 \Phi^* (\Pi^* - 1) \end{aligned} \tag{4.3.39}$$

and

$$b = \frac{2f_v^*}{c^*} v_7 w_7 > 0 \tag{4.3.40}$$

where

$$\Phi^* = \frac{f_v^* c^* \beta_{hv} \delta_h \nu_h}{BM_2^* M_1^*} + \frac{f_v^* c^* \beta_{hv}}{M_1^*}\left(1 + \frac{\nu_h}{M_2^*} + \frac{\gamma_h \nu_h}{M_3^* M_2^*}\right) + \frac{\nu_h c^{*2} \beta_{hv} N_h^*}{M_2^* M_1^*}\left(\beta_{vh} + \frac{\gamma_h \tilde{\beta}_{vh}}{M_3^*}\right),$$

$$\Pi^* = \frac{\frac{30c^*\beta_{hv}N_h^* f_v^* \delta_h \nu_h}{(30N_h^* + N_v^*)BM_2^* M_1^*}(R_0^2 + 1) + \frac{\mu_{2h} f_v^* \delta_h \nu_h c^* \beta_{hv} N_h^*}{BM_2^{*2} M_1^{*2}}(M_1^* + M_2^*) + \chi}{\Phi^*} \tag{4.3.41}$$

and

$$\chi = \frac{\delta_h c^{*3} \tilde{\beta}_{vh} N_v^* \nu_v \nu_h^2 \beta_{hv}^2 N_h^{*2} \gamma_h \mu_{2h}}{B M_4^* M_3^{*2} M_2^{*2} M_1^{*2}}.$$

Then using Theorem 3.4.1, we obtain the following characterisation of the bifurcation at $R_0 = 1$.

Theorem 4.3.9. *The malaria model (4.3.1) exhibits a backward bifurcation at $R_0 = 1$ if $\Pi^* > 1$ as per Eq. (4.3.39) and a forward bifurcation if $\Pi^* < 1$.*

Remark 4.3.10. *It should be noted that when there is no one dying due to the disease, i.e. $\delta_h = 0$, we have $\Pi^* = 0$ and hence, the bifurcation at $R_0 = 1$ is supercritical. Further, it is easy to see that Π^* is an increasing function of δ_h at least in some neighbourhood of $\delta_h = 0$.*

CHAPTER 5. NONSTANDARD FINITE DIFFERENCE SCHEMES

5.1 Introduction

The literature on the relevance of the Nonstandard Finite Difference (NSFD) schemes in gaining useful insights on the solutions of real world models of differential equations was reviewed in Chapter 1. The NSFD schemes proposed in this Chapter are aimed at preserving the backward bifurcation property of the previously discussed continuous models of differential equations for the transmission of diseases. This is a challenge because the disease-free equilibrium is nonhyperbolic and thus the widely used concept and technique of elementary stable NSFD schemes does not apply. However, observing that epidemiological models have a specific decomposition into productive and destructive terms, we use Mickens's rules in an innovative manner to construct reliable NSFD schemes in Section 5.3. This approach is successfully applied to the SIS model in Section 5.4 and the malaria model in Section 5.5 since the resulting NSFD schemes are dynamically consistent with respect to the backward bifurcation property from both a theoretical and numerical perspective in Section 5.6. Of course, the Chapter starts with preliminaries on NSFD schemes in Section 5.2. As mentioned earlier, the results on NSFD schemes for the SIS and malaria models presented in this Chapter are published in Anguelov et al. [9].

5.2 Preliminaries

In general, the continuous system

$$\frac{dz}{dt} = H(z), \;\; z(0) \in \mathbb{R}^n, \tag{5.2.1}$$

where $H : D \subseteq \mathbb{R}^n \to \mathbb{R}^n$ is sufficiently smooth on a compact set D, cannot be completely solved by analytic techniques. Consequently, numerical methods are of fundamental importance in gaining more useful insights into the solution of the differential equation.

We consider a difference equation

$$D_{\Delta t} z^k = G_{\Delta t}(H, z^k), \tag{5.2.2}$$

which gives rise to a sequence $\{z^k\}_{k=0}^{\infty}$ of approximations to the solution $z(t)$ of (5.2.1) at the discrete time steps $\{t_k = k\Delta t\}_{k=0}^{\infty}$, where $\Delta t \equiv h$ is the step size:

$$z(t_k) \approx z^k. \tag{5.2.3}$$

It is implicitly understood that $D_{\Delta t} z^k$ is an approximation of the derivatives $\dfrac{dz}{dt}$, while $G_{\Delta t}(H, z^k)$ approximates the function $H(z)$. It is also important to note that the algorithm in (5.2.2) permits one to find the discrete solution z^{k+1} at the time t_{k+1} assuming that the discrete solution z^k is known at the time t_k.

We start with some general concepts about the numerical method (5.2.2). We fix a time t^* which can be represented in the form

$$t^* = k^* \Delta t = t_{k^*} \tag{5.2.4}$$

for different values of k^* and Δt.

Definition 5.2.1. *(Lambert [54]) The difference scheme (5.2.2) of a well-posed initial value problem (5.2.1) is said to be convergent if, we have that*

$$\lim_{\substack{\Delta t \to 0 \\ t^* = k^* \Delta t}} z^k = z(t^*) \tag{5.2.5}$$

holds for all $t^* \geq 0$ *and for all solutions* $\{z^k\}$ *of the difference equation (5.2.2).*

As mentioned earlier, the exact solution is not always known. It is therefore difficult to check the convergence based on Definition 5.2.1. To overcome this difficulty, we consider three additional concepts.

Definition 5.2.2. *(Lambert [54]) The local truncation error,* R_{k+1}*, of the numerical method (5.2.2) is the amount by which the exact solution* $z(t)$ *fails to satisfy the relation (5.2.2). That is*

$$R_{k+1} := D_{\Delta t} z(t_k) - G_{\Delta t}(H, z(t_k)). \tag{5.2.6}$$

Definition 5.2.3. *(Lambert [54]) The method (5.2.2) is said to be consistent with the differential equation if, for all initial value problems, the local truncation error* R_{k+1} *satisfies*

$$\lim_{\Delta t \to 0} R_{k+1} = 0. \tag{5.2.7}$$

Definition 5.2.4. *(Lambert [54]) Let* $\{\delta^k\}_{k=0}^{\infty}$ *and* $\{\tilde{\delta}^k\}_{k=0}^{\infty}$ *be any two perturbations of the scheme (5.2.2), and let* $\{Z^k\}_{k=0}^{\infty}$ *and* $\{\tilde{Z}^k\}_{k=0}^{\infty}$ *be the resulting solutions. If there exist constants* S *and* Δt_0 *such that for all* $\Delta t \in (0, \Delta t_0]$*, we have*

$$\|Z^k - \tilde{Z}^k\| \leq S\epsilon \ \text{ whenever } \ \|\delta^k - \tilde{\delta}^k\| \leq \epsilon \ \text{ and } \ k \geq 0, \tag{5.2.8}$$

then the scheme (5.2.2) is said to be zero-stable.

The challenge mentioned earlier about checking the convergence of the scheme is addressed by the following fundamental result.

Theorem 5.2.5. *Assume that Eq. (5.2.1) is well-posed. Then the difference scheme (5.2.2) is convergent if and only if it is both consistent with the differential equation and zero-stable.*

Convergence of the scheme is important. A numerical method that is not convergent is not useful. In addition to convergence, we want schemes which replicate the dynamics of the continuous systems. A typical example is the so called exact scheme. For the exact scheme, the local truncation error is zero.

Definition 5.2.6. *(Mickens [61]) A numerical method (5.2.2) that approximates (5.2.1) is called an exact scheme whenever the difference equation (5.2.2) and the differential equation (5.2.1) have the same general solutions at the discrete time $t = t_k$. In particular, with $z(t)$ being the solution of the initial value problem (5.2.1) we have $z^k = z(t_k)$.*

Definition 5.2.7. *(Anguelov and Lubuma [3]) The difference equation (5.2.2) is called a nonstandard finite difference (NSFD) scheme if at least one of the following conditions is satisfied:*

(i) In the first order discrete derivative $D_{\Delta t} z^k \approx \dot{z}(t_k)$, the classical denominator $h = \Delta t$ is replaced by a nonnegative function $\phi : (0, \infty) \to (0, \infty)$ satisfying the asymptotic relation (Rule 2)

$$\phi(\Delta t) = \Delta t + \mathcal{O}\left([\Delta t]^2\right). \tag{5.2.9}$$

(ii) In the expression $G_{\Delta t}(H, z^k)$, the nonlinear terms are approximated in a nonlocal manner. For example a term like $z^2(t_k)$ is approximated by $z^{k+1} z^k$ instead of z^{k^2} (Rule 3).

Remark 5.2.8. *Note that Definition 5.2.7 retains only two rules out of the five rules by Mickens [62] to construct NSFD schemes. For convenience, the other rules are stated below.*

(i) The order of the discrete derivative should be equal to the order of the corresponding derivative of the differential equation. (Rule 1)

(ii) The special condition that holds for the solution of differential equations should also hold for the solutions of the finite difference scheme. (Rule 4)

(iii) The scheme should not introduce extraneous or spurious solutions. (Rule 5)

The power of the NSFD schemes over standard numerical schemes is their potential to be dynamically consistent with respect to the continuous model and is clarified in the next definition.

Definition 5.2.9. *(Anguelov and Lubuma [3]) Assume that the solution of (5.2.1) satisfies a property P. The difference equation (5.2.2) is said to be qualitatively stable or dynamically consistent with respect to property P if for all step sizes $\Delta t > 0$, the discrete solution for (5.2.2) satisfies P.*

Below, we describe the minimal desirable property P for any scheme.

Definition 5.2.10. *(Anguelov and Lubuma [3]) A difference scheme (5.2.2) that approximates the differential equation (5.2.1) is said to be elementary stable if, for any value of the step size Δt, its fixed-points are exactly the equilibrium points of the differential system (5.2.1) and these fixed-points for the difference scheme have the same linear stability or instability properties as the differential system.*

To illustrate the concept of an elementary stable NSFD scheme, we describe a general result that is presented in Kama [50]. The dynamics of the system (5.2.1) will be captured by a fixed nonzero number given by

$$Q \geq \max\left\{\frac{|\lambda|^2}{2|Re\lambda|},\ \lambda \in \Omega,\ Re\lambda \neq 0\right\} \text{ where } \Omega = \bigcup\left\{\sigma\left(J|_{z=z^*}\right),\ H(z^*) = 0\right\}. \quad (5.2.10)$$

Note that the set Ω of all eigenvalues of the Jacobian matrices J is supposed to be finite. We define a nonnegative function $\phi(\Delta t)$ satisfying the asymptotic relation in Definition 5.2.7 as

$$\phi(\Delta t) = \frac{\varphi(q\Delta t)}{q} \text{ where } 0 < \varphi < 1 \text{ and } q \geq Q. \quad (5.2.11)$$

We then consider the nonstandard forward Euler scheme

$$\frac{z^{k+1} - z^k}{\phi} = H(z^k), \quad (5.2.12)$$

as well as the nonstandard backward Euler scheme

$$\frac{z^{k+1} - z^k}{\phi} = H(z^{k+1}), \quad (5.2.13)$$

as approximations. We are now in a position to state the following result.

Theorem 5.2.11. *(Kama [50]) Assume that all equilibrium points of (5.2.1) are hyperbolic. Then the NSFD schemes (5.2.12) - (5.2.13) with ϕ given in Eq. (5.2.11) are elementary stable.*

Remark 5.2.12. *As pointed out earlier Theorem 5.2.11 is not valid when the equilibrium points are not hyperbolic.*

5.3 NSFD scheme for a class of epidemiological models

As reported by Wood et al. [86] most epidemiological models contain a productive and a destructive component. In this thesis we consider the following class of such models:

$$\frac{dz_i}{dt} = H_i(z(t)) = g_i(z(t)) - z_i(t)r_i(\eta) - z_i(t)p_i(z(t)), \quad i = 1, 2, \cdots, n, \tag{5.3.1}$$

where $g_i, p_i : \mathbb{R}^n \to \mathbb{R}_+$ are sufficiently smooth functions, $g_i \geq 0$, $r_i \geq 0$, and η is a set of parameters. We assume that the system (5.3.1) has an equilibrium point z^* such that $p_i(z^*) = 0$.

In the spirit of the elementary stable NSFD schemes (5.2.12) and (5.2.13), we introduce for (5.3.1) a NSFD scheme of the form:

$$\frac{z_i^{k+1} - z_i^k}{\phi} = g_i(z^k) - z_i^k r_i(\eta) - z_i^{k+1} p_i(z^k). \tag{5.3.2}$$

In order to preserve positivity and elementary stability of the dynamical system (5.3.1), the complex denominator function ϕ in (5.2.11) is chosen in a more explicit and specific manner as follows:

(i) The number q captures the parameters of the model and the eigenvalues $\lambda_1, \cdots, \lambda_n$ of the Jacobian matrix of H at $z = z^*$:

$$q \geq \max\Big\{r_i(\eta) : \ i = 1, \cdots, n\Big\} \cup \Big\{\frac{|\lambda_i|^2}{2|Re\lambda_i|} : \ i = 1, \cdots, n \ \text{ and } \ Re\lambda_i \neq 0\Big\}. \tag{5.3.3}$$

(ii) The function $0 < \varphi < 1$ is taken such that

$$\phi = \phi(\Delta t) = \frac{1 - e^{-q\Delta t}}{q}. \tag{5.3.4}$$

Then we have

$$\phi(\Delta t) < \frac{1}{q} \leq \frac{1}{r_i(\eta)} : \ i = 1, \cdots, n \tag{5.3.5}$$

and

$$\phi(\Delta t) < \frac{1}{q} \leq \frac{2|Re\lambda_i|}{|\lambda_i|^2} : \ i = 1, \cdots, n, \ \ Re\lambda_i \neq 0. \tag{5.3.6}$$

Eq. (5.3.2) can be written explicitly as:

$$z_i^{k+1} = \frac{(1 - \phi r_i(\eta))z_i^k + \phi g_i(z^k)}{1 + \phi p_i(z^k)}. \tag{5.3.7}$$

Using Eq. (5.3.5) we have the following result.

Theorem 5.3.1. *The NSFD scheme (5.3.2), with ϕ given in (5.3.4), is dynamically consistent with respect to the positivity property of the continuous model.*

As observed in the proof of Theorem 5.2.11, we have the following result.

Proposition 5.3.2. *(i) The point z^* is an equilibrium point of the model (5.3.1) if and only if z^* is a fixed-point of the NSFD scheme (5.3.7).*

(ii) The Jacobian matrix J for the model (5.3.1) evaluated at z^* is linked to the Jacobian matrix J_{NS} for the NSFD scheme evaluated at z^* through the formula

$$J_{NS} = I_n + \phi J. \tag{5.3.8}$$

(iii) If z^* is hyperbolic, then z^* is asymptotically stable for (5.3.1) if and only if it is asymptotically stable for (5.3.7).

Proof. (i) The statement follows from the equivalence, for every $i = 1, \cdots, n$, of the following equations

$$\begin{aligned} z_i^* &= \frac{(1-\phi r_i(\eta))z_i^* + \phi g_i(z^*)}{1+\phi p_i(z^*)}, \\ z_i^* + z_i^*\phi p_i(z^*) &= z_i^* - \phi r_i(\eta) z_i^* + \phi g_i(z^*), \\ \phi(g_i(z^*) - z_i^* p_i(z^*) - r_i(\eta) z_i^*) &= 0, \\ H_i(z^*) &= 0. \end{aligned} \tag{5.3.9}$$

(ii) Let J and J_{NS} be the associated Jacobian matrices of the differential system (5.3.1) and the NSFD scheme (5.3.7) respectively, evaluated at the equilibrium point z^* with λ and λ_{NS} as their respective eigenvalues.

Then,

$$\begin{aligned} J &= \left(\left(\frac{\partial g_i}{\partial z_j} \right) \Big|_{z^*} - (I_n r_i) \Big|_{z^*} - \left(z_i \frac{\partial p_i}{\partial z_j} \right) \Big|_{z^*} \right)_{1 \le i,j \le n} \\ &= \left(\left(\frac{\partial g_i}{\partial z_j} \right) \Big|_{z^*} - \left(z_i \frac{\partial p_i}{\partial z_j} \right) \Big|_{z^*} + diag(-r_i) \right)_{1 \le i,j \le n} \end{aligned} \tag{5.3.10}$$

and

$$\begin{aligned} J_{NS} &= \left(\left(\phi \frac{\partial g_i}{\partial z_j^k} \right) \Big|_{z^*} - (I_n - \phi r_i) \Big|_{z^*} - \left(\phi z_i^k \frac{\partial p_i}{\partial z_j^k} \right) \Big|_{z^*} \right)_{1 \le i,j \le n} \\ &= I_n + \phi \left(\left(\frac{\partial g_i}{\partial z_j^k} \right) \Big|_{z^*} - \left(z_i^k \frac{\partial p_i}{\partial z_j^k} \right) \Big|_{z^*} + diag(-r_i) \right)_{1 \le i,j \le n} \\ &= I_n + \phi J. \end{aligned} \tag{5.3.11}$$

Eq. (5.3.11) can be written as

$$J = \frac{J_{NS} - I_n}{\phi}. \tag{5.3.12}$$

Thus, the eigenvalues of the matrices J and J_{NS} are related by the the following equation:

$$\begin{aligned} det(J-\lambda_i I_n) &= det\left(\frac{J_{NS}-I_n}{\phi}-\lambda_i I_n\right) \\ &= \left(\frac{1}{\phi}\right)^n det\left(J_{NS}-(1+\lambda_i\phi)I_n\right). \end{aligned} \tag{5.3.13}$$

(iii) Let z^* be an asymptotically stable equilibrium of (5.3.1). Then, considering that it is hyperbolic, we have $Re\lambda_i < 0,\ i = 1, \cdots, n$. It follows from (ii) that the eigenvalues of J_{NS} are $\lambda_{NS,i} = 1 + \phi\lambda_i,\ i = 1, \cdots, n$. We have

$$\begin{aligned} |\lambda_{NS,i}|^2 &= (1+\phi\lambda_i)(1+\phi\overline{\lambda_i}) \\ &= \phi^2|\lambda_i|^2 - 2\phi|Re\lambda_i| + 1. \end{aligned} \tag{5.3.14}$$

It follows from (5.3.6) that $|\lambda_{NS,i}| < 1$. Let z^* be an unstable fixed-point of (5.3.1). Hence there exists an eigenvalue λ_j such that $Re\lambda_j > 0$. Then

$$\begin{aligned} |\lambda_{NS,j}|^2 &= (1+\phi\lambda_j)(1+\phi\overline{\lambda_j}), \\ &= \left[(1+\phi Re\lambda_j)+i\phi Im\lambda_j\right]\left[(1+\phi Re\lambda_j)-i\phi Im\lambda_j\right], \\ &= (1+\phi Re\lambda_j)^2+\phi^2(Im\lambda_j)^2, \\ &= 1+2\phi Re\lambda_j+\phi^2(Re\lambda_j)^2+\phi^2(Im\lambda_j)^2, \\ &= 1+2\phi Re\lambda_j+\phi^2[(Re\lambda_j)^2+(Im\lambda_j)^2], \\ &= 1+2\phi|Re\lambda_j|+\phi^2|\lambda_j|^2 > 1. \end{aligned} \tag{5.3.15}$$

Therefore, z^* is unstable for (5.3.7).

□

Remark 5.3.3. *It cannot be claimed that the scheme (5.3.7) fully replicates the properties of z^* when it is not hyperbolic. However, we have the following property*

$$\lambda_i = 0 \ \Leftrightarrow\ \lambda_{NS,i} = 1, \tag{5.3.16}$$

that is if the system (5.3.1) is in the bifurcational state discussed in the theorem by Castillo-Chavez and Song [16], so is the scheme (5.3.7). The dynamic consistency with respect to backward bifurcation for specific models is discussed in the following Subsections.

Corollary 5.3.4. *Let the continuous dynamical system (5.3.1) have a nonhyperbolic equilibrium-point z^* such that $\lambda_i = 0$ for some i (i.e. a simple eigenvalue of the matrix J). If this differential system undergoes a bifurcation at the point z^*, then the NSFD scheme (5.3.7) will undergo a bifurcation at the fixed-point z^*.*

Lemma 5.3.5. *(Casulli and Greenspan [19]) If the sequence $\{e_i\}$, $i = 0, 1, 2, \cdots, n$ satisfies*

$$\|e_{i+1}\| \leq \beta\|e_i\| + \alpha, \quad i = 0, 1, 2, \cdots, n-1,$$

where β and α are nonnegative constants and $\beta \neq 1$, then

$$\|e_i\| \leq \beta^i\|e_0\| + \frac{\beta^i - 1}{\beta - 1}\alpha, \quad i = 1, 2, \cdots, n.$$

Theorem 5.3.6. *The NSFD scheme (5.3.7) is convergent and of order one.*

Proof. This theorem can be proved by showing that the local truncation error R_{k+1} in Definition 5.2.2 is such that $R_{k+1} = \mathcal{O}(\Delta t)$, combined with Theorem 5.2.6. However, given the specific structure of the model (5.3.1) and of the NSFD scheme (5.3.7), we provide below a direct proof inspired by Lambert [54]. Let us write (5.3.7) in the form:

$$z_i^{k+1} = G_i(\phi, z^k) \tag{5.3.17}$$

where $G_i(\phi, z^k)$ is given by the right hand side of (5.3.7). We fix t^* which can be written as

$$t^* = k\Delta t = t_k \tag{5.3.18}$$

for different values of k and different Δt. Our task is to show that

$$\|z^k - z(t^*)\|_\infty := \max_i |z_i^k - z_i(t^*)| \leq M\Delta t, \quad M > 0, \tag{5.3.19}$$

where M represents various constants linked to the fact that the right-hand side of (5.3.1) is Lipschitz on the compact set in which the model is considered. Expand $z_i(t_{k+1}) = z_i(t_k + \Delta t)$

in a Taylor series at t_k to obtain:

$$z_i(t_k+\Delta t) = z_i(t_k) + \Delta t \frac{dz_i}{dt}(t_k) + \frac{1}{2}(\Delta t)^2 \frac{d^2 z_i}{dt^2}(\xi) \text{ where } \xi \in (t_k, t_{k+1}), \tag{5.3.20}$$

which in view of (5.3.1) gives

$$z_i(t_k+\Delta t) \leq z_i(t_k) + \Delta t(g_i(z(t_k)) - z_i(t_k) r_i(\eta) - z_i(t_k) p_i(z(t_k))) + \frac{(\Delta t)^2}{2} M. \tag{5.3.21}$$

Likewise for the discrete solution, we have

$$z_i^{k+1} = z_i^k + \phi \frac{\partial z_i^{k+1}}{\partial \phi}\Big|_{\phi=0} + \frac{1}{2}(\phi)^2 \frac{\partial^2 z_i^{k+1}}{\partial \phi^2}\Big|_{\phi=0} + \mathcal{O}(\phi)^3. \tag{5.3.22}$$

Evaluating the derivatives and using the asymptotic relation (5.2.9), we have

$$z_i^{k+1} \leq z_i^k + \Delta t \left(g_i(z^k) - r_i z_i^k - z_i^k p_i(z^k)\right) + \frac{(\Delta t)^2}{2} M. \tag{5.3.23}$$

From (5.3.21) and (5.3.23), we have

$$\begin{aligned} z_i^{k+1} - z_i(t_{k+1}) \leq\ & (z_i^k - z_i(t_k)) + \Delta t \left[g_i(z^k) - r_i z_i^k - z_i^k p_i(z^k) - g_i(z(t_k)) + r_i z_i(t_k) + z_i(t_k) p_i(z^k)\right] \\ & + \frac{(\Delta t)^2}{2} M. \end{aligned} \tag{5.3.24}$$

Let $e_k^i = z_i^k - z_i(t_k)$, then

$$\begin{aligned} \|e_{k+1}^i\| &\leq \|z_i^k - z_i(t_k)\| + \Delta t \|H_i(z^k) - H_i(z(t_k))\| + \frac{(\Delta t)^2}{2} M \\ &\leq \|z_i^k - z_i(t_k)\| + \Delta t L_i \|z^k - z(t_k)\| + \frac{(\Delta t)^2}{2} M \\ &\leq \|z^k - z(t_k)\|_\infty + \Delta t L_i \|z^k - z(t_k)\|_\infty + \frac{(\Delta t)^2}{2} M \\ &\leq (1 + \Delta t L_i)\|z^k - z(t_k)\|_\infty + \frac{(\Delta t)^2}{2} M. \end{aligned} \tag{5.3.25}$$

Consequently, the following relation holds

$$\|e_{k+1}\|_\infty \leq (1 + \Delta t L)\|e_k\|_\infty + \frac{(\Delta t)^2}{2} M \text{ where } L = \max\{L_i\}. \tag{5.3.26}$$

By applying Lemma 5.3.5 we obtain

$$\|e_k\|_\infty \leq (1+\Delta tL)^k \|e_0\|_\infty + \frac{(1+\Delta tL)^k - 1}{1+\Delta tL - 1}\frac{(\Delta t)^2}{2}M. \tag{5.3.27}$$

Since we have $\|e_0\|_\infty = 0$, Eq. (5.3.27) becomes

$$\|e_k\|_\infty \leq \frac{\left(e^{k\Delta tL} - 1\right)}{L}\frac{\Delta t}{2}M. \tag{5.3.28}$$

□

5.4 NSFD scheme for the SIS model

The SIS model (4.2.1), with vaccination, written in the equivalent form (4.2.5) has the productive-destructive structure described in the model (5.3.1). In view of the analysis in Section 5.3, we propose the following NSFD scheme

$$\begin{cases} \dfrac{S^{k+1} - S^k}{\psi(\Delta t)} = -\beta S^{k+1}\dfrac{I^k}{N} - \phi_v S^k + cI^k \\ \dfrac{I^{k+1} - I^k}{\psi(\Delta t)} = \beta S^{k+1}\dfrac{I^k}{N} + \sigma\beta(N - S^{k+1} - I^{k+1})\dfrac{I^k}{N} - cI^k \end{cases} \tag{5.4.1}$$

where the number q is determined according to the eigenvalues of the matrix J in (4.2.27) as follows:

$$\psi(\Delta t) = \frac{1 - e^{-q\Delta t}}{q} \quad \text{with} \quad q \geq \phi_v = |\lambda_1|. \tag{5.4.2}$$

Note that the eigenvalue $\lambda_2 = \rho c$ is not considered in accordance with (5.2.10) because it depends on a bifurcation parameter and could easily destroy hyperbolicity of the equilibrium point.

Remark 5.4.1. *There are many ways of constructing NSFD schemes for (4.2.5) (see Mickens [61], Mickens [60], Mickens [62]). The one proposed here has the following advantages:*

(i) By adding the two equations in (5.4.1), we obtain

$$\frac{S^{k+1} + I^{k+1} - (S^k + I^k)}{\psi} = \sigma\beta(N - S^{k+1} - I^{k+1})\frac{I^k}{N} - \phi_v S^k. \tag{5.4.3}$$

Thus, the following conservation law associated with the continuous system (4.2.5) is preserved:

$$\dot{S} + \dot{I} = \sigma\beta(N - S - I)\frac{I}{N} - \phi_v S. \tag{5.4.4}$$

As shown in Mickens [63], conservation laws are important in the design of dynamically consistent NSFD schemes.

(ii) Rule 3 on the nonlocal approximation of nonlinear terms is reinforced to guarantee among other things the nonnegativity of discrete schemes.

(iii) The condition (5.4.2) defining q is in line with the general relation in (5.3.3).

(iv) Last but not least, the NSFD scheme (5.3.7) is in the spirit of Section 5.2. It thus enables us to appply Corollary 5.3.4 directly without investigating the eigenvalues for the discrete case.

We now want to apply Theorem 3.5.1 which pertains to the direction of the bifurcation of the discrete model (5.4.1). Upon rearrangement, the NSFD scheme (5.4.1) takes the following Gauss-Seidel cycle which is appropriate for computational purposes:

$$\left.\begin{aligned} S^{k+1} &= \frac{(1-\psi\phi_v)S^k + \psi c I^k}{1 + \beta\psi\dfrac{I^k}{N}} = F_1(z_1, z_2) \\ I^{k+1} &= \frac{\psi\beta(1-\sigma)S^{k+1}\dfrac{I^k}{N} + (1 + c\rho\psi)I^k}{1 + \psi\sigma\beta\dfrac{I^k}{N}} = F_2(z_1, z_2) \end{aligned}\right\} \tag{5.4.5}$$

where

$$(z_1, z_2) = (S^k, I^k), \quad \rho = R_0 - 1 \quad \text{is the bifurcation parameter,} \tag{5.4.6}$$

and the corresponding Jacobian matrix is

$$J_{NS} = \begin{pmatrix} -\psi\phi_v + 1 & \psi c \\ 0 & 1 \end{pmatrix} = I + \psi J, \tag{5.4.7}$$

as described in Eq. (5.3.11), where I is the identity matrix. The eigenvalues of J_{NS} are

$$\lambda_1 = 1 - \psi\phi_v < 1 \qquad \text{and} \qquad \lambda_2 = 1.$$

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

$$\mathbf{w} = \begin{pmatrix} \dfrac{c}{\phi_v} \\ \\ 1 \end{pmatrix} \text{ and } \mathbf{v} = \begin{pmatrix} 0 \\ \\ 1 \end{pmatrix}. \tag{5.4.8}$$

At $(z_1, z_2, \rho) = (0, 0, 0)$, we obtain the following result

$$\frac{\partial^2 F_2}{\partial z_1^2} = 0, \quad \frac{\partial^2 F_2}{\partial z_1 \partial z_2} = \frac{\psi\beta(1-\sigma)(1-\psi\phi_v)}{N}, \quad \frac{\partial^2 F_2}{\partial z_2^2} = \frac{2\psi^2 c\beta(1-\sigma)}{N} - \frac{2\sigma\beta\psi}{N}. \tag{5.4.9}$$

Consequently, the coefficients a and b in Eq. (3.5.1) are given by

$$\tilde{a} = \psi a \tag{5.4.10}$$

and

$$\tilde{b} = \psi b > 0, \tag{5.4.11}$$

where a and b are as defined in equations (4.2.30) and (4.2.31), respectively. We have established the following result.

Theorem 5.4.2. *The NSFD scheme (5.4.1) is dynamically consistent with respect to the backward bifurcation property of the continuous SIS model with vaccination. That is, the discrete SIS model (5.4.1) undergoes the backward bifurcation at $R_0 = 1$ under the condition (4.2.26) in Theorem 4.2.1.*

5.5 NSFD scheme for the malaria model

In this section, the malaria model (4.3.1) is written in the equivalent form:

$$\begin{cases} \dot{N}_h = \Lambda_h + \psi_h N_h - f_h N_h - \delta_h I_h, \\ \dot{E}_h = c\beta_{hv} I_v S_h - M_1 E_h, \\ \dot{I}_h = \nu_v E_h - M_2 I_h, \\ \dot{R}_h = \gamma_h I_h - M_3 R_h, \\ \dot{N}_v = \psi_v N_v - f_v N_v, \\ \dot{E}_v = c(\beta_{vh} I_h + \tilde{\beta}_{vh} R_h) S_v - M_4 E_v, \\ \dot{I}_v = \nu_v E_v - f_v I_v. \end{cases} \tag{5.5.1}$$

The right hand side of Eq. (5.5.1) has a positive function of inflows and a component which models outflows. Thus, it has the productive-destructive structure given in Eq. (5.3.1). Therefore, we propose the following NSFD scheme:

$$\begin{cases} \dfrac{N_h^{k+1} - N_h^k}{\phi} = \Lambda_h + \psi_h N_h^k - f_h N_h^k - \delta_h I_h^k, \\ \dfrac{E_h^{k+1} - E_h^k}{\phi} = c\beta_{hv} I_v^k S_h^{k+1} - M_1 E_h^k, \\ \dfrac{I_h^{k+1} - I_h^k}{\phi} = \nu_v E_h^k - M_2 I_h^k, \\ \dfrac{R_h^{k+1} - R_h^k}{\phi} = \gamma_h I_h^k - M_3 R_h^k, \\ \dfrac{N_v^{k+1} - N_v^k}{\phi} = \psi_v N_v^k - f_v N_v^k, \\ \dfrac{E_v^{k+1} - E_v^k}{\phi} = c(\beta_{vh} I_h^k + \tilde{\beta}_{vh} R_h^k) S_v^{k+1} - M_4 E_v^k, \\ \dfrac{I_v^{k+1} - I_v^k}{\phi} = \nu_v E_v^k - f_v I_v^k. \end{cases} \tag{5.5.2}$$

Here the complex denominator function is given by

$$\phi = \phi(\Delta t) = \frac{1 - e^{-q\Delta t}}{q} \quad \text{where} \quad q \geq \max\left\{ f_h, f_v, M_1, M_2, M_3, M_4, \frac{|\lambda|^2}{2|Re\lambda|} \right\}, \tag{5.5.3}$$

and λ represents all the eigenvalues of the Jacobian matrix in Eq. (4.3.29) with negative real parts. (For an alternative NSFD scheme for the model Eq. (4.3.1), we refer the reader to

Anguelov et al. [7]). The analogue of the comments in Remark 5.4.1 can be made for the NSFD scheme (5.5.2). The scheme (5.5.2) can be written in the following explicit form

$$\begin{cases} N_h^{k+1} = \phi\Lambda_h + \phi\psi_h N_h^k + (1-\phi f_h)N_h^k - \phi\delta_h I_h^k, \\ E_h^{k+1} = \dfrac{\phi c\beta_{hv} I_v^k (N_h^{k+1} - I_h^{k+1} - R_h^{k+1}) + (1-\phi M_1)E_h^k}{1 + c\phi\beta_{hv} I_v^k}, \\ I_h^{k+1} = \phi\nu_h E_h^k + (1-\phi M_2)I_h^k, \\ R_h^{k+1} = \phi\gamma_h I_h^k + (1-\phi M_3)R_h^k, \\ N_v^{k+1} = \phi\psi_v N_v^k + (1-\phi f_v)N_v^k, \\ E_v^{k+1} = \dfrac{\phi c(\beta_{vh} I_h^k + \tilde{\beta}_{vh} R_h^k)(N_v^{k+1} - I_v^{k+1}) + (1-\phi M_4)E_v^k}{1 + \phi c(\beta_{vh} I_h^k + \tilde{\beta}_{vh} R_h^k)}, \\ I_v^{k+1} = \phi\nu_v E_v^k + (1-\phi f_v)I_v^k. \end{cases} \tag{5.5.4}$$

The Jacobian matrix of the map in Eq. (5.5.4) evaluated at the DFE=$(N_h^*, 0, 0, 0, N_v^*, 0, 0)$ is given by

$$\begin{aligned} J_{NS} &= \begin{pmatrix} 1-\phi B & 0 & -\phi\delta_h & 0 & 0 & 0 & 0 \\ 0 & 1-\phi M_1^* & 0 & 0 & 0 & 0 & \phi c^*\beta_{hv} N_h^* \\ 0 & \phi\nu_h & 1-\phi M_2^* & 0 & 0 & 0 & 0 \\ 0 & 0 & \phi\gamma_h & 1-\phi M_3^* & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1-\phi(f_v^* - \mu_{1v}) & 0 & 0 \\ 0 & 0 & \phi c^*\beta_{vh} N_v^* & \phi c^*\tilde{\beta}_{vh} N_v^* & 0 & 1-\phi M_4^* & 0 \\ 0 & 0 & 0 & 0 & 0 & \phi\nu_v & 1-\phi f_v^* \end{pmatrix} \\ &= I + \phi J, \end{aligned} \tag{5.5.5}$$

as described in Eq. (5.3.11), where I is the identity matrix. As mentioned earlier the similarity between the NSFD schemes (5.3.2) and (5.5.2) enables us to appply Corollary 5.3.4 directly instead of investigating the eigenvalues of J_{NS}. The coefficients that determine the

direction of the bifurcation at $R_0 = 1$ as given in Eq. (3.5.1) of Theorem 3.5.1 are

$$\tilde{a} = \phi a \quad \text{and} \quad \tilde{b} = \phi b > 0 \tag{5.5.6}$$

where a and b are defined in equations (4.3.39) and (4.3.40). Then using Theorem 3.5.1, we obtain the following result which shows that the numerical scheme (5.5.2) correctly replicates the properties of the bifurcation for model (4.3.1) as stated below.

Theorem 5.5.1. *The NSFD scheme (5.5.2) is dynamically consistent with the bifurcation property of the continuous malaria model in the sense that the discrete scheme (5.5.2) exhibits a backward bifurcation at $R_0 = 1$ if $\Pi^* > 1$ (i.e. $a > 0$) and a forward bifurcation if $\Pi^* < 1$ (i.e. $a < 0$).*

5.6 Numerical simulations

In this section, we provide diagrams of the NSFD schemes (5.4.1) and (5.5.2). These diagrams support the fact that the NSFD schemes preserve the backward/forward bifurcation properties of the continuous SIS and malaria models.

For the NSFD scheme of the SIS with vaccination, the diagrams below are plotted using data set in Table 5.1. When $R_0 = 0.04 < 1$, Fig. 5.1 illustrates that the disease may die out or persist depending on wider ($\phi_v = 0.02$) or smaller ($\phi_v = 0.001$) vaccine coverage. This supports the results of Theorems 4.2.1 and 4.2.4. Backward bifurcation is shown in Fig. 5.2(a) with $\sigma = 0.0999$; and the vaccine is very effective. In this scenario we observe the existence of a positive endemic equilibrium even though $R_0 < 1$. In Fig. 5.2(b), $\sigma = 0.9001$ which means that the vaccine is not very effective. The bifurcation in this case is forward.

For the NSFD scheme of the malaria model, diagrams are plotted using the data set in Chitnis et al. [23]; for convenience we reproduce it in Table 5.2. The diagram in Fig. 5.3(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 4.3.3 (i.e $\delta_h^{crit} = 2.49 \times 10^{-4}$) and $R_0 = 1.0289$. This scenario is similar to the illustration in Fig. 2.5. As the bifurcation parameter c^* is varied, the value of the basic reproduction number R_0 also varies. The value of $\zeta_1 = 0.00072$ coincides with $R_0 = 1$ and thus, $c^* = \zeta_1$ is a bifurcation point. In this case, there is a unique small stable endemic equilibrium when $R_0 > 1$. The epidemiological implication of this phenomenon is that, the major requirement to prevent further spread of the disease is to reduce the basic reproduction number below one. 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. 5.3(b) with $\delta_h = 2.7 \times 10^{-4}$ and $R_0 = 0.9988$. This situation is similar to the case illustrated in Fig. 2.6. The bifurcation point is $\zeta_1 = 0.00074$ and again it coincides with $R_0 = 1$. The existence of a backward bifurcation makes disease control much harder because reducing R_0 below one is no longer sufficient. In order to bring the disease under control, measures should be taken to reduce c^* below 0.00072 which coincides with a specific value of $R_0 < R_0^c$ as discussed in Remark 4.2.4. In Fig. 5.4 we plot the proportion of infectious individuals using two different initial populations to illustrate the possibility of the disease persistence even though $R_0 < 1$.

We illustrate the result of Theorem 4.3.3 by plotting the graph of the coefficient a given in Eq. (4.3.39) against the disease-induced death rate δ_h in Fig. 5.5.

Parameters	**set**1	**set** 2
N	500	500
$I(0)$	20	0
Δt	1	—
c	0.01	0.02
β	0.02	0.2
ϕ_v	0.02 & 0.001	0.02
σ	0.02	0.9001 & 0.0999

Table 5.1: Data set for the SIS model.

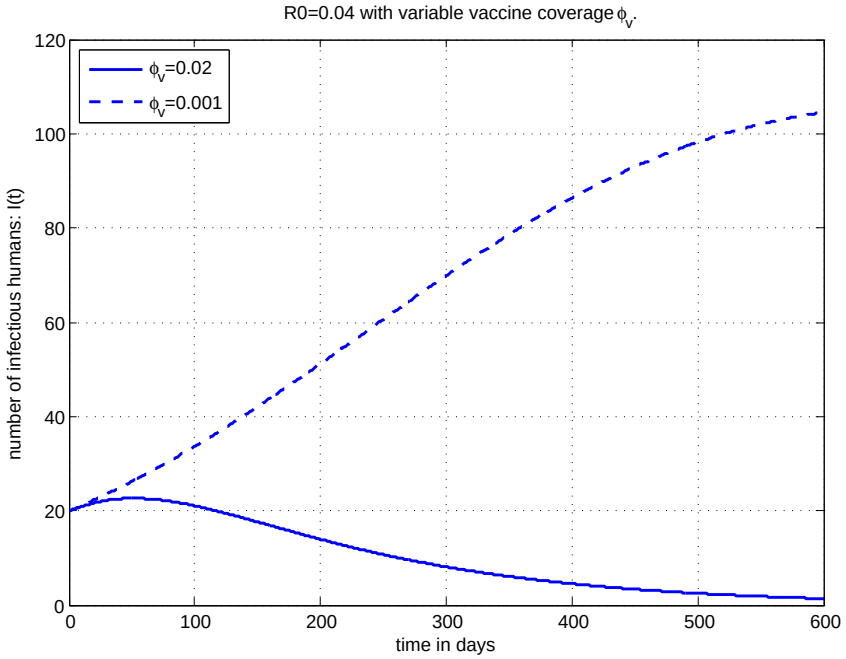
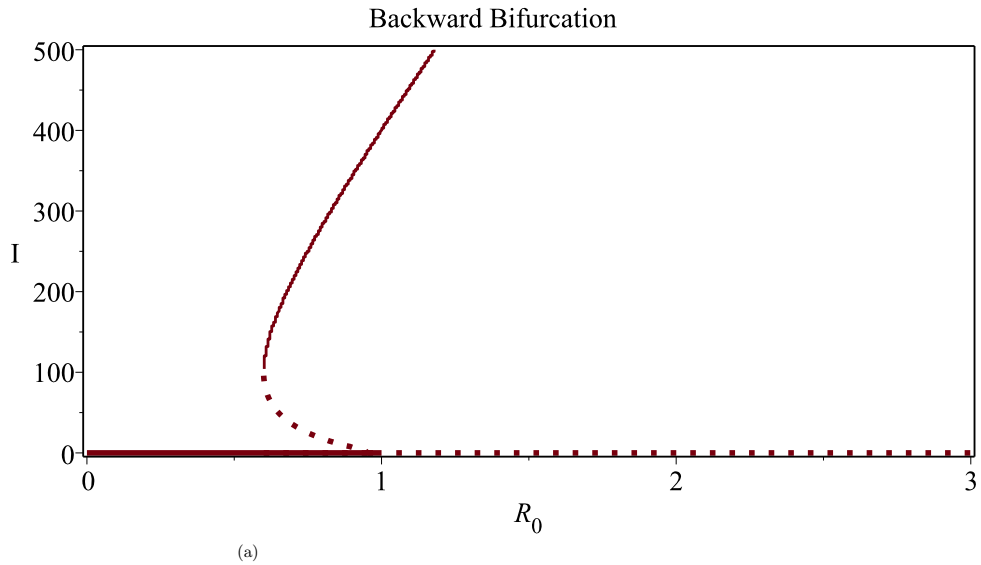


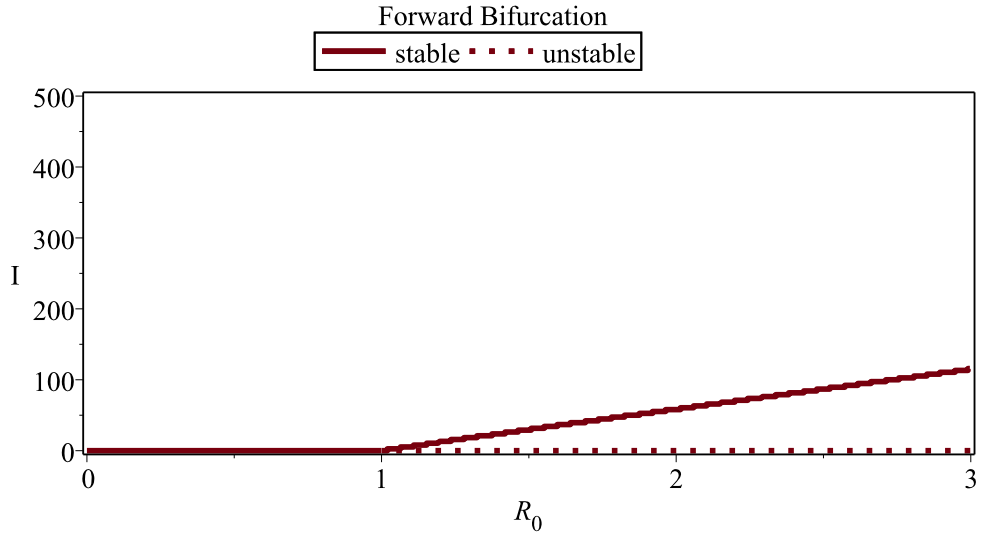
Figure 5.1: Dynamics of the SIS model versus vaccination coverage: Data set 1.

Parameters	**set**1
Λ_h	3.285×10^{-2}
ψ_h	7.666×10^{-5}
ψ_v	0.4
β_{vh}	0.8333
$\tilde{\beta}_{vh}$	8.333×10^{-2}
σ_v	0.6
σ_h	18
ν_h	8.333×10^{-2}
ν_v	0.1
γ_h	3.704×10^{-3}
ρ_h	1.460×10^{-2}
μ_{1h}	4.212×10^{-5}
μ_{2h}	10^{-7}
μ_{1v}	0.1429
μ_{2v}	2.279×10^{-4}
β_{hv}	2×10^{-2}

Table 5.2: Data set for the malaria model.



(a)



(b)

Figure 5.2: Bifurcation direction versus vaccine efficacy (Data set 2):(a) Backward bifurcation (effective vaccine, $\sigma = 0.0999$). (b) Forward bifurcation (ineffective vaccine, $\sigma = 0.9001$).

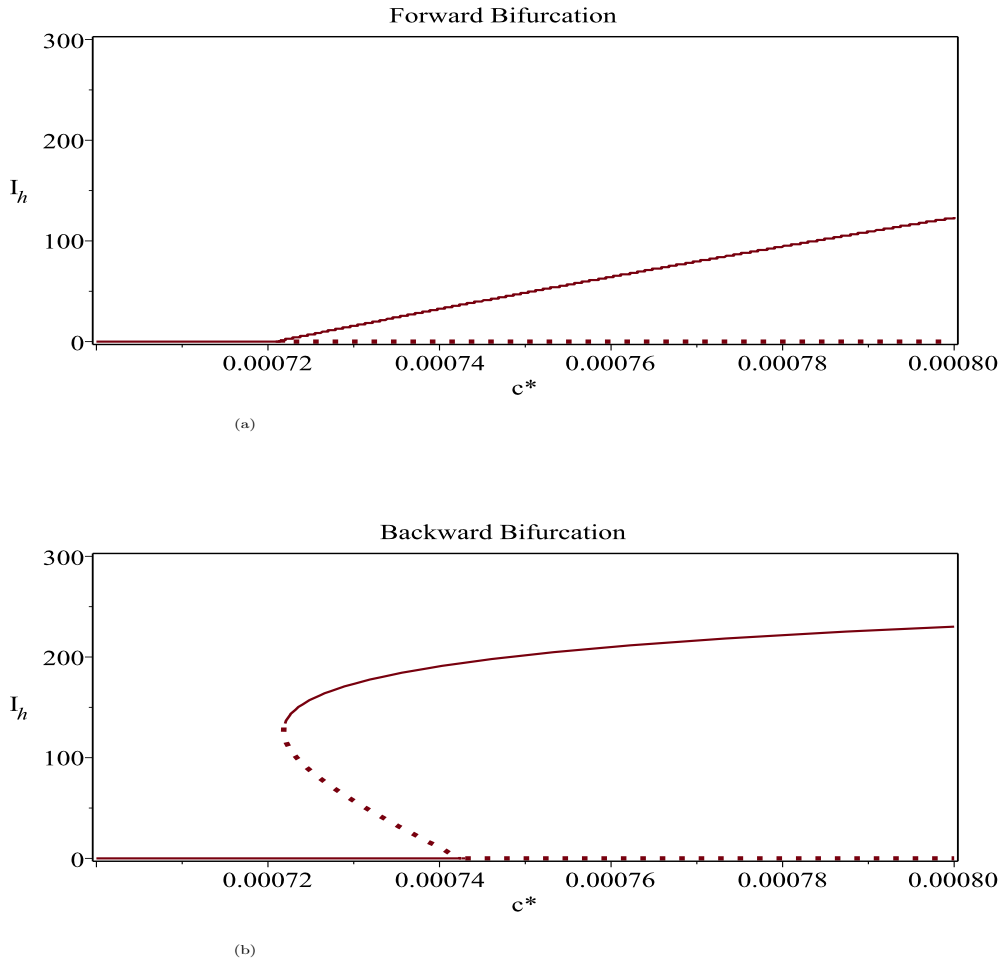


Figure 5.3: Bifurcation direction versus disease-induced death rate (δ_h): (a) Forward bifurcation ($\delta_h = 0.3419 \times 10^{-4}$). (b) Backward bifurcation ($\delta_h = 2.7 \times 10^{-4}$).

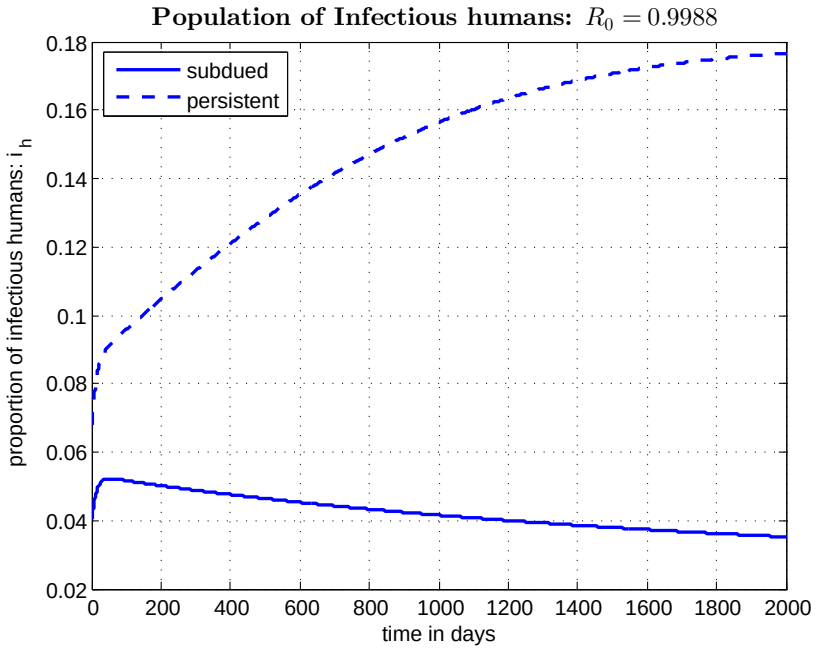


Figure 5.4: The population of infectious humans may increase or decline depending on the initial population, even though the basic reproduction number is less than one. This diagram illustrates that there is a possibility of the occurrence of a backward bifurcation at $R_0 = 1$.

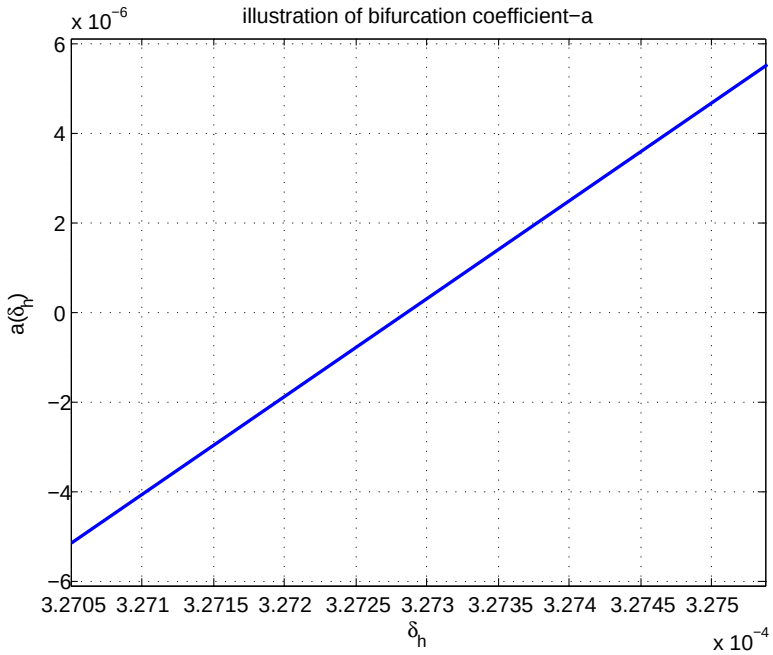


Figure 5.5: The graph of the bifurcation coefficient a as a function of the disease-induced death rate δ_h.

CHAPTER 6. CONCLUSION

This thesis is motivated by two works of great importance in the field, and a current gap in our knowledge which requires investigation. The first element pertains to a theorem stated and proved in Castillo-Chavez and Song [16] regarding sufficient conditions for a continuous dynamical system to undergo the backward bifurcation phenomenon. The theorem is extensively used in epidemiology. This is mostly done through computer software codes and numerical simulations (see Garba et al. [37], Gumel et al. [40], Hussaini et al. [49], Hassan et al. [44]). In applying the theorem, it becomes impossible not to appreciate the power of the involved Centre Manifold Theory.

The second motivation for this thesis is a conjecture stated in Chitnis et al. [23] for a complex and strongly nonlinear malaria model. The conjecture reads as follows: *The model undergoes the backward bifurcation phenomenon whenever the disease-induced death rate is large enough.* This conjecture is illustrated by numerical simulations in Chitnis et al. [23] and to some extent in Anguelov et al. [7].

The third motivation is that no rigorous analysis has been done for the few existing NSFD schemes that approximate epidemiological models that undergo the backward bifurcation phenomenon. Thus far the preservation of this property by NSFD schemes was only illustrated through numerical tests (as seen in Garba et al. [36]).

In this thesis, we have addressed the above concerns and carried out an in-depth study of sufficient conditions that guarantee the occurrence of the backward bifurcation phenomenon in two epidemiological models. Our findings can be summarised as follows:

1. We re-centred the context of the theorem in Castillo-Chavez and Song [16] by highlighting its advantages over the laborious power series approximations method. We further showed that, even if the higher order terms are not truncated in the theorem, the signs of the coefficients a and b remain the main determining factors of the bifurcation direction at $R_0 = 1$.
2. We stated and proved a result, which is new, to the best of the author's knowledge, on the discrete analogue of the main theorem in Castillo-Chavez and Song [16]. Our result is designed for bifurcation analysis of discrete dynamical systems. The importance and relevance of this result is clear in the light of the fact that, transposing theorems on continuous dynamical systems to discrete dynamical systems is often a challenge. A typical example is the Poincaré-Bendixon theorem (Brauer and Castillo-Chavez [13]).
3. We constructed NSFD schemes for the SIS and malaria models by Villavicencio-Pulido et al. [79] and Chitnis et al. [23], respectively.
4. We established theoretically and computationally that the NSFD schemes preserve the backward bifurcation property of the two continuous models. To the best of the author's knowledge, this thesis is one of a few works which deal with NSFD schemes for epidemiological models that undergo a backward bifurcation. The addition of an in-depth analysis of the schemes made sure that we have contributed value to the few existing works (see Garba et al. [37], Anguelov et al. [7]).

The results obtained in this thesis and challenges encountered during the process, have lead to numerous research questions which require further investigation. Some of them are listed below.

(i) To investigate the existence of a critical value $R_0^c < 1$ of the basic reproduction number such that the disease-free equilibrium of the considered malaria model is glob-

ally asymptotically stable (GAS) for $R_0 < R_0^c$ as shown for the SIS model (see Remark 4.2.4). From the public health perspective, this is one of the ways to control and manage communicable diseases (Donaldson and Rutter [29]).

(ii) To show that the NSFD scheme preserves the GAS property as stated in item (i) above.

(iii) To extend the findings and ideas of this thesis to some malaria models developed after the one by Chitnis et al. [23] and to epidemiological models in which the backward bifurcation phenomenon is due to causes other than the disease-induced death rate (see Hussaini et al. [49], Gumel et al. [40]).

(iv) To investigate an extension of the theorem by Castillo-Chavez and Song [16] to a case when zero is a double eigenvalue. Although the Takens-Bogdanov normal form is analysed in Kuznetsov [53], the challenging nature of the question envisaged here cannot be under-estimated in applications, specifically in epidemiology.

(v) To investigate the design of dynamically consistent NSFD schemes for continuous dynamical systems which undergo the backward bifurcation but do not have the productive-destructive structure. This requires further understanding of the dynamics of the system under consideration, which needs to be incorporated into the NSFD scheme by using Mickens' rules and other relevant strategies (see Mickens [61],[62],[64], Roeger and Mickens [73], Roeger [71]).

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