Utrecht University

# HOPF BIFURCATION IN EPIDEMIOLOGICAL MODELS WITH DELAYS

DEPARTMENT OF MATHEMATICS

BACHELOR THESIS

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June 18, 2021

**Abstract**

Epidemiological models with time delays often display oscillations, this thesis will present the conditions needed for either stable or unstable periodic solutions to occur within these models. We will specifically look at models for which the stability changes as a function of the time delay and provide formulas to determine the stability of periodic cycles. Then we will analyse some specific models using the MATLAB package DDE-BIFTOOL.

# Contents

# Part I
# Introduction

We begin by discussing epidemic models. A mathematical model describing the spread of a disease generally consists of a system of functions which give the population of a certain group with respect to time. Because it is easier to formulate models using the rate of change of these functions a model will mostly consist of a system of differential equations.

## 1 Standard epidemiological model

The most simple model contains three populations. The susceptible population $S(t)$ which consists of the individuals who could become infected, the infected population $I(t)$ consisting of individuals who contracted the disease and the recovered population $R(t)$ of those who overcame the infection. We will distinguish between models with constant population size and models without constant population size.

### 1.1 Constant population

The standard model describing the rates of change of the populations $S$, $I$ and $R$ is given by the following system of differential equations:

$$\begin{cases} \dot{S}(t) &= -\dfrac{\beta IS}{N}, \\ \dot{I}(t) &= \dfrac{\beta IS}{N} - \gamma I, \\ \dot{R}(t) &= \gamma I, \end{cases} \tag{1.1.1}$$

Where $N = S + I + R$ denotes the total population. This model was first introduced in 1927 by W.O. Kermack and A.G. McKendrick [1]. Here the rate of change of the susceptible population is assumed to depend on the product of the susceptible population with the infected population and a constant $\beta$. This is because every time a susceptible individual comes in contact with an infected one there is a chance the susceptible individual will become infected, using the constant $\beta$ we can account for the probability someone becomes infected. Because these individuals will join the infected population we have a term $\beta IS/N$ in the differential equation for $I$ where we had $-\beta IS/N$ in the equation for the susceptible population, we will call this term the **incidence rate**. Because individuals in the infected population will also transfer to the recovered population we lastly have a term $-\gamma I$ in the equation for the infected population and a term $\gamma I$ in the equation for the recovered population.

This model does not include a natural death rate or birth rate for the population, looking at the derivative of the total population we will therefore get:

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0. \tag{1.1.2}$$

So we have a constant population size. This model can be used to model a population over a small amount of time where the death and birth rates will not influence the population in a big way. Instead of working with a total population we could also work with fractions of the population by substituting $\bar{S} = S/N$, $\bar{I} = I/N$ and $\bar{R} = R/N$. The system then becomes:

$$\begin{cases} \dot{\bar{S}} &= -\beta \bar{S}\bar{I}, \\ \dot{\bar{I}} &= \beta \bar{S}\bar{I} - \gamma \bar{I}, \\ \dot{\bar{R}} &= \gamma \bar{I}. \end{cases} \tag{1.1.3}$$

Because we divided by the total population we now get $\bar{S} + \bar{I} + \bar{R} = (S + I + R)/N = 1$.

**Definition 1** (Basic reproduction number)**.** *The **basic reproduction number** $R_0$ of a disease is the expected number of susceptible individuals which become infected by a single infected person.*

In system (1.1.1) the basic reproduction number is $R_0 = \beta/\gamma$, because the time between contacts is $\beta^{-1}$ and the time before removal form the infected population is $\gamma^{-1}$.

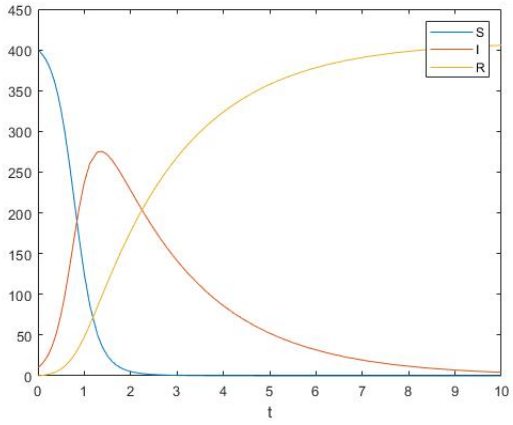



Figure 1.1: Solution to system (1.1.1) for $\beta = 5$ and $\gamma = 0.5$.

In figure (1.1) we see the solution to system (1.1.1), we can see the infected population start to infect the susceptible population who in turn gain immunity and the infected population will therefore deplete.
Notice that the equation for $\dot{R}$ is uncoupled from the first two in (1.1.1). Removing it leaves us only with the equations for $\dot{S}$ and $\dot{I}$. The phase portrait for SI then becomes:

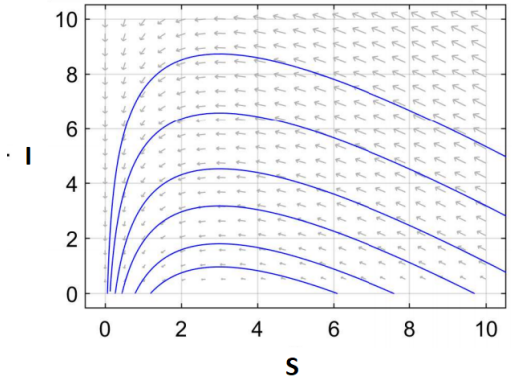



Figure 1.2: Phase portrait of susceptible and infected populations for system (1.1.1)

## 1.2 Non-constant population

The next model we will look at does include a inflow/immigration rate and a natural death rate, so it is more suitable for modeling epidemics over longer periods of time but will also be more complex. The model is given by the following system of differential equations:

$$\begin{cases} \dot{S}(t) &= A - \mu S(t) - \dfrac{\beta I(t) S(t)}{N(t)}, \\ \dot{I}(t) &= \dfrac{\beta I(t) S(t)}{N(t)} - \gamma I(t) - \mu I(t), \\ \dot{R}(t) &= \gamma I(t) - \mu R(t), \end{cases} \tag{1.2.1}$$

Where $A$ is the inflow/immigration rate and $\mu$ is the natural death rate. This model only differs from (1.1.1) in the fact that individuals in all groups will have a term for the natural death rates within the populations and the inflow/immigration rate in the susceptible population. An example of an inflow rate is the birth rate in models describing human populations. For the derivative of the population we get the following equation:

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = A - \mu N(t) \tag{1.2.2}$$

So we again get a constant population if we have a initial population of $N(0) = A/\mu$, if $N(0) \neq A/\mu$ we can solve equation (1.2.2) and get the following:

$$N(t) = \frac{A}{\mu} + \left( N(0) - \frac{A}{\mu} \right) e^{-\mu t}. \tag{1.2.3}$$

So we see $N(t) \to A/\mu$ as $t \to \infty$ and therefore converges to a constant population. The basic reproduction number of this system is $R_0 = \frac{\beta}{\mu+\gamma}$. Taking the same parameter values as (1.1) with the additional $A = 10$ and $\mu = 0.1$ we get the following figure:

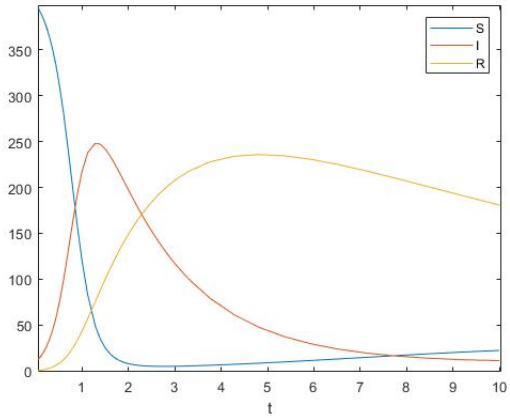



Figure 1.3: Solution to system (1.1.1) for $\beta = 5$, $\gamma = 0.5$, $A = 10$ and $\mu = 0.1$.

We see that the figure is very similar to (1.1), only difference being that the susceptible population will once again start to grow together with the infected population and the recovered population will start to decline due to the natural death rate.

While the incidence rate used in system (1.2.1) is the standard one, we can also use what is known as the mass action incidence found in [2]. For this we assume the following:

(i) $cN$ is the number of contacts per unit time infected individuals make.

(ii) $\frac{S}{N}$ is the probability that a contact involves a susceptible individual, and therefore $cN\frac{S}{N} = cS$ is the number of contacts which one infected individual makes with susceptible individuals.

(iii) $pcS$ is the number of susceptible individuals per unit of time who become infected per infected individual. Here $p$ is the probability of a susceptible individual becoming infected after having been in contact with an infected individual.

The incidence rate then becomes $\beta SI$ where $\beta = pc$. Note that because we assumed in (i) that the number of contacts per unit time is proportional to the population size we assume that the number of contacts increases with an increased population size, which was not the case with the standard incidence rate. These incidence rates agree when the population size remains constant, but differ if the total population size is variable. The mass action incidence rate can therefore be used to model diseases like influenza or SARS, where contacts increase as population size and therefore population density increases. The standard incidence rate can be used for diseases where there is a certain bound on the number of contacts an infected individual has, this is the case for sexually transmitted diseases where the amount of contacts does not increase with population size. In this thesis we will mostly look at diseases which are not sexually transmitted, so we will mostly look at systems similar to the following:

$$\begin{cases} \dot{S}(t) &= A - \mu S(t) - \beta S(t)I(t), \\ \dot{I}(t) &= \beta S(t)I(t) - \gamma I(t) - \mu I(t), \\ \dot{R}(t) &= \gamma I(t) - \mu R(t), \end{cases} \tag{1.2.4}$$

Notice that the third equation is again uncoupled from the first two equations, so we only have to look at the equations for the susceptible and infected populations. A phase portrait for these two populations is found in the following figure where $\beta = 0.1$, $\gamma = 0.3$, $\mu = 0.01$ and $A = 1$:

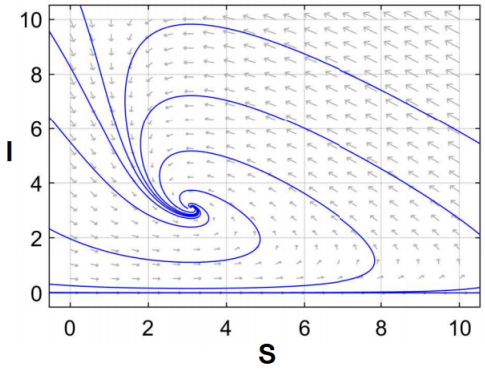



Figure 1.4: Phase portrait of susceptible and infected populations for system (1.2.4)

In this case we have a very different phase portrait then in (1.2). Where in that portrait the solutions converged towards zero we now have the solution converging towards a nonzero **equilibrium**. This is because instead of the population being constant and all the individuals of the population eventually ending up in the recovered population we know have a constant inflow in the susceptible population.

# 2 Variations of the standard model

Now that we defined the standard model in (1.2.1) we can start to add elements which make the system more complicated and therefore better suited to specific diseases. We will now look at adding another sub-population and changing the incidence rate to a nonlinear one.

## 2.1 The exposed population

There are many types of diseases which have a significant incubation period, meaning that an individual is infected but cannot yet spread the disease to other subjects. During this period we will place these individuals in a separate population, namely the exposed population $E$. A model incorporating this is called an SEIR model, we will consider the following one:

$$\begin{cases} \dot{S}(t) &= A-\mu S(t)-\beta I(t)S(t), \\ \dot{E}(t) &= \beta I(t)S(t)-\mu E(t)-aE(t), \\ \dot{I}(t) &= aE(t)-\mu I(t)-\gamma I(t), \\ \dot{R}(t) &= \gamma I(t)-\mu R(t). \end{cases} \tag{2.1.1}$$

Here instead of the susceptible population directly feeding into the infected population we see that the susceptible population first transfers into the exposed population via the incidence rate.

## 2.2 Models with time delays

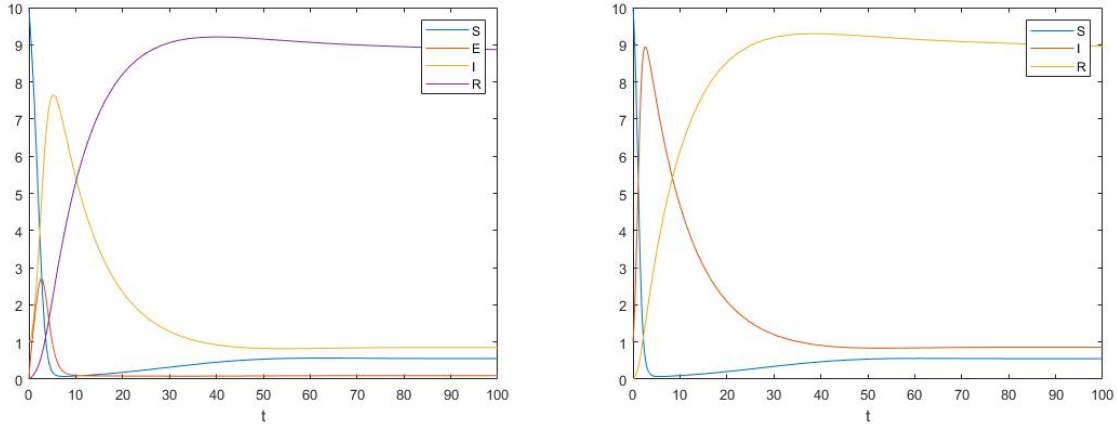
We can also substitute $t-\tau$ instead of just $t$ where $\tau$ indicates the time delay, which transforms the model into a system of **delayed differential equations (DDE's)**. For example, we can delay the transfer from the infected population to the recovered population in order to add a infected period to our model in the case of diseases like SARS of influenza or for computer viruses we can add a delay to the incidence rate to simulate the time it takes for infected nodes to start infecting the susceptible nodes. In the case of SARS or influenza we can look at the following system for example:

$$\begin{cases} \dot{S}(t) &= A-\mu S(t)-\beta S(t)I(t), \\ \dot{I}(t) &= \beta S(t)I(t)-\gamma I(t-\tau)-\mu I(t), \\ \dot{R}(t) &= \gamma I(t-\tau)-\mu R(t), \end{cases} \tag{2.2.1}$$

Here $\tau$ indicates the infected period, so when a susceptible subject enters the infected stage it will take $\tau$ units of time before it can transfer to the recovered population. Adding a delayed term to the incidence rate can also be used to model the time it takes for the infection to develop in a subject, so we could use this instead of the separate exposed population. In a way adding a separate exposed population is similar to adding a delay because it takes more time for a susceptible subject to transfer to the infected population. An immediate drawback to using delayed terms in models is that the system becomes harder to analyse.

### 2.2.1 Delayed term instead of exposed population

In the previous section we introduced the exposed population, this makes it so that there is a time between the susceptible and infected state, which could also be modelled using time delays instead. As an example we will look at the following simulations of 2.1.1 and 1.2.4 for $A = 0.1, \mu = 0.01, \beta = 0.2, \gamma = 0.1$ and $a = 1$:




We can clearly see the infected population reaching its maximum value at an earlier point in the normal SIR model then in the SEIR model, which illustrates that using a separate exposed sub-population is in some ways like using a delayed term in the equation.

### 2.2.2 Ways to incorporate delays

We will now discuss different ways to add time delays to our models.

- First of all we can add a time delay to our incidence rate for models where it takes a while for a susceptible individual to become infected after contact. As we discussed previously this serves a similar purpose as adding an exposed population to our model.

$$\begin{aligned} \dot{S}(t) &= -\beta I(t-\tau)S(t-\tau) + \text{other terms}, \\ \dot{I}(t) &= \beta I(t-\tau)S(t-\tau) + \text{other terms}. \end{aligned} \tag{2.2.2}$$

- A time delay can be added to the transfer rate between the infected and recovered population to model the time it takes for an individual to recover from the disease.

$$\begin{aligned} \dot{I}(t) &= -\gamma I(t-\tau) + \text{other terms}, \\ \dot{R}(t) &= \gamma I(t-\tau) + \text{other terms}. \end{aligned} \tag{2.2.3}$$

- In order to add a temporary immunity to our model we can add a delay to the transfer term from the recovered population to the susceptible population. In these models it is assumed that their is no immunity after having recovered from the disease or virus. The time delay will let us be able to add a time in which an individual is recovered and after this time it will again be possible to enter the susceptible population.

$$\begin{aligned} \dot{S}(t) &= \varepsilon R(t-\tau) + \text{other terms}, \\ \dot{R}(t) &= -\varepsilon R(t-\tau) + \text{other terms}. \end{aligned} \tag{2.2.4}$$

## 2.3 Nonlinear incidence rate

In the standard model we defined we assumed that the incidence rate is bi-linear, which is a strong assumption because the infection rate almost certainly does not linearly increase with the size of the infected population. The infection rate per infected individual most likely will decrease as the infected population increases because of the behavioral change in the susceptible population, in [3] it is assumed that instead of a bi-linear incidence rate we have one involving a term $g(I)$ where $g : \mathbb{R}_+ \to \mathbb{R}_+$ satisfies the following conditions:

(i) $g(0) = 0$;

(ii) $g$ is bounded, so there exists a constant $c \in \mathbb{R}_{>0}$ such that $g(x) \leq c$ for all $x \in \mathbb{R}_+$;

(iii) the derivative of $g$ exists and is bounded on any compact interval of $\mathbb{R}_+$, with $g'(0) > 0$;

(iv) $g(x) < g'(0)x$ for all $x \in \mathbb{R}_+$.

Here (iv) ensures the rate of change of the function will decrease as $I$ gets big. An example for $g$ is the following:

$$g(I) = \kappa \frac{I}{1 + (I/\alpha)}. \tag{2.3.1}$$

Where $\kappa, \alpha > 0$, we see that as $I$ gets bigger the infection rate will decrease relative to the size of the infected population. The incidence rate in this case will be equal to $\frac{\beta IS}{1+(I/a)}$. We can also let this function $g$ depend on the ratio $I/S$ like in [4] which results in the following($\alpha = 1$):

$$g(I/S) = \frac{\kappa(I/S)}{1 + I/S} = \frac{\kappa I}{S + I}. \tag{2.3.2}$$

Here the incidence rate will equal $\frac{\beta SI}{S+I}$.

# 3 Structure of this thesis

This thesis is divided into a theoretical part and case studies. In the theoretical part we will discuss the theory behind ordinary differential equations and delayed differential equations, which define our epidemiological models. We will specifically look at Hopf bifurcation in these systems, a bifurcation is defined as the phase portrait of a system changing significantly when we change a parameter a certain amount and Hopf bifurcation is a specific type of bifurcation we will define in the next part. For ordinary differential equations the parameter can be any constant within our system and for delayed differential equations we will specifically look at changing the time delay values in order to find bifurcations. Next we will look at how to find and study the properties of Hopf bifurcation numerically using DDE-BIFTOOL, which implements the formula's we will find in the part about delayed differential equations. This tool will help us analyse different case studies. Lastly in the theoretical part we will see how to simulate models in MATLAB using dde23.

In the case studies we will look at 8 different DDE systems which are used to model either diseases or computer viruses within networks. We will look at academic papers discussing these models and we will check the validity of these papers using DDE-BIFTOOL and attempt to recreate their simulations using dde23. For two of these models we get the same results as described in the paper, in the rest of the examples we find different results then found in the papers. In three of these models the Hopf bifurcation was subcritical instead of supercritical and in the last two there was no Hopf bifurcation at all. The counterexamples found in these models are considered to be the contributions of this thesis.

# Part II
# Hopf Bifurcation in Ordinary Differential Equations

In this part we will look at the theory behind ordinary differential equations in order to better understand the epidemiological models described in the Introduction. We will look at equilibria and what it means for an equilibrium to be stable, after this we will describe what it means for a system to undergo a Hopf bifurcation and look at the two types of Hopf bifurcation and the necessary conditions for them to occur. For convenience we will abbreviate ordinary differential equations as ODE's.

## 4 Equilibria and their stability

Consider the following system of differential equations with parameter $\alpha \in \mathbb{R}$:

$$\dot{x}(t) = f(x(t), \alpha), \quad x(t) \in \mathbb{R}^n, t \in \mathbb{R}, \tag{4.0.1}$$

where $f : \mathbb{R}^n \times \mathbb{R} \to \mathbb{R}^n$ is smooth. We start with a few definitions.

**Definition 2** (Equilibrium)**.** *The system (4.0.1) has an **equilibrium** at a point $x_0(\alpha) \in \mathbb{R}^n$ when $f(x_0(\alpha), \alpha) = 0$.*

**Definition 3** (Orbit)**.** *For a solution $x = x(t, x_0)$ of (4.0.1) where $x(0, x_0) = x_0 \in \mathbb{R}^n$ we define the **orbit** with the starting point $x_0$ as*

$$Or(x_0) = \{x \in \mathbb{R}^n \mid x = x(t, x_0), t \in \mathbb{R}\} \tag{4.0.2}$$

*and orient it by the advance of time.*

We will call the collection of all these orbits the **phase portrait**.

**Definition 4** (Flow)**.** *Given a solution $x = x(t, x_0)$ starting at $x_0 \in \mathbb{R}^n$, the **flow** is an operator $\varphi^t : \mathbb{R}^n \to \mathbb{R}^n$ that is defined by*

$$\varphi^t(x_0) = x(t, x_0). \tag{4.0.3}$$

Note that an equilibrium $x_0(\alpha)$ is a point such that $\phi^t(x_0(\alpha)) = x_0(\alpha)$ for all $t \in \mathbb{R}$. An equilibrium $x_0(\alpha)$ is called **stable** if

(i) for any neighbourhood $U$ of $x_0(\alpha)$ there exists a neighbourhood $V$ of $x_0(\alpha)$ such that for all $t \geq 0$ holds $\varphi^t x \in U$ provided $x \in V$;

(ii) there is a neighbourhood $U_0$ of $x_0(\alpha)$ such that for all $x \in U_0$ holds $\varphi^t x \to x_0$ when $t \to \infty$.

An equilibrium is called **unstable** if it is not stable. In order to determine the stability of an equilibrium for a system like (4.0.1) we need to calculate the eigenvalues of the Jacobian matrix of $f$. The **characteristic matrix** is given by:

$$\Delta(\lambda) = \lambda \mathbb{I} - Df(x_0, \alpha). \tag{4.0.4}$$

The eigenvalues of the Jacobian matrix are the roots of the characteristic polynomial given by $\det \Delta(\lambda)$. The relation between these eigenvalues and the stability of an equilibrium is given by the following theorem:

**Theorem 4.1** (Lyapunov)**.** *Consider a system of differential equations*

$$\dot{x}(t) = f(x(t)), \quad x(t) \in \mathbb{R}^n, t \in \mathbb{R}, \tag{4.0.5}$$

*where $f$ is smooth. Suppose it has an equilibrium $x_0$ and denote by $A$ the Jacobian matrix of $f(x)$ evaluated at the equilibrium. Then $x_0$ is stable if all eigenvalues $\lambda_1, \lambda_2, \ldots, \lambda_n$ of $A$ satisfy $\Re(\lambda) < 0$. If there is at least one eigenvalue with $\Re(\lambda) > 0$, the equilibrium $x_0$ is unstable.*

**Example 4.1.** Setting $x = (x_1, x_2, x_3)$ with $x_1 = S$, $x_2 = I$, and $x_3 = R$, we can write system (1.2.1) in the following way:

$$\dot{x}(t) = f(x(t)), \tag{4.0.6}$$

where

$$f(x) = \begin{pmatrix} A - \mu x_1 - \frac{\beta x_1 x_2}{x_1 + x_2 + x_3} \\ \frac{\beta x_1 x_2}{x_1 + x_2 + x_3} - \gamma x_2 - \mu x_2 \\ \gamma x_2 - \mu x_3 \end{pmatrix}. \tag{4.0.7}$$

The equilibria of this system are found by solving $f(x) = 0$, which gives the following stable equilibria:

(i) The disease-free equilibrium $x_0 = \left(\frac{A}{\mu}, 0, 0\right)$ when $R_0 \leq 1$.

(ii) The endemic equilibrium $x_* = \left(\frac{(\gamma+\mu)A}{\beta\mu}, \frac{A}{\beta}(R_0-1), \frac{\gamma A}{\beta\mu}(R_0-1)\right)$ for $R_0 > 1$.

Here $R_0 = \frac{\beta}{\gamma+\mu}$ is the basic reproduction number. This example illustrates the importance of the reproduction number in epidemic models, when $R_0 \leq 1$ we eventually get to a point where there are no more infected nodes but if $R_0 > 1$ we get a nonzero steady state.

# 5 Bifurcation

Our goal is to study how the phase portrait of system (4.0.1) changes when we change the parameter $\alpha$. In order to do this, we will need to classify the phase portraits of these differential equations. For this we will define what it means for two phase portraits to be topologically equivalent:

**Definition 5** (Topological equivalence)**.** *We will call two systems of differential equations* ***topologically equivalent*** *if there exists a homeomorphism $h : \mathbb{R}^n \to \mathbb{R}^n$ mapping orbits of the first system onto orbits of the second one, preserving the direction of time.*

Because it is easier to study these differential equations locally, namely around an equilibrium, we will further define what it means for phase portraits to be locally topologically equivalent near an equilibrium:

**Definition 6** (**Local topological equivalence**)**.** *Consider two systems of differential equations:*

$$\dot{x}(t) = f(x(t)), \quad x(t) \in \mathbb{R}^n, t \in \mathbb{R}, \tag{5.0.1}$$

$$\dot{y}(t) = g(y(t)), \quad y(t) \in \mathbb{R}^n, t \in \mathbb{R}, \tag{5.0.2}$$

*where $f, g$ are smooth functions. Assume (5.0.1) and (5.0.2) have equilibria $x_0 \in \mathbb{R}^n$ and $y_0 \in \mathbb{R}^n$ respectively, then the phase portraits of these differential equations are called* ***locally topologically equivalent*** *near these equilibria if there exists a homeomorphism $h : \mathbb{R}^n \to \mathbb{R}^n$ that is*

(i) *defined in a small neighborhood $U \subset \mathbb{R}^n$ of $x_0$;*

(ii) *satisfies $y_0 = h(x_0)$;*

(iii) *maps orbits of (5.0.1) in $U$ onto orbits of (5.0.2) in $V = h(U) \subset \mathbb{R}^n$, preserving the direction of time.*

**Theorem 5.1.** *The systems (5.0.1) and (5.0.2) with equilibria $x_0$ and $y_0$ are locally topologically equivalent near these equilibria if they have no eigenvalues with $\Re(\lambda) = 0$ and the same number $n_-$ and $n_+$ of eigenvalues with $\Re(\lambda) < 0$ and with $\Re(\lambda) > 0$, respectively.*

Going back to the differential equation defined in (4.0.1), when we change the parameter $\alpha$ to $\alpha'$ there are two possibilities: either the phase portraits of $\dot{x}(t) = f(x(t), \alpha)$ and $\dot{x}(t) = f(x(t), \alpha')$ remain topologically equivalent, or this is not the case.

**Definition 7** (Bifurcation)**.** *The appearence of a topologically nonequivalent phase portrait under variation of parameters is called a* ***bifurcation****.*

We will call the exact value of the parameter at which the phase portrait changes the **bifurcation (critical) value**.

Since generic systems of equations satisfying the same bifurcation conditions at an equilibrium are often locally topologically equivalent it is handy to define a simple system satisfying specific bifurcation and nondegeneracy conditions. This leads to the next definition:

**Definition 8** (Topological normal form)**.** *Consider a polynomial system:*

$$\dot{\xi} = g(\xi, \beta, \sigma), \xi \in \mathbb{R}^n, \beta \in \mathbb{R}^k, \sigma \in \mathbb{R}^l, \tag{5.0.3}$$

*Together with (5.0.3) consider another system,*

$$\dot{x} = f(x, \alpha), x \in \mathbb{R}^n, \alpha \in \mathbb{R}^k, \tag{5.0.4}$$

*having at $\alpha = 0$ an equilibrium $x = 0$. System (5.0.3) is called a* ***topological normal form*** *for the bifurcation if any generic system (5.0.4) with an equilibrium at $x = 0$ satisfying the same bifurcation conditions at $\alpha = 0$ is locally topologically equivalent near the origin to system (5.0.3) for some value of $\sigma$.*

# 6 Hopf bifurcation in 2 dimensions

There are many types of bifurcation, we will focus on Hopf bifurcation. This type of bifurcation is defined as follows:

**Definition 9** (Hopf bifurcation)**.** *The bifurcation corresponding to the presence of* $\lambda_{1,2} = \pm i\omega_0$ *where* $\omega_0 > 0$*, is called a* ***Hopf bifurcation****.*

In this type of bifurcation we have a periodic solution for specific values of the parameter, this is specifically relevant in epidemiological models because having repeating outbreaks is not a good scenario while confronted with a disease or computer virus. We will first introduce the normal form of this bifurcation and then discuss the conditions for which Hopf bifurcation occurs.

## 6.1 Normal form of Hopf bifurcation in 2 dimensions

We begin by introducing the topological normal form of Hopf bifurcation as defined in the last section. Consider the following 2-dimensional differential equation with one parameter:

$$\begin{cases} \dot{x}_1 &= \alpha x_1 - x_2 - x_1(x_1^2 + x_2^2), \\ \dot{x}_2 &= x_1 + \alpha x_2 - x_2(x_1^2 + x_2^2). \end{cases} \tag{6.1.1}$$

This system has equilibrium $x_1 = x_2 = 0$ for all $\alpha$ and its Jacobian matrix is equal to

$$A = \begin{pmatrix} \alpha & -1 \\ 1 & \alpha \end{pmatrix} \tag{6.1.2}$$

The matrix $A$ has eigenvalues $\lambda_{1,2} = \alpha \pm i$ and therefore satisfies the simple condition for Hopf bifurcation given in Definition 1.7 for $\alpha = 0$. Introducing the variable $z = x_1 + ix_2$ we get the equation:

$$\dot{z} = (\alpha + i)z - z|z|^2, \;\; z \in \mathbb{C}. \tag{6.1.3}$$

Setting $z = \rho e^{i\varphi}$, we get the polar form of system (6.1.1):

$$\begin{cases} \dot{\rho} &= \rho(\alpha - \rho^2), \\ \dot{\varphi} &= 1. \end{cases} \tag{6.1.4}$$

We have an equilibrium at $\rho = 0$ for all $\alpha$, we will look at the phase portraits for $\alpha < 0$,$\alpha = 0$ and $\alpha > 0$ (see Figure 1):
**Case 1** ($\alpha < 0$):
In this case $\dot{\rho} < 0$ so all solutions of system (6.1.4) will converge towards the equilibrium at $\rho = 0$ and we therefore have a linearly stable equilibrium.
**Case 2** ($\alpha = 0$):
The system (6.1.4) in this case will become:

$$\begin{cases} \dot{\rho} &= -\rho^3, \\ \dot{\varphi} &= 1. \end{cases} \tag{6.1.5}$$

Again we have $\dot{\rho} < 0$ for $\rho > 0$, so every solution starting at some point $x_0 \in \mathbb{R}^2$ will converge to the equilibrium $\rho = 0$, but no longer linearly. Nevertheless the equilibrium is still stable.
**Case 3** ($\alpha > 0$):
For this case we have a periodic solution if $\rho = \sqrt{\alpha}$. If $\rho < \sqrt{\alpha}$ we have $\dot{\rho} > 0$ so solutions starting there will tend towards the periodic solution and the equilibrium at $\rho = 0$ is unstable. If $\rho > \sqrt{\alpha}$ we have $\dot{\rho} < 0$ and solutions starting here will also tend towards the periodic solution.

So we again see there is bifurcation with bifurcation value $\alpha = 0$, because the phase portraits of $\alpha < 0$ and $\alpha > 0$ are different. The system (6.1.1) is one of the normal forms of Hopf bifurcation, every system that is locally topologically equivalent to this system demonstrate a **supercritical** Hopf bifurcation. The other normal form corresponding to **subcritical** Hopf bifurcation is given by:

$$\begin{cases} \dot{x}_1 &= \alpha x_1 - x_2 + x_1(x_1^2 + x_2^2), \\ \dot{x}_2 &= x_1 + \alpha x_2 + x_2(x_1^2 + x_2^2), \end{cases} \tag{6.1.6}$$

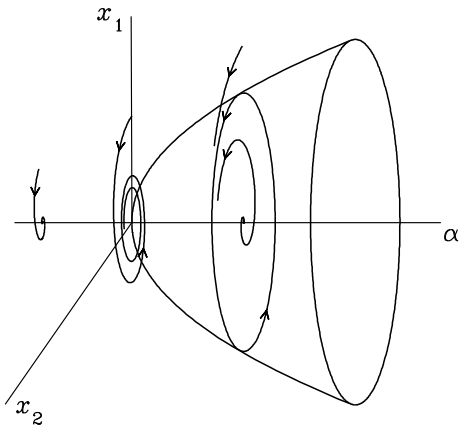



Figure 6.1: Supercritical Hopf bifurcation

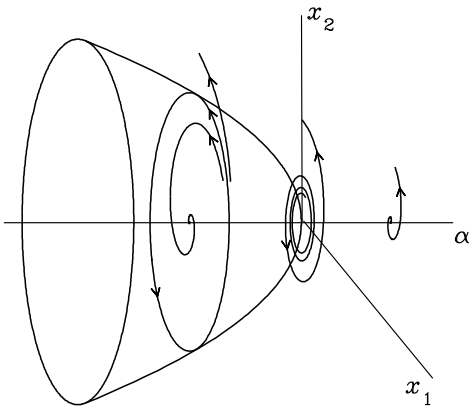



Figure 6.2: Subcritical Hopf Bifurcation

Changing this system into its polar form like we did with (6.1.1) we get the system:

$$\begin{cases} \dot{\rho} &= \rho(\alpha+\rho^2), \\ \dot{\varphi} &= 1. \end{cases} \tag{6.1.7}$$

There is again an equilibrium at $\rho = 0$ for all $\alpha$, we will again look at the different values $\alpha$ can have:
**Case 1** ($\alpha > 0$):
In this case we get $\dot{\rho} > 0$ for all values of $\rho$ so solutions will converge away from the equilibrium at $\rho = 0$ so this equilibrium is unstable.
**Case 2** ($\alpha = 0$):
In this case we have the system:

$$\begin{cases} \dot{\rho} &= \rho^3, \\ \dot{\varphi} &= 1. \end{cases} \tag{6.1.8}$$

So again $\dot{\rho} > 0$ for all $\rho > 0$ and therefore the equilibrium at $\rho = 0$ is still unstable.
**Case 3** ($\alpha < 0$):
Similar to case 3 in the supercritical system we have a periodic solution at $\rho = \sqrt{\alpha}$. For $\rho < \sqrt{\alpha}$ we get $\dot{\rho} < 0$ so solutions tend towards the equilibrium at $\rho = 0$ and if $\rho > \sqrt{\alpha}$ we have $\dot{\rho} > 0$ so it will converge away from the periodic solution.

These normal forms can be found in [5]. Now using the normal forms we can formulate the following theorem [6]:

**Theorem 6.1** (Topological normal form for Hopf bifurcation)**.** *Consider the following two dimensional system with one parameter:*

$$\dot{x} = f(x,\alpha), \quad x \in \mathbb{R}^2, \alpha \in \mathbb{R}, \tag{6.1.9}$$

*where $f$ is smooth, and suppose this system has the equilibrium $x=0$ for all sufficiently small $|\alpha|$ with eigenvalues:*

$$\lambda_{1,2} = \mu(\alpha) \pm i\omega(\alpha) \tag{6.1.10}$$

*with $\mu(0)=0, \omega(0)=\omega_0>0$. Further assume that*

(AH.1) *$l_1(0) \neq 0$, where $l_1$ is the first Lyapunov coefficient;*

(AH.2) $\mu'(0) \neq 0$

*Then system (6.1.9) is locally topologically equivalent near the origin to the following system:*

$$\begin{pmatrix} \dot{\xi}_1 \\ \dot{\xi}_2 \end{pmatrix} = \begin{pmatrix} \beta & -1 \\ 1 & \beta \end{pmatrix} \begin{pmatrix} \xi_1 \\ \xi_2 \end{pmatrix}) + \sigma(\xi_1^2+\xi_2^2) \begin{pmatrix} \xi_1 \\ \xi_2 \end{pmatrix}, \tag{6.1.11}$$

*where $\sigma = \operatorname{sign} l_1(0) = \pm 1$.*

We will explain what the Lyapunov coefficient mentioned in the previous theorem is when we defined Hopf bifurcation in $n$ dimensions.

# 7 Hopf bifurcation in $n$ dimensions

To study Hopf bifurcation in systems with dimension higher then 2 we will need a way to reduce the dimension of the given system near equilibria. In order to do this we will need the center manifold theorem.

## 7.1 The Center manifold theorem and reduction principle

Consider the following $n$-dimensional differential equation:

$$\dot{x} = f(x), \quad x \in \mathbb{R}^n \tag{7.1.1}$$

Where $f$ is sufficiently smooth, $f(0)=0$. Then (7.1.1) has equilibrium at $x_0=0$. Let $A$ be the Jacobian matrix evaluated at $x_0$ with eigenvalues $\lambda_1, \lambda_2, ..., \lambda_n$. Denote by $n_+, n_0$ and $n_-$ the number of eigenvalues having $\Re(\lambda) > 0$, $\Re(\lambda)=0$ and $\Re(\lambda)<0$, respectively. The eigenvalues $n_0$ are often called **critical**. Denote by $T^c$ the union of generalized eigenspaces of the $n_0$ eigenvalues. Lastly, let $\varphi^t$ denote the flow of (7.1.1), see Definition 1.3. Now we can formulate the following theorem:

**Theorem 7.1** (Center manifold theorem)**.** *There is a locally defined smooth $n_0$ dimensional invariant manifold $W^c_{loc}(0)$ of (7.1.1) that is tangent to $T^c$ at $x=0$. There exists a neighbourhood $U$ of $x_0=0$ such that if $\varphi^t x \in U$ for all $t \geq 0 (t \leq 0)$, then $\varphi^t x \to W^c_{loc}(0)$ when $t \to \infty (t \to -\infty)$.*

**Definition 10** (Center manifold)**.** *We call $W^c_{loc}(0)$ as defined in the previous theorem the **Center manifold***

Note that in the case that there is Hopf bifurcation we have $n_0=2$ so there will be a center manifold of dimension 2, so we can reduce the differential equation to 2 dimensions and use the theory from the 2-dimensional case to define the normal form for n dimensions.

Going back to system (7.1.1) we will first split the equation into an $n_0$ dimensional equation and another differential equation having dimension $n_+ + n_-$. We obviously require $n_0 \neq 0$, since otherwise the equilibrium is **hyperbolic** and does not bifurcate. Using a basis in $T^c$ and its $A$-invariant complement (**eigenbasis**), we can write (7.1.1) as follows:

$$\begin{cases} \dot{u} &= Bu + g(u,v), \\ \dot{v} &= Cv + h(u,v), \end{cases} \tag{7.1.2}$$

where $u \in \mathbb{R}^{n_0}, v \in \mathbb{R}^{n_+ + n_-}$, $B$ a $n_0 \times n_0$ dimensional matrix having eigenvalues on the imaginary axis and $C$ an $(n_+ + n_-) \times (n_+ + n_-)$ dimensional matrix with no eigenvalues on the imaginary axis. The functions $g, h$ are nonlinear. The center manifold $W^c$ of this system can be represented locally as the following graph:

$$W^c = \{(u,v) | v = V(u)\} \tag{7.1.3}$$

Where $V : \mathbb{R}^n_0 \to \mathbb{R}^n_+ + n_-$ is smooth. Now we are able to reduce the differential equation to $n_0$ dimensions using the following theorem:

**Theorem 7.2** (Reduction Principle)**.** *System (7.1.2) is locally topologically equivalent near the origin to the following system:*

$$\begin{cases} \dot{u} &= Bu + g(u, V(u)), \\ \dot{v} &= Cv, \end{cases} \tag{7.1.4}$$

Because the equations for $u$ and $v$ are uncoupled the behaviour of the system on the center manifold is only determined by the first equation, so for Hopf bifurcation we can use the theory for the 2-dimensional case to handle the $n$-dimensional case also.

The equation for $v$ can be replaced by the equations of a **standard saddle**:

$$\begin{cases} \dot{v} &= -v, \\ \dot{w} &= w, \end{cases} \tag{7.1.5}$$

Where $(v,w) \in \mathbb{R}^{n_-} \times \mathbb{R}^{n_+}$. This leads us to the following system of equations:

$$\begin{cases} \dot{u} &= Bu + g(u, V(u)), \\ \dot{v} &= -v, \\ \dot{w} &= w, \end{cases} \tag{7.1.6}$$

## 7.2 Center manifolds in parameter-dependent systems

Now that we know how to reduce differential equations using the center manifold theorem we will look at parameter dependent differential equations next:

$$\dot{x} = f(x, \alpha), \quad x \in \mathbb{R}^n, \alpha \in \mathbb{R}. \tag{7.2.1}$$

Where for $\alpha = 0$ there is a non-hyperbolic equilibrium $x = 0$ with $n_0, n_+$ and $n_-$ the eigenvalues $\lambda$ with $\Re(\lambda) = 0$, $\Re(\lambda) > 0$ and $\Re(\lambda) < 0$ respectively. We will look at the following extended system:

$$\begin{cases} \dot{\alpha} &= 0, \\ \dot{x} &= f(x, \alpha). \end{cases} \tag{7.2.2}$$

This system has an equilibrium at $(\alpha, x) = (0,0)$ with Jacobian matrix:

$$J = \begin{pmatrix} 0 & 0 \\ f_\alpha(0,0) & f_x(0,0) \end{pmatrix} \tag{7.2.3}$$

Having $n_0 + 1$ eigenvalues on the imaginary axis and $n - n_0$ with negative or positive real parts. If we now apply the center manifold theorem to system (7.2.2) we find a center manifold $W^c \subset \mathbb{R} \times \mathbb{R}^n$, $\dim(W^c) = n_0 + 1$. Because $\dot{\alpha} = 0$ the hyperplanes $\Pi_{\alpha_0} = \{(\alpha, x) | \alpha = \alpha_0\}$ are invariant with respect to (7.2.2) so we can split $W^c$ into the following invariant manifolds:

$$W^c_\alpha = W^c \cap \Pi_\alpha \tag{7.2.4}$$

Which leads to the following lemma:

**Lemma 7.3.** System (7.2.2) has a parameter-dependent local invariant manifold $W^c_\alpha$.

Note that for $\alpha = 0$ we get the center manifold $W^c_0$ from the last section. So we can also reduce the systems with parameters to the suspension of restriction to the manifold by the standard saddle.

## 7.3 Normal form of Hopf bifurcation in $n$ dimensions

Now that we know how to reduce the dimension of systems, we can introduce the normal form for Hopf bifurcation in $n$ dimensions, which is defined as the following system:

$$\begin{cases} \dot{x}_1 &= \alpha x_1 - x_2 \pm x_1(x_1^2 + x_2^2), \\ \dot{x}_2 &= x_1 + \alpha x_2 \pm x_2(x_1^2 + x_2^2), \\ \dot{x}^s &= -x^s, \\ \dot{x}^u &= x^u. \end{cases} \tag{7.3.1}$$

Where for $n_s$ the amount of eigenvalues with $\Re(\lambda) < 0$ and $n_u$ the amount of eigenvalues with $\Re(\lambda) > 0$ we have $x^s \in \mathbb{R}^{n_s}$ and $x^u \in \mathbb{R}^{n_u}$. When the nondegeneracy conditions (AH.1) and (AH.2) from Theorem 1.3 hold we will get that an $n$ dimensional system is locally topologically equivalent to one of the systems defined above.

## 7.4 First Lyapunov coefficient

In order to calculate the first Lyaponov coefficient at $\alpha = 0$ of system (4.0.1), we have to write the function $f$ as its Taylor expansion:

$$f(x,0) = Df(0)x + \frac{1}{2}D^2f^0(x,x) + \frac{1}{6}D^3f^0(x,x,x) + O(\|x\|^4), \tag{7.4.1}$$

where the second and third differentials of $f$ are defined as:

$$\begin{aligned} D^2f_j^0(x,y) &= \sum_{k,j=1}^{n} \frac{\partial^2 f_j(\xi,0)}{\partial\xi_k\partial\xi_l}\bigg|_{\xi=0} x_k y_l, \\ D^3f_j^0(x,y,z) &= \sum_{k,l,m=1}^{n} \frac{\partial^3 f_j(\xi,0)}{\partial\xi_k\partial\xi_l\partial\xi_m}\bigg|_{\xi=0} x_k y_l z_m, \end{aligned} \tag{7.4.2}$$

where $j = 1,2,\ldots,n$. Now let $q \in \mathbb{C}^n$ be a complex right eigenvector of $A = Df(0)$ corresponding to the eigenvalue $i\omega_0$, and let $p^T \in \mathbb{C}^n$ be left eigenvector of $A = Df(0)$ corresponding to the same eigenvalue $i\omega_0$. Assume that $pq = 1$. Then the first Lyapunov coefficient is equal to

$$l_1(0) = \frac{1}{\omega_0}\Re(c_1), \tag{7.4.3}$$

where $c_1$ is given by

$$c_1 = \frac{1}{2}p\left[D^3f^0(q,q,\bar{q}) + 2D^2f^0(q,-A_0^{-1}D^2f^0(q,\bar{q})) + D^2f^0(\bar{q},D^2f^0(\bar{q},(2i\omega_0 I_n - A_0)^{-1}D^2f^0(q,q))\right]. \tag{7.4.4}$$

A derivation of this formula can be found in [6].

**Example 7.1** (Lyapunov coefficient)**.** Consider the 2-dimensional system $\dot{x} = f(x,\alpha)$ with

$$x = \begin{pmatrix} u \\ v \end{pmatrix}, \quad f(x,0) = \begin{pmatrix} 0 & -\omega_0 \\ \omega_0 & 0 \end{pmatrix}\begin{pmatrix} u \\ v \end{pmatrix} + \begin{pmatrix} P(u,v) \\ Q(u,v) \end{pmatrix}, \tag{7.4.5}$$

Using eigenvalues $q = p = \frac{1}{\sqrt{2}}\begin{pmatrix} 1 \\ -i \end{pmatrix}$ we get the following formula for the first Lyapunov coefficient:

$$l_1(0) = \frac{1}{8\omega_0}(P_{uuu} + P_{uvv} + Q_{uuv} + Q_{vvv}) + \frac{1}{8\omega_0^2}(P_{uv}(P_{uu} + P_{vv}) - Q_{uv}(Q_{uu} + Q_{vv}) - P_{uu}Q_{uu} + P_{vv}Q_{vv}) \tag{7.4.6}$$

Here the lower indices mean the partial derivatives evaluated at $x = 0$.

Using this formula to calculate the Lyapunov coefficients of the normal forms of Hopf bifurcation we get the value $l_1(0) = -2$ for the supercritical normal form defined in (6.1.1) and $l_1(0) = 2$ for the subcritical normal form defined in (6.1.6). We can determine whether an equilibrium satisfies the conditions for Hopf bifurcation using the sign of the Lyapunov coefficient. If sign $l_1(0) = 1$ for a certain system, it is locally topologically equivalent to the normal form defined in (6.1.6) and we will call it **subcritical**. If sign $l_1(0) = -1$ it is locally topologically equivalent to the normal form defined in (6.1.1) and we will call it **supercritical**.

# Part III
# Hopf bifurcation in Delayed Differential Equations

In this Part we will study differential equations with a time delay as a parameter, which we will abbreviate as DDE's, to see why they are useful we will first look at an example taken from [7]:

**Example 7.2** (Linear delay equation)**.** Starting with a model for population size, let $N(t)$ denote the density of adults at time $t$. Because it takes a while for kids to grow up we will denote the juvenile period to be $h$ units of time. Assume the rate at which adults produce offspring to be $\alpha$, $\mu$ is the probability of dying per unit of time and $\rho$ is the probability a child will survive the juvenile period. When we set $r = \alpha\rho$ the model can be described by the following differential equation:

$$\dot{N}(t) = -\mu N(t) + rN(t-h), \tag{7.4.7}$$

The $h$ is this equation is called the time delay.

There are many biological reasons to include a time delay in a model. In the example above the time delay is used to model the time it takes for a kid to become an adult but there are many other reasons to include a time delay. In epidemiological models, in particular, there are many time delays: It can be used to include a temporary immunity in computer virus models, an infectious period can be included using time delays, and we can instead of a separate exposed population include a time delay in the incidence rate which provides a more natural way to model the exposed population. In this Part we will look at the theory behind delayed differential equations. We will first define a delayed differential equation, then we discuss what equilibria are and what it means for an equilibrium to be linearly stable. We will also look at the conditions for Hopf bifurcation in these systems and describe differences and similarities DDE's and ODE's have.

## 8 Delayed differential equations

In general we will look at systems of the following form:

$$\dot{x}(t) = f(x(t), x(t-\tau_1), x(t-\tau_2), ..., x(t-\tau_m)), \;\; x(t) \in \mathbb{R}^n, \tag{8.0.1}$$

where $f : \mathbb{R}^{(m+1)n} \to \mathbb{R}^n$ smooth and $0 =: \tau_0 < \tau_1 < ... < \tau_m =: h < \infty$. For ordinary differential equations it was sufficient for a solution to have a single starting point, now for $t = 0$ we see that we need a function which tells us what the solution was before $t = 0$ on an interval of length $h$. We will call this the **initial data** $\phi \in C([-h, 0], \mathbb{R}^n)$. The **global solution** for this system will be an element $x \in C([-h, 0], \mathbb{R}^n) \cap C^1([0, \infty], \mathbb{R}^n)$ and for this solution at a time $t \geq 0$ the **history** $x_t \in C([-h, 0], \mathbb{R}^n)$, $x_t(\theta) := x(t + \theta)$. The initial-value problem for the DDE then becomes:

$$\begin{cases} \dot{x}(t) &= F(x_t), t \geq 0, \\ x_0 &= \phi. \end{cases} \tag{8.0.2}$$

Here $F : C([-h, 0], \mathbb{R}^n) \to \mathbb{R}^n, F(\phi) = f(\phi(0), \phi(-\tau_1), \phi(-\tau_2), ..., \phi(-\tau_m))$ is smooth. For convenience we will denote $X = C([-h, 0], \mathbb{R}^n)$ for the Banach-space of continuous functions endowed with the supremum-norm:

$$\|\phi\| := \sup\{\|\phi(x)\| \mid -h \geq x \geq 0\}. \tag{8.0.3}$$

When $F$ is globally Lipschitz with respect to this norm we have a unique global solution $x = x(., \phi)$, for any $\phi \in X$, which is continuous with respect to $\phi$. If $F$ is only locally Lipschitz the solution is only guaranteed to exist on a small interval. In the ordinary differential equations case we had orbits of solutions uniquely determined by a single point, so it made sense to have a state space made up out of points. In the DDE case we have an orbit starting at a time $t$ uniquely determined by a given function defined on $[t - h, t]$ so the state space will also consist of functions defined on the interval $[-h, 0]$. In the ODE case we had a state being defined by an $n$ dimensional point for an $n$ dimensional system, here in the DDE case we need a function defined on an interval so an infinite amount of points. For this reason we call a system of delayed differential equations an infinite dimensional dynamical system.

For ordinary differential equations, we had a function called the flow assigning to an initial point a point along the solution at time $t$. In the DDE case, we need a mapping defined on the space of functions which gives the state at a later time, provided the initial state is given. For an initial state $\phi \in C([-h, 0], \mathbb{R}^n)$ let $S(t)$ denote the operator that assigns to it a state $t$ units of time later. This operator satisfies the following:

(i) $S(0) = \mathrm{id}$,

(ii) $S(t+s) = S(t)S(s)$, for all $t, s \geq 0$.

The second property is due to the uniqueness of the solutions. The family $\{S(t)\}_{t\geq 0}$ forms a **semigroup** of operators.

**Definition 11** (Strongly continuous semigroup / $C_0$-semigroup)**.** *Let $X$ be a complex Banach space and let, for each $t \geq 0$, $T(t) : X \to X$ be a bounded linear operator. Then the family $\{T(t)\}_{t\geq 0}$ is called a **strongly continuous semigroup**, whenever the following properties hold:*

*(i)* $T(0) = id$,

*(ii)* $T(t)T(s) = T(t+s)$, *for* $t, s \geq 0$,

*(iii) for all* $x \in X$, $\|T(t)x - x\| \to 0$ *as* $t \downarrow 0$.

Using semigroups we can define the **(forward) orbit** for a given initial state $\varphi$ to be the subset $\{S(t)\varphi | t \geq 0\} \subset X$ and the collection of all these orbits could again be called the **phase portrait**. Note however, that orbits of DDEs can overlap.

# 9 Equilibria and stability

In the case of delayed differential equations the equilibria will again be constant solutions, so for a system defined in (8.0.1) a constant solution $x(t) = x^* \in \mathbb{R}^n$ is called an equilibrium if:

$$f(x^*, x^*, x^*, ..., x^*) = 0. \tag{9.0.1}$$

The linearized system of (8.0.1) is given by:

$$\dot{y}(t) = A_0 y(t) + \sum_{j=1}^{m} A_j y(t - \tau_j), \quad y(t) \in \mathbb{R}^n, \quad A_j = D_j f(x^*, x^*, x^*, ..., x^*), \quad j = 0, 1, ..., m. \tag{9.0.2}$$

Solutions to this linear DDE define a $C_0$-semigroup $T(t)$ on $X = C([-h, 0], \mathbb{R}^n)$. The **characteristic matrix** can now be introduced as

$$\Delta(\lambda) = \lambda \mathbb{I}_n - A_0 - \sum_{j=1}^{m} A_j e^{-\lambda \tau_j}. \tag{9.0.3}$$

with the characteristic equation $\det \Delta(\lambda) = 0$. Its roots are called the eigenvalues of $x^*$. Just like in the ODE case the equilibrium $x^*$ is asymptotically stable if all eigenvalues satisfy $\Re(\lambda) < 0$, and unstable if there is at least one eigenvalue with $\Re(\lambda) > 0$ [7]. Notice that, unlike in the ODE case where we had the eigenvalues defined as the roots of a polynomial, we now have them as the roots of a quasi-polynomial with exponential functions in its coefficients. So where in the ODE case we had maximum $n$ eigenvalues for an $n$ dimensional system we now in the DDE case have an infinite number of eigenvalues regardless of the dimension.

# 10 Hopf bifurcation in DDE's

## 10.1 Center manifold theorem and normal form

Now consider a DDE with $m$ delays for $x(t) \in \mathbb{R}^n$ and having a parameter $\alpha \in \mathbb{R}$:

$$\dot{x}(t) = f(x(t), x(t - \tau_1), x(t - \tau_2), ..., x(t - \tau_m), \alpha), \tag{10.1.1}$$

With $f : \mathbb{R}^{(m+1)n} \times \mathbb{R} \to \mathbb{R}^n$ smooth and $0 = \tau_0 < \tau_1, ..., \tau_m = h$. Just like in the ODE case we assume there is an equilibrium at $x = 0$ when $\alpha = 0$. Let $\lambda_j \in \mathbb{C}$ be the roots of the characteristic equation

$$0 = \det \left( \lambda \mathbb{I}_n - A_0 - \sum_{j=1}^{m} A_j e^{-\lambda \tau_j} \right), \quad A_j = D_j f(0, 0), j = 0, 1, .., m. \tag{10.1.2}$$

We again write $n_0, n_+$ and $n_-$ for the numbers of eigenvalues satisfying $\Re(\lambda) = 0$, $\Re(\lambda) > 0$, and $\Re(\lambda) = 0$, respectively. The center manifold in the DDE case will be mostly the same as the one in the ODE case, only difference being that instead of the flow we use the solution operator defined in the previous section.

Let $W^c_\alpha \subset X$ be the center manifold, then the restriction of the solution operator $S_\alpha(t)$ to $W^c_\alpha$ is locally generated by a smooth ODE:

$$\dot{\xi} = g(\xi, \alpha), \xi \in \mathbb{R}^{n_0}, \alpha \in \mathbb{R} \tag{10.1.3}$$

For Hopf bifurcation the smooth normal form on $W^c_\alpha$ is:

$$\dot{z} = (\mu(\alpha) + i\omega(\alpha))z + c(\alpha)z|z^2| + O(|z|^4), \tag{10.1.4}$$

here $\mu(0) = 0, \omega(0) = \omega_0 > 0$ and $c(0) = c_1$. In order for (10.1.4) to be locally topologically equivalent to

$$\dot{w} = (\beta + i)w + \sigma w|w^2|, \tag{10.1.5}$$

where $\sigma = \pm 1$, we need the first Lyapunov coefficient to be nonzero, $l_1 := \frac{1}{\omega_0}\mathrm{Re}(c_1) \neq 0$. Then $\sigma = \text{sign } l_1$.

So the conditions for Hopf bifurcation are the same as in the ODE case. For system (10.1.1) we need eigenvalues $\lambda(\alpha) = \mu(\alpha) + i\omega(\alpha)$ with $\mu(0) = 0$, $\omega(0) = \omega_0 > 0$. In order for this system to be locally topologically equivalent to the normal form we need $\mu'(0) \neq 0$ and $l_1(0) \neq 0$.
Instead of the parameter $\alpha$ we could also let the center manifold depend on one of the delays $\tau_j$ in order to determine if there is a bifurcation with respect to that time delay.

## 10.2 Computation of the first Lyapunov coefficient for DDE's

Now again consider a DDE as defined in (10.1.1), and assume there exist roots $\lambda_{1,2} = \pm i\omega_0$ of the characteristic equation defined in (10.1.2). Just as in the ODE case we have to calculate the first Lyapunov coefficient in DDE's, and we will now give the corresponding formula. Choose $q, p^T \in \mathbb{C}^n$ such that:

$$\Delta(i\omega_0)q = 0,\ p\Delta(i\omega_0) = 0,\ p\Delta'(i\omega_0)q = 1. \tag{10.2.1}$$

Then the eigenfunctions will be given by:

$$\phi(\theta) = e^{i\omega_0\theta}q, \tag{10.2.2}$$

Now using sun-star calculus [7] one can derive the following formula for the first Lyapunov coefficient:

$$l_1 = \frac{1}{\omega_0}\mathrm{Re}(c_1), \tag{10.2.3}$$

where $c_1$ equals:

$$c_1 = \frac{1}{2}p\left[D^2F(0)(\overline{\phi}, e^{2i\omega_0\theta}\Delta(2i\omega_0)^{-1}D^2F(0)(\phi,\phi) + 2D^2F(0)(\phi,\Delta(0)^{-1}D^2F(0)(\phi,\overline{\phi})) + D^3F(0)(\phi,\phi,\overline{\phi})\right] \tag{10.2.4}$$

Where $F : C([-h,0],\mathbb{R}^n) \to \mathbb{R}^n, F(\phi) = f(\phi(0), \phi(-\tau_1), \phi(-\tau_2), ..., \phi(-\tau_m))$. So the second differentials of $F$ become:

$$\begin{aligned} D^2F(0)(\phi,\phi) &= D^2f^0(\Phi,\Phi), \\ D^2F(0)(\phi,\overline{\phi}) &= D^2f^0(\Phi,\overline{\Phi}). \end{aligned} \tag{10.2.5}$$

And the third differential is:

$$D^3F(0)(\phi,\phi,\overline{\phi}) = D^3f^0(\Phi,\Phi,\overline{\Phi}), \tag{10.2.6}$$

where $\Phi = (\phi(0), \phi(-\tau_1), ..., \phi(-\tau_m))$, and for $Q, P, R \in \mathbb{R}^{n(m+1)}$ with components $q^j_k, p^j_k, r^j_k$ the (multi-)linear forms $D^2f^0(Q,P)$ and $D^3f^0(Q,P,R)$ are defined as:

$$\begin{aligned} D^2f^0(Q,P): &= \sum_{k_1,k_2=1}^{n}\sum_{j_1,j_2=0}^{m}\frac{\partial^2 f(0)}{\partial x^{j_1}_{k_1}\partial x^{j_2}_{k_2}}q^{j_1}_{k_1}p^{j_2}_{k_2}, \\ D^3f^0(Q,P,R): &= \sum_{k_1,k_2,k_3=1}^{n}\sum_{j_1,j_2,j_3=0}^{m}\frac{\partial^3 f(0)}{\partial x^{j_1}_{k_1}\partial x^{j_2}_{k_2}\partial x^{j_3}_{k_3}}q^{j_1}_{k_1}p^{j_2}_{k_2}r^{j_3}_{k_3}. \end{aligned} \tag{10.2.7}$$

A derivation of this formula can be found in [7].

**Example 10.1.** Consider the following scalar nonlinear DDE:

$$\dot{x}(t) = f(x(t), x(t-\tau), \mu), \quad f(x(t), x(t-\tau), \mu) = (-\frac{\pi}{2} + \mu)x(t-\tau)(1+x(t)). \tag{10.2.8}$$

We see that $f(0, x(t-\tau), \mu) = 0$ for all $\mu$ so there is an equilibrium at $x^* = 0$, the Jacobian matrix of $f$ evaluated at $x^*$ is:

$$A = (A_0, A_1) = Df(0,0,\mu) = \left(-\frac{\pi}{2} + \mu, \left(-\frac{\pi}{2} + \mu\right)(1+x)\right)\Big|_{x=x^*} = \left(0, -\frac{\pi}{2} + \mu\right). \tag{10.2.9}$$

So according to (9.0.3) the characteristic matrix is scalar and is equal to:

$$\Delta(\lambda) = \lambda - \left(-\frac{\pi}{2} + \mu\right) e^{-\lambda\tau}. \tag{10.2.10}$$

To prove that system (10.2.8) undergoes a Hopf bifurcation we will need a pair of purely imaginary roots to this equation at the critical parameter value $\tau_0$, the speed of the eigenvalue $\mathrm{Re}(\frac{d\lambda}{d\tau})$ needs to be non-zero when crossing the critical parameter value and the first Lyapunov coefficient needs to be non-zero. We will start by substituting $\lambda = i\omega_0$ into the characteristic matrix and set it equal to 0:

$$\left(-\frac{\pi}{2} + \mu\right)(\cos(\omega_0\tau) - i\sin(\omega_0\tau)) = i\omega_0. \tag{10.2.11}$$

Splitting this equation into its real and imaginary components we get the following system:

$$\begin{cases} \sin(\omega_0\tau) &= \dfrac{\omega_0}{-(\frac{\pi}{2} + \mu)}, \\ \cos(\omega_0\tau) &= 0. \end{cases} \tag{10.2.12}$$

The second equation will give us the critical parameter values $\tau_k$ for $k \in \mathbb{Z}$:

$$\tau_k = \frac{\pi}{2\omega_0} + \frac{k\pi}{\omega_0}. \tag{10.2.13}$$

In order to calculate $\omega_0$ we can square both equations and add them afterwards:

$$1 = \sin^2(\omega_0\tau) + \cos^2(\omega_0\tau) = \frac{\omega_0^2}{(\frac{\pi}{2} + \mu)^2} \tag{10.2.14}$$

Which gives $\omega_0 = \pm(\frac{\pi}{2} + \mu)$. We will now prove that $\frac{d(\Re(\lambda))}{d\tau}|_{\tau=\tau_0} \neq 0$, to do this we will calculate the derivative of (10.2.10) and find an expression for $(\frac{d\lambda}{d\tau})^{-1}$:

$$0 = \frac{d\lambda}{d\tau} + \tau\left(-\frac{\pi}{2} + \mu\right)e^{-\lambda\tau}\frac{d\lambda}{d\tau} + \left(-\frac{\pi}{2} + \mu\right)e^{-\lambda\tau}\lambda \tag{10.2.15}$$

$$\left(\frac{d\lambda}{d\tau}\right)^{-1} = -\left(\frac{\tau}{\lambda} + \frac{1}{(-\frac{\pi}{2} + \mu)\lambda e^{-\lambda\tau}}\right) \tag{10.2.16}$$

Therefore we have:

$$\mathrm{sign}\left\{\frac{d(\Re(\lambda))}{d\tau}\right\}_{\tau=\tau_0} = \mathrm{sign}\left\{\Re\left(\frac{d\lambda}{d\tau}\right)^{-1}\right\}_{\tau=\tau_0, \lambda=i\omega_0} = \mathrm{sign}\left\{-\frac{1}{(\frac{\pi}{2} + \mu)^2}\right\} = -1. \tag{10.2.17}$$

The only thing left to prove is that the first Lyapunov coefficient is non-zero, for this we need to find $p, q \in \mathbb{C}$ such that:

$$\Delta(i\omega_0)q = 0, p\Delta(i\omega_0) = 0, p\Delta'(i\omega_0)q = 1. \tag{10.2.18}$$

For this part we will let $\mu = 0$, the critical parameter value then becomes $\tau_0 = 1$ and $\omega_0 = \frac{\pi}{2}$. Because $\Delta(i\omega_0) = 0$ we can choose $q = 1$, The derivative of the characteristic equation is:

$$\Delta'(\lambda) = 1 - \frac{\pi}{2}e^{-\lambda} \tag{10.2.19}$$

So the last equation of (10.2.18) becomes:

$$p(1 - \frac{\pi}{2}e^{-\lambda})|_{\lambda=i\omega_0}q = p(1 + i\frac{\pi}{2}) = 1. \tag{10.2.20}$$

So we can set $p = \frac{2}{2+i\pi}$. The eigenfunction $\phi$ defined in (10.2.2) is equal to:

$$\phi(\theta) = e^{i\frac{\pi}{2}\theta}. \tag{10.2.21}$$

Take $Q = (q^0, q^1)$, $P = (p^0, p^1)$ and $R = (r^0, r^1)$ then the derivative of $f$ at 0 are:

$$\begin{aligned} D^2 f^0(Q,P) &= \sum_{j_1,j_2=0}^{1} \frac{\partial^2 f(0)}{\partial x^{j_1} \partial x^{j_2}} q^{j_1} p^{j_2} = 0 \cdot q^0 p^0 - \frac{\pi}{2} q^0 p^1 - \frac{\pi}{2} q^1 p^0 + 0 \cdot q^1 p^1 \\ &= -\frac{\pi}{2}(q^0 p^1 + q^1 p^0), \\ D^3 f^0(Q,P,R) &= 0. \end{aligned} \tag{10.2.22}$$

So the derivatives of $F$ become:

$$\begin{aligned} D^2F(0)(\phi,\phi) &= D^2 f^0((\phi(0),\phi(-1)),(\phi(0),\phi(-1))) = -\frac{\pi}{2}(1 \cdot -i + -i \cdot 1) = \pi i, \\ D^2F(0)(\phi,\overline{\phi}) &= D^2 f^0((\phi(0),\phi(-1)),(\overline{\phi}(0),\overline{\phi}(-1))) = -\frac{\pi}{2}(1 \cdot -i + i \cdot 1) = 0. \end{aligned} \tag{10.2.23}$$

So $c_1$ will be equal to:

$$\begin{aligned} c_1 &= \frac{1}{2} p D^2F(0)(\overline{\phi}, e^{2i\omega_0\theta}\Delta(2i\omega_0)^{-1} D^2F(0)(\phi,\phi)) \\ &= \frac{1}{2+i\pi} D^2F(0)\left(\overline{\phi}, \frac{2i}{2i-1}\right) \\ &= \frac{1}{2+i\pi} D^2 f^0\left((1,i), \left(\frac{2i}{2i-1}, \frac{2i}{2i-1}\right)\right) \\ &= \frac{1}{4+\pi^2}\frac{\pi}{5}(2 - 3\pi - (6+\pi)i). \end{aligned} \tag{10.2.24}$$

So the first Lyapunov coefficient of (10.2.8) is equal to:

$$l_1 = \frac{1}{\omega_0}\mathrm{Re}(c_1) = \frac{2}{5}\frac{2-3\pi}{4+\pi^2} \approx -0.2141. \tag{10.2.25}$$

Since $l_1 < 0$ we have a supercritical Hopf bifurcation.

# 11 Comparison of ODE's and DDE's

We will now compare the results form ordinary differential equations and delayed differential equations. First we will look at the characteristic equation, in the ODE case we saw that we get the eigenvalues by calculating the roots of the characteristic equation which was just a polynomial. So for an $n$-dimensional system we get $n$ eigenvalues. The difference in the DDE case is that we get a quasi-polynomial with exponential functions as its coefficients, so we can get an infinite amount of eigenvalues. Because of the exponential terms we now get an infinite number of eigenvalues regardless of the dimension of the system.

The conditions for Hopf bifurcation are mostly the same for ODE's and DDE's, but because we have delayed terms in DDE's we get a function as an initial condition instead of a single point. In both cases the first Lyapunov coefficients are given by:

$$l_1(0) = \frac{1}{\omega_0}\mathrm{Re}(c_1) \tag{11.0.1}$$

The expressions for $c_1$ are also very similar:

- ODE:

$$c_1 = \frac{1}{2} p \left[D^3 f^0(q,q,\overline{q}) + 2D^2 f^0(q, -A_0^{-1} D^2 f^0(q,\overline{q})) + D^2 f^0(\overline{q}, D^2 f^0(\overline{q}, (2i\omega_0 I_n - A_0)^{-1} D^2 f^0(q,q))\right].$$

- DDE:

$$c_1 = \frac{1}{2} p [D^3F(0)(\phi,\phi,\overline{\phi}) + 2D^2F(0)(\phi, \Delta(0)^{-1} D^2F(0)(\phi,\overline{\phi})) + D^2F(0)(\overline{\phi}, e^{2i\omega_0\theta}\Delta(2i\omega_0)^{-1} D^2F(0)(\phi,\phi)]$$

Instead of the function working on a vector like in the ODE case, in the DDE case we have a separate function $F$ depending on $f$ and working on the eigenfunctions of the equilibrium instead of the eigenvectors. Other then that we see that the two expressions are very similar.

# 12 Numerical analysis of DDE's

DDE's of higher dimensions can be tedious and very labour intensive to analyse, we will therefore look at ways of studying these equations numerically. In this section we will show how to compute Hopf bifurcation critical parameters and its direction using the DDE-BIFTOOL package for MATLAB. We will also show how to simulate DDE´s in MATLAB.

## 12.1 DDE-BIFTOOL

DDE-BIFTOOL [8] is a package that can be used to analyse numerically bifurcations in systems of delayed differential equations. It can perform the following computations:

- Computing stability of equilibria using the roots to the characteristic equation;
- Continuation of of equilibria with respect to a single parameter;
- Continuation of Hopf and fold bifurcations of steady states in two parameters;
- Continuation of the periodic solution with respect to a single parameter starting at a Hopf point or manually set up periodic orbit;
- Approximation of Floquet multipliers for periodic orbits;
- Switching to secondary branches at a period doubling bifurcation or a branch point;
- Continuation of fold, torus and period doubling bifurcations in two parameters;
- Calculating normal form coefficients for Hopf and fold bifurcations.

We will illustrate some of these functionalities in the following example:

**Example 12.1.** We will use the example from the last section, namely the following differential equation:

$$\dot{x}(t) = f(x(t), x(t-\tau), \mu), \quad f(x(t), x(t-\tau), \mu) = (-\frac{\pi}{2} + \mu)x(t-\tau)(1+x(t)). \tag{12.1.1}$$

We start by creating a directory in the demo folder of DDE-BIFTOOL, we will be calling it example. In this directory we will create a file named `gen_sym_example.m` containing the following code:

```
%%Differential equation
%
%$$\dot x(t) = -(\pi/2+mu)*x(t-\tau)(1+x(t))
%
%Parameters are |mu| and |tau|

%%Add paths and load sym package in GNU Octave is used
clear
ddebiftoolpath='../../';
addpath(strcat(ddebiftoolpath,'ddebiftool'),...
strcat(ddebiftoolpath,'ddebiftool_extra_symbolic'));
if dde_isoctave()
    pkg load symbolic
end

%%Create parameter names as strings and define fixed parameters
parnames={'mu','tau'};

%%Create symbols for parameters, states and delays states
syms(parnames{:});
par=cell2sym(parnames);

%%Define system using symbolic algebra
syms X Xtau
dX_dt = (-pi/2+mu)*Xtau*(1+X);

%%Differentiate and generate code, exporting it to sym_example
[fstr,derivs]=dde_sym2funcs(...
    [dX_dt],...
    [X,Xtau],...
```

```
    par,...
    'filename','sym_example'...
);
```

The code will generate the system file, so we can start to analyse the system numerically. In order to do this we need to create a new file `example.m` in the same directory, containing the following code:

```
%%load DDE-BifTool into MATLAB path
clear
close all
ddebiftoolpath='../../';
addpath(strcat(ddebiftoolpath,'ddebiftool'),...
    strcat(ddebiftoolpath,'ddebiftool_extra_psol'),...
    strcat(ddebiftoolpath,'ddebiftool_extra_nmfm'),...
    strcat(ddebiftoolpath,'ddebiftool_utilities'));

format compact
set(groot,'defaultTextInterpreter','LaTeX');
%%Initial parameters and state
parnames={'mu','tau'};
cind=[parnames;num2cell(1:length(parnames))];
ind=struct(cind{:});
bounds = {'max_bound',[ind.tau 10],'max_step',[0,0.5],...
    'min_bound',[ind.tau 0]};
%%Set user-defined functions
%use the right-hand side and derivatives created via symbolic toolbox
funcs=set_symfuncs(@sym_example,'sys_tau',@()ind.tau);
```

Now we will create a file in order to set up the steady state to this differential equation and calculate its stability. According to the example the steady state is $x^* = 0$. We will use parameter values $\mu = 0, \tau = 0.5$. The steady state can be contructed using the function `dde_stst_create`, and we will compute the stability with the function `p_stabil`. We can set the minimal real part of the eigenvalues to a certain value in order to limit the amount of eigenvalues which are computed. Lastly we will plot the eigenvalues:

```
%% Construct steady-state point
mu=1;
stst=dde_stst_create('x',[0]);
stst.parameter(ind.mu)=mu;
stst.parameter(ind.tau)=0.5;
% Compute stability
method_stst=df_mthod(funcs,'stst');
method_stst.stability.minimal_real_part=-20;
stst.stability=p_stabil(funcs,stst,method_stst.stability);
% Plot eigenvalues
figure(1); clf;
plot(stst.stability.l1,'*')
title('Stability plot of stst')
xlabel('$\Re(\lambda)$')
ylabel('$\Im(\lambda)$')
stst.stability.l1
```

Executing this code for we get a plot for the eigenvalues in figure (12.1)

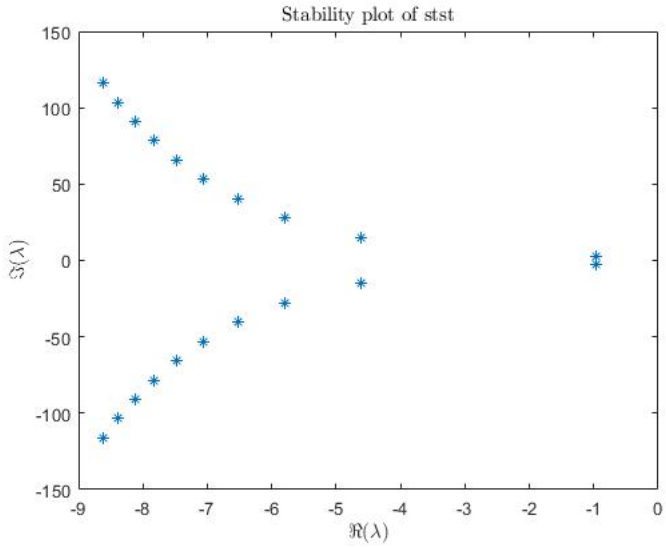



Figure 12.1: Eigenvalues of system (12.1.1) for equilibrium $x = 0$ with $\tau = 0.5$ and $\mu = 0$

Next we will initialize a steady state branch using the function `SetupStst`. This function will create a bunch of equilibria for different values of a certain parameter starting at the original steady state. We will continue the equilibria with respect to $\tau$, the branch is visible in figure (12.2):

```
%% Initialization of branch of steady-states
contpar=ind.tau;
steadystate_br=SetupStst(funcs,'x',stst.x,'parameter',stst.parameter,...
    'step',0.1,'contpar',contpar,'max_step',[contpar,0.3],bounds{:});
%% Continue steady-state branch
figure(2);clf;ax2=gca;
xlabel('$\tau$')
ylabel('$\mu$')
n_steps=40;
steadystate_br=br_contn(funcs,steadystate_br,n_steps,'plotaxis',ax2);
```

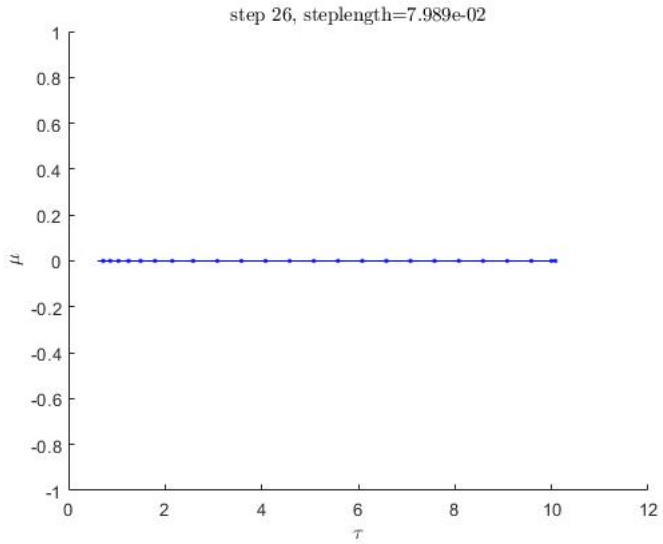



Figure 12.2: Steady state branch

Lastly we will use the `LocateSpecialPoints` function to detect Hopf bifurcation along the branch and the `GetStability` function to calculate the first Lyapunov coefficient when a bifurcation is detected:

```
%% Detect bifurcations on steady-state branch
[steadystate_br,¬,ind_hopf,bif1types]=LocateSpecialPoints(funcs,...
    steadystate_br);
nunst_eqs=GetStability(steadystate_br);
fprintf('near point %d\n',ind_hopf);
```

This code gives the following output:

```
>> examplehopf
StstCodimension1: calculate stability if not yet present
StstCodimension1: (provisional) 3 Hopf  detected.
br_insert: detected 1 of 3: hopf. Normalform:
    L1: -0.2141
br_insert: detected 2 of 3: hopf. Normalform:
    L1: -0.0720
br_insert: detected 3 of 3: hopf. Normalform:
    L1: -0.0412
near point 5
near point 16
near point 25
```

Inspecting the first occurence using `steadystate_br.point(5)` we see that there is a Hopf bifurcation at $\tau = 1$ with Lyapunov coefficient $L_1 = -0.2141$ like we got in the example in the previous section. In order to see if we have a supercritical or subcritical Hopf bifurcation, we can continue the periodic solution with respect to $\tau$ with the following code, which results in figure (12.3):

```
%% Branch off at Hopf bifurcation
disp('Branch off at Hopf bifurcation');
fprintf('Initial correction of periodic orbits at Hopf:\n');
[per1,suc]=SetupPsol(funcs,steadystate_br,ind_hopf,...
'print_residual_info',1,'intervals',20,'degree',4,...
'max_bound',[contpar,8],'max_step',[contpar,0.5],'matrix','full');
figure(3);clf;ax3=gca;
xlabel('$k$')
ylabel('Amplitude')
per1=br_contn(funcs,per1,60,'plotaxis',ax3);
```

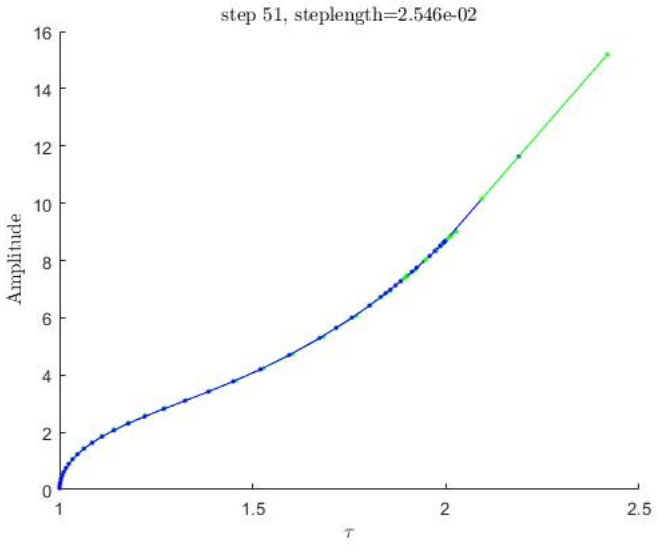



Figure 12.3: Continuation of the periodic solution; green line segments indicate predictions.

We can see that there are only periodic solutions after the Hopf point, which confirms a supercritical Hopf bifurcation. If there were periodic solutions before the found Hopf point we would have a subcritical Hopf bifurcation.

## 12.2 dde23

Now we will look at a tool to find numerically solutions to systems of DDE's, i.e. to simulate them. We will be using dde23, which is a standard function in MATLAB. We will use the same example as with DDE-BIFTOOL. First we need to create a function which defines the system of equations:

```
function dXdt=ddeExde(t,y,Z)
ylag=Z(:,1);              %define the delayed terms
mu=1;                     %set the parameters
dXdt=[-pi/2*ylag*(1+y)];%defining the system
```

Because we are studying a delayed differential equation we also have to define the history:

```
function s=ddeSExhist(t)
s=[0.5];
```

In our case this is a single constant function but for higher dimensional systems we can add more initial values. Lastly we will create a function to solve the system:

```
function ddeEx
sol=dde23(@ddeExde,0.9,@ddeExhist,[0,200]) %solve the equation using dde23
%plot the solution
figure;
plot(sol.x,sol.y);
xlabel('t');
ylabel('x');
```

In the dde23 function we have to enter a system of equations(`@ddeExde`), a delay, the history(`@ddeExhist`) and the interval which we want to simulate the solution in. Instead of a single delay like in this example we can also add multiple delays, we just have to write $[\tau_1, \tau_2, ..., \tau_n]$ instead of our 0.9. Note that dde23 can also be used to study systems of ODE's, in this case instead of a delay we can just write []. This program will create the Figure 12.4

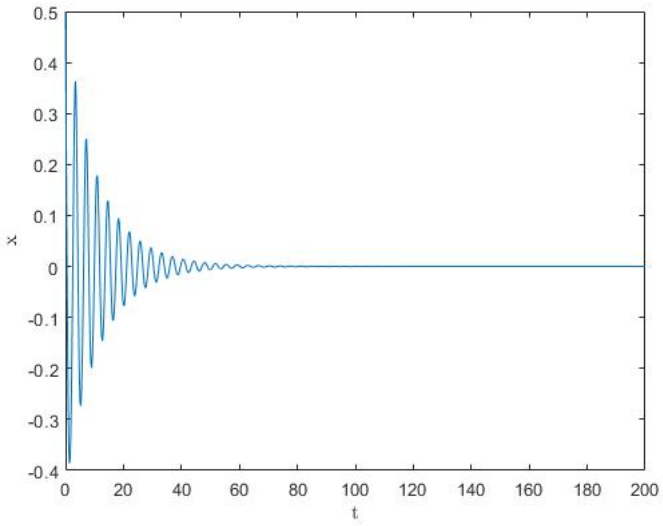



Figure 12.4: Simulation of system (12.1.1) using dde23

# Part IV
# Case Studies

In this Part we will look at 8 different models which are analysed in recent publications. We will use DDE-BIFTOOL to either confirm or find counterexamples to the analysis done in these papers. After that we will simulate these models in order to confirm our numerical analysis. For every model we will first define the model and see what it can be used for, then we will summarize the results found in the papers, and finally check these results with numerical analysis.

## 13 Introduction to the models

### 13.1 Three-dimensional models

First we will look at three models with three state variables, one SIR model found in [9] and two models utilizing separate immature and mature populations found in [10] and [11]. In these models complexity is added trough the nonlinear incidence rate and the first model also has a temporary immunity to make it possible for recovered individuals to return to the susceptible population. In the other two models the time delay is used in the incidence rate in order to delayed transfer to the infected population. The two models using an aged structure can be used for diseases which affect adults in a different way than kids. Examples of this include sexually transmitted diseases or diseases like SARS. The other model can be used for diseases where individuals are still susceptible to even after having contracted them.

### 13.2 SEIR models

Next we will look at three SEIR models found in [12], [13] and [14]. Two of these models have a linear incidence rate while the other one has a nonlinear incidence rate. The first and last model can be used to model diseases and the middle one is used to model computer viruses within a network. The delayed terms in these models are used in three different ways: Two use it to model a temporary immunity rate and one for a delayed immunity.

### 13.3 SIQR models

Lastly we look at two SIQR models described in [15] and [4]. These models have a separate **quarantined population** which contains the individuals who have been infected but are no longer able to infect other individuals. Both of these models are used for computer viruses within networks so the quarantined population contains those computers who are known to be infected and therefore cut off from the network. One of these models uses a nonlinear incidence rate while the other uses a linear incidence rate. In both of these models the delay is used to add a period where the virus is present within the computer but cannot yet infect other computers within the network. They also both assume that recovered nodes cannot return to the susceptible population, so after recovering from the virus the computer becomes immune.

## 14 SIR model with nonlinear incidence rate and temporary immunity

### 14.1 Model

Consider the following model found in [9]:

$$\begin{cases} \dot{S}(t) &= \mu - \mu S(t) - \phi f(I(t))S(t) + \gamma I(t-\tau)e^{-\mu\tau}, \\ \dot{I}(t) &= \phi f(I(t))S(t) - (\mu+\gamma)I(t), \\ \dot{R}(t) &= \gamma I(t) - \gamma I(t-\tau)e^{-\mu\tau} - \mu R(t), \end{cases} \tag{14.1.1}$$

Where $S, I, R$ represent the susceptible, infected and recovered populations respectively. the function $f$ is a nonlinear function satisfying $f(0) = 0, f'(0) > 0, f''(I) < 0$ and $\lim_{t\to\infty} f(I) = c < \infty$. The parameters $\mu, \gamma, \phi$ represent the natural death rate, the recovery rate and the recruitment rate from susceptible to infected respectively. Because this model does not include disease induced death and has a temporary immunity this system could be used to model diseases like the flu. Other diseases with temporary immunity like salmonella and *Chlamydia trachomati* would also be suitable for this model. This system is very similar to the standard model (1.2.1), the differences are that the incidence rate is nonlinear and that there is a term $\gamma I(t-\tau)e^{-\mu\tau}$ which allows this system to model diseases with a temporary immunity.

## 14.2 Results

We first look at the analysis done in [9]. The first two equations of this system do not depend on the third one so they omit this equation without loss of generality. In the paper they assume $f(I) = I/(1+I)$, the model then becomes:

$$\begin{cases} \dot{S}(t) &= \mu - \mu S(t) - \phi \dfrac{I(t)S(t)}{1+I(t)} + \gamma I(t-\tau)e^{-\mu\tau}, \\ \dot{I}(t) &= \phi \dfrac{I(t)S(t)}{1+I(t)} - (\mu+\gamma)I(t), \end{cases} \tag{14.2.1}$$

The basic reproduction ratio is $R_0 = \frac{\phi}{\mu+\gamma}$. This system (14.2.1) has two equilibria, namely the disease free equilibrium $E_0 = (1,0)$ when $R_0 \leq 1$ and an endemic equilibrium $E^* = (S^*, I^*)$ when $R_0 > 1$ where:

$$\begin{cases} S^* &= \dfrac{(\mu+\gamma)(2\mu+\gamma-\gamma)e^{-\mu\tau}}{\mu(\phi+\mu+\gamma) - \gamma\phi(1-e^{-\mu\tau})}, \\ I^* &= \dfrac{\mu(\phi-\mu-\gamma)}{\mu(\phi+\mu+\gamma) - \gamma\phi(1-e^{-\mu\tau})}, \end{cases} \tag{14.2.2}$$

For the stability of the disease-free equilibrium they prove the following theorem:

**Theorem 14.1.** *The disease-free equilibrium, $E_0 = (1,0)$, is stable when $R_0 = 1$.*

The characteristic equation $\Delta(\lambda)$ of the linearization of system (14.2.1) at the equilibrium $E^*$ is given by:

$$\lambda^2 + \left(2\mu + \gamma + \frac{\phi I^*}{1+I^*} - \frac{\phi S^*}{(1+I^*)^2}\right)\lambda - \frac{\mu\phi S^*}{(1+I^*)^2} + \mu(\mu+\gamma) + \frac{\phi(\mu+\gamma)I^*}{1+I^*} - \frac{\gamma\phi I^* e^{-(\mu+\lambda)\tau}}{1+I^*} = 0. \tag{14.2.3}$$

They assume the following hypotheses:
(H1). $R_0 > 1$.
(H2). $\mu(\phi+\mu+\gamma) > \gamma\phi(1-e^{-\mu\tau}$.
(H3). $\dfrac{(2\mu^2+\gamma^2(1-e^{-\mu\tau}))(2\mu^2+\gamma(1-e^{-\mu\tau})(2\mu+\gamma e^{-\mu\tau}))}{\gamma^2(\mu+\gamma)^2(1-e^{-\mu\tau})^2} < 1.$
When these three hypotheses are satisfied then equation (14.2.3) has a pair of purely imaginary roots $\pm i\omega_j$ when $\phi = \phi_j$ where $\phi_j$ is given by:

$$\phi_j = \frac{X(\omega_j)\omega_j + P\omega_j^3 - B^2\gamma e^{-\mu\tau}\sin\omega_j\tau}{Y(\omega_j)\omega_j - B\mu\gamma e^{-\mu\tau}\sin\omega_j\tau}. \tag{14.2.4}$$

Here $X$ and $Y$ are given by:

$$\begin{aligned} X &= B\mu(\mu+A) + B\gamma(\mu+\gamma)(1-e^{-\mu\tau}\cos\omega\tau), \\ Y &= B\gamma(1-e^{-\mu\tau}\cos\omega\tau), \end{aligned} \tag{14.2.5}$$

For $A = \mu - \gamma(1-e^{-\mu\tau})$ and $B = \mu(\mu+\gamma)$. Then they prove the following theorem:

**Theorem 14.2.** *Suppose that (H1),(H2) and (H3) are satisfied.*

1. *If $\phi \in (\mu+\gamma, \phi_0)$, then the endemic equilibrium $E^*$ is asymptotically stable.*
2. *If $\Re(\lambda'(\phi_j)) \neq 0$, then a the system undergoes a Hopf bifurcation at the endemic equilibrium $E^*$ when $\phi = \phi_j$ for $j = 1,2,...,n$.*

After this they proceed to compute the normal form of the Hopf bifurcation.

## 14.3 Numerical analysis

In [9] they fix the following values for $\mu, \gamma$ and $\tau$:

$$\mu = 8, \gamma = 0.7, \tau = 1,$$

Using DDE-BIFTOOL, and setting $\Re(\lambda) \geq -10$ we get the following approximations for the eigenvalues $\lambda$:

$$\lambda = -8.8 \pm 1283.3i, -8.8 \pm 1270.3i, -9,1 \pm 1297.8i, -9.5 \pm 1313.4i, -9.7 \pm 4.4i$$

and we also get an eigenvalue $\lambda = -8$ so without imaginary part. This already points toward a fold bifurcation rather then a Hopf one. In the paper they say that at $\phi = \phi_0 = 79,073$ but I only found a fold bifurcation at the point $\phi = 8.3306$.

In [16] this model is also discussed, they propose the parameter values $\phi = 10, \mu = 0.1$ and $\gamma = 5$ and use $\tau$ as the bifurcation parameter. Plotting the solution for $\tau = 1$ and $\tau = 3$ for initial values $S_0 = I_0 = 0.5$ we get the following figures:

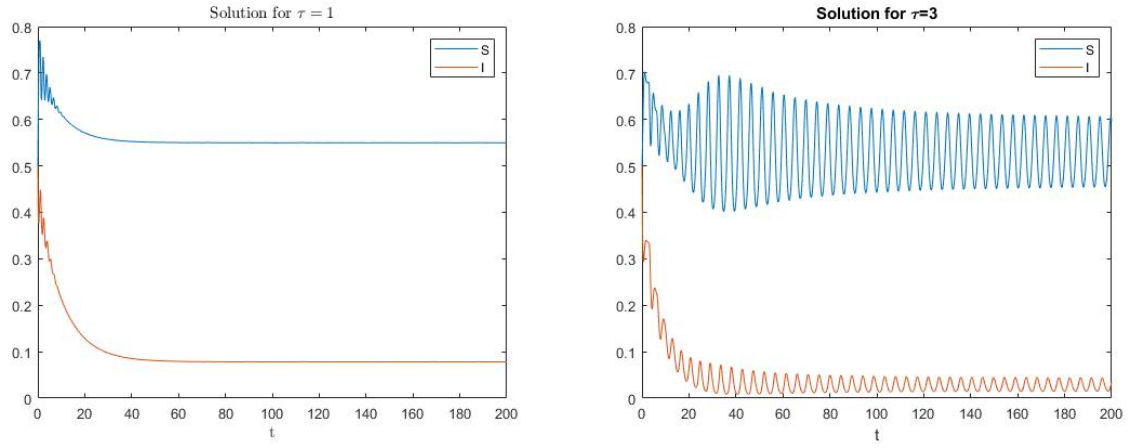



Figure 14.1: Solution for $\tau = 1$ and $\tau = 3$

Here we see the solution for $\tau = 1$ head towards the equilibrium and for $\tau = 3$ heading towards a stable periodic solution which suggests a supercritical hopf bifurcation.

# 15 Epidemic model using immature and mature populations with nonlinear incidence rate.

## 15.1 Model

Consider the following model also found in [10]:

$$\begin{cases} \dot{S}_1(t) &= aS_2(t) - (\mu_1 + b)S_1(t), \\ \dot{S}_2(t) &= S_1(t) - \mu_2 S_2^2(t) - \dfrac{\beta S_2(t) I(t-\tau)}{1+\alpha I(t-\tau)}, \\ \dot{I}(t) &= \dfrac{\beta S_2(t) I(t-\tau)}{1+\alpha I(t-\tau)} - (\mu_3 + \delta) I(t), \end{cases} \tag{15.1.1}$$

Here $a, b, \mu_1, \mu_2, \mu_3, \alpha, \beta, \delta > 0$, $S_1(t)$ represents the immature population and $S_2(t), I(t)$ represent the mature susceptible and infectious populations respectively.

The initial conditions of this system are:

$$\begin{gathered} S_1(\theta) = \phi_1(\theta), S_2(\theta) = \phi_2(\theta), I(\theta) = \phi_3(\theta), \\ \phi_i(\theta) \geq 0, \phi_i(0) > 0 \end{gathered} \tag{15.1.2}$$

For $i \in 1,2,3$, $t \in [-\tau, 0]$ and $\Phi = (\phi_1(\theta), \phi_2(\theta), \phi_3(\theta)) \in C([-\tau,0], \mathbb{R}^3_+ 0)$.

The basic reproduction number is given by:

$$R_0 = \frac{ab\beta}{\mu_2(b+\mu_1)(\mu_3+\delta)}. \tag{15.1.3}$$

Because this model distinguishes between immature and mature populations it can be used for diseases which have more opportunities to spread among adults. Examples are gonorrhea, syphilis, SARS and AIDS. Instead of the standard incidence rate $\beta SI/N$ the nonlinear rate $\frac{\beta IS}{1+\alpha I}$ is used. This is to account for the behavioral change

of susceptible individuals while their number increases or from the crowding effect of infective individuals. This model also differs from the standard model defined in (1.2.1) by not including a recovered population because it is assumed that the disease is fatal. Another difference is that the death rate for the mature population depends on $S_2^2$.

## 15.2 Results

This system is studied in [10]. There are three equilibria for this model, namely:

(i) the trivial equilibrium $E_0(0,0,0)$,

(ii) The disease-free equilibrium $E_1(S_1^0, S_2^0, 0)$ where:

$$S_1^0 = \frac{a^2 b}{\mu_2 (b+\mu_1)^2}, \quad S_2^0 = \frac{ab}{\mu_2(b+\mu_1)}, \tag{15.2.1}$$

(iii) the endemic equilibrium $E^*(S_1^*, S_2^*, I^*)$ where:

$$\begin{aligned} S_1^* &= \frac{aS_2^*}{b+\mu_1}, I^* = \frac{2\mu_2(b+\mu_1)(\mu_3+\delta)}{A}(R_0-1), \\ S_2^* &= \frac{ab\alpha - \beta(b+\mu_1) + \sqrt{(ab\alpha-\beta(b+\mu_1))^2 + 4\alpha\mu_2(b+\mu_1)^2(\mu_3+\delta)}}{2\alpha\mu_2(b+\mu_1)}, \\ A &= \beta\sqrt{(ab\alpha-\beta(b+\mu_1))^2 + 4\alpha\mu_2(b+\mu_1)^2(\mu_3+\delta)} + 2\alpha\mu_2(b+\mu_1)(\mu_3+\delta) - \beta(ab\alpha - \beta(b+\mu_1)), \end{aligned} \tag{15.2.2}$$

The stability of the equilibria is analysed in the following theorems:

**Theorem 15.1.** *$E_0$ is always unstable. If $R_0 < 1$ the disease-free equilibrium $E_1$ is locally asymptotically stable and unstable if $R_0 > 1$.*

**Theorem 15.2.** *let*

$$\begin{aligned} p_0 &= (b+\mu_1)(\mu_3+\delta)\left(2\mu_2 S_2^* + \frac{\beta I^*}{1+\alpha I^*}\right) - ab(\mu_3+\delta), \\ q_0 &= -(\mu_3+\delta)\frac{2\mu_2 S_2^*(b+mu_1) - ab}{1+\alpha I^*}, \end{aligned} \tag{15.2.3}$$

*If $R_0 > 1$ and $p_0 > q_0$, the unique endemic equilibrium $E^*(S_1^*, S_2^*, I^*)$ is locally asymptotically stable for all $\tau > 0$. If $R_0 > 1$ and $p_0 < q_0$, there exists a $\tau_0$ such that if $0 \le \tau < \tau_0$, $E^*$ is locally asymptotically stable and $E^*$ becomes unstable if $\tau > \tau_0$. System (15.1.1) undergoes a Hopf bifurcation at $E^*$ when $\tau = \tau_0$.*

There is no attempt at calculating the normal form of this Hopf bifurcation in the paper.

## 15.3 Numerical analysis

In the numerical section of [10] they propose the following parameter values:

$$a = 5,\ b = 2,\ \mu_1 = 0.1,\ \mu_2 = 0.01,\ \mu_3 = 0.1,\ \alpha = 0.0001,\ \beta = 0.01,\ \delta = 0.8. \tag{15.3.1}$$

For $\tau = 1$ the eigenvalues of this system are (with minimal real part -4):

$$\begin{aligned} \lambda = &-0.3458 \pm 0.8028i,\ -2.2453 \pm 5.2794i,\ -2.7832 \pm 11.3853i,\ -3.1415 \pm 17.5429i, \\ &-3.4171 \pm 23.7539i,\ -3.6379 \pm 29.9928i,\ -3.8208 \pm 36.2467i,\ -3.9765 \pm 42.5094i, \end{aligned} \tag{15.3.2}$$

Because they all have negative real parts this equilibrium is stable for $\tau = 1$. Continuing the equilibria for higher values of $\tau$ and checking for Hopf bifurcation we get an instance of Hopf bifurcation at the critical value $\tau_0 = 1.2374$ with the first Lyapunov coefficient being $L_1 = -1.2789 \cdot 10^{-6}$. So we have a supercritical Hopf bifurcation, now plotting the solution we get:

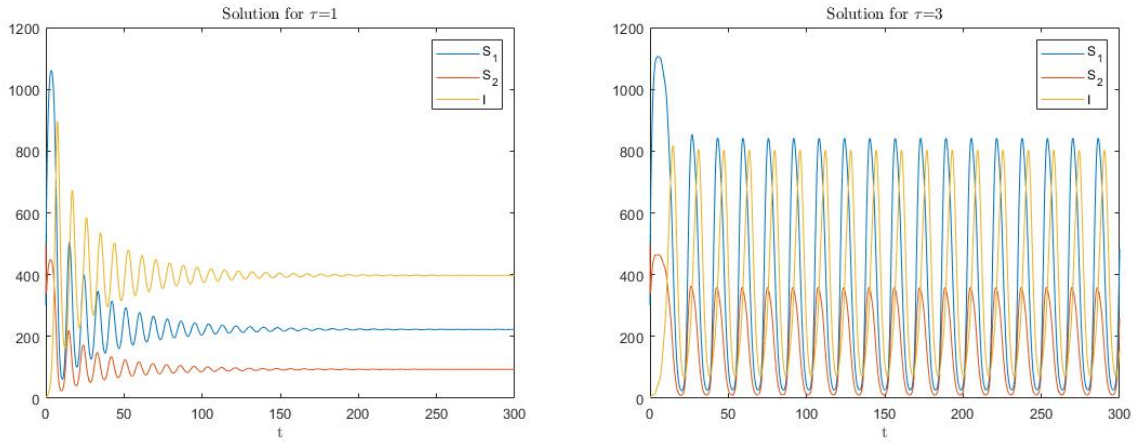



Figure 15.1: Solution for $\tau = 1$ and $\tau = 3$

In these figures we again see that there is a supercritical Hopf bifurcation, for $\tau = 1$ the solution tends towards the equilibrium and for $\tau = 3$ it tends towards the stable periodic solution. Continuing the periodic solution with respect to the amplitude gives the following figure:

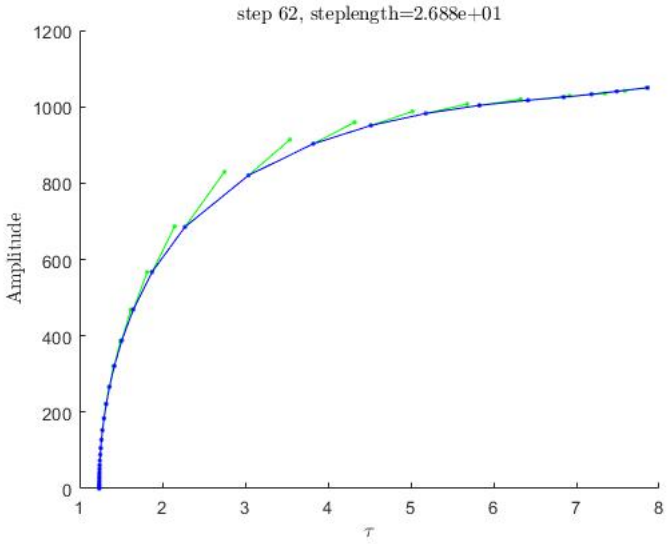



Figure 15.2: Continuation of periodic solution starting at critical value $\tau_0 = 1.2374$

We only see periodic solutions after the determined Hopf point, so this again confirms a supercritical Hopf bifurcation.

# 16 XSI model with nonlinear incidence rate.

## 16.1 Model

The following model can be found in [11]:

$$\begin{cases} \dot{X}(t) &= A + BS(t) - (\omega + r)X(t), \\ \dot{S}(t) &= \omega X(t) - \mu S(t) - \dfrac{\sigma S(t) I(t-\overline{\tau})}{1+\zeta I(t-\overline{\tau})} + \eta I(t), \\ \dot{I}(t) &= \dfrac{\sigma S(t) I(t-\overline{\tau})}{1+\zeta I(t-\overline{\tau})} - (\mu + \varepsilon + \eta) I(t), \end{cases} \tag{16.1.1}$$

With $A, B, \omega, r, \mu, \sigma, \varepsilon$ and $\eta$ positive constants and $X$, $S$ and $I$ represent the immature, mature and infected populations respectively. The conversion rate from immature to mature individuals is $\omega$, $r$ and $\mu$ are the natural death

rates of the immature and mature populations respectively and $\sigma$ represents the probability per unit time that the infection is passed between two individuals taking part in contact.
Setting $x = \frac{\omega+r}{A}X$, $s = \frac{B}{A}S$, $i = \frac{\eta B}{A(\omega+r)}$ and using the dimensionless time scale $\bar{t} = (\omega+r)t$ (for simplicity $\bar{t}$ will be substituted by $t$) the system can be rewritten as follows:

$$\begin{cases} \dot{x}(t) &= 1 - x(t) + s(t), \\ \dot{s}(t) &= bx(t) - ds(t) - \dfrac{\alpha s(t) i(t-\tau)}{1+mi(t-\tau)} + i(t), \\ \dot{i}(t) &= \dfrac{\beta s(t) i(t-\tau)}{1+mi(t-\tau)} - ci(t), \end{cases} \tag{16.1.2}$$

Where

$$b = \frac{\omega B}{(\omega+r)^2},\ d = \frac{\mu}{\omega+r},\ \alpha = \frac{\sigma A}{\eta B},\ m = \frac{\zeta A(\omega+r)}{\eta B},\ \beta = \frac{\sigma A}{(\omega+r)B},\ c = \frac{\mu+\varepsilon+\eta}{\omega+r},\ \tau = (\omega+r)\bar{\tau}. \tag{16.1.3}$$

The initial conditions for this system are given by:

$$(\phi, \psi_1, \psi_2) \in C([-\tau, 0], \mathbb{R}^3_{\geq 0}),\ \ \phi(0) > 0,\ \psi_i(0) > 0,\ i = 1, 2. \tag{16.1.4}$$

This model is very similar to the one defined in (15.1.1) and can therefore also be used for diseases which spread differently among immature populations like SARS and AIDS. Differences are that the disease is not assumed to be fatal and the death rate of the mature population depends only on $S$ which is more standard.

## 16.2 Results

System (16.1.2) is analysed in [11]. It has two equilibria:

(i) The disease free equilibrium $E_0 = (x^0, s^0, i^0) = (\frac{d}{d-b}, \frac{b}{d-b}, 0)$.

(ii) The positive equilibrium $E_+(x^*, x^*, i^*)$ which exists when $R_0 = \frac{\beta b + bc}{dc} > 1$ (H1) and $dcm - bcm + \alpha c - \beta > 0$ (H2), where:

$$x^* = 1 + \frac{c(1+mi^*)}{\beta},\ s^* = x^* - 1,\ i^* = \frac{\beta b + bc - dc}{dcm - bcm + \alpha c - \beta}. \tag{16.2.1}$$

The stability of these equilibria is analysed in the following theorems:

**Theorem 16.1.** *For system (16.1.2), the disease-free equilibrium $E_0 = (\frac{d}{d-b}, \frac{b}{d-b}, 0)$ is*

*(i) asymptotically stable if $R_0 < 1$;*

*(ii) linear neutrally stable if $R_0 = 1$;*

*(iii) unstable if $R_0 > 1$.*

Denote

$$f = \frac{i^*}{1+mi^*},\ j = \frac{c}{\beta(1+mi^*)}. \tag{16.2.2}$$

The characteristic equation at the positive equilibrium $E_+$ is:

$$\lambda^3 + (1+d+\alpha f+c)\lambda^2 + ((1+c)(d+\alpha f) + c - \beta f - b)\lambda + c(d+\alpha f) - (\beta j\lambda^2 + (1+d)\beta\lambda + (d-b)\beta j)e^{-\lambda\tau} = 0 \tag{16.2.3}$$

Let $\lambda = \pm i\omega_0$ be roots of this equation. Define

$$\begin{aligned} D &= 1 + d + c + \alpha f, \\ E &= d + \alpha f - b + c(d+1) + f(c\alpha - \beta), \\ F &= cd - bc + f(c\alpha - \beta), \\ G &= (d-b)\beta j. \end{aligned} \tag{16.2.4}$$

and

$$\begin{aligned} p &= D^2 - 2E - \beta^2 j^2, \\ q &= E^2 - 2FD - \beta^2 j^2 (1+d)^2 + 2\beta j G, \\ r &= F^2 - G^2. \end{aligned} \tag{16.2.5}$$

**Theorem 16.2.** *For system (16.1.2), the following statements hold:*

*(i) if $r \geq 0$ and $p^2 - 3q \leq 0$ then the positive equilibrium $E_+$ is absolutely stable.*

*(ii) if (H1), (H2) hold and $\beta d + dc - bc < 0$, $d\alpha - b\alpha + 2b\beta + beta > 0$. Then $E^*$ is stable for all $\tau \in [0, \tau_0)$ and unstable for $\tau > \tau_0$. The system undergoes a Hopf bifurcation at $\tau = \tau_0$. Where*

$$\tau_0 = \frac{1}{\omega_0} \arccos\left( \frac{(-\omega_0^3 + E\omega_0)\beta j(1+d)\omega_0 + (-D\omega_0^2 + F)(-\beta j\omega_0^2 + G)}{\beta^2 j^2 \omega_0^2 (1+d)^2 + (-\beta j\omega_0^2 + G)^2} \right). \tag{16.2.6}$$

They then proceed to calculate the normal form of the Hopf bifurcation.

## 16.3 Numerical analysis

In the paper they suggest the the following values for these parameters:

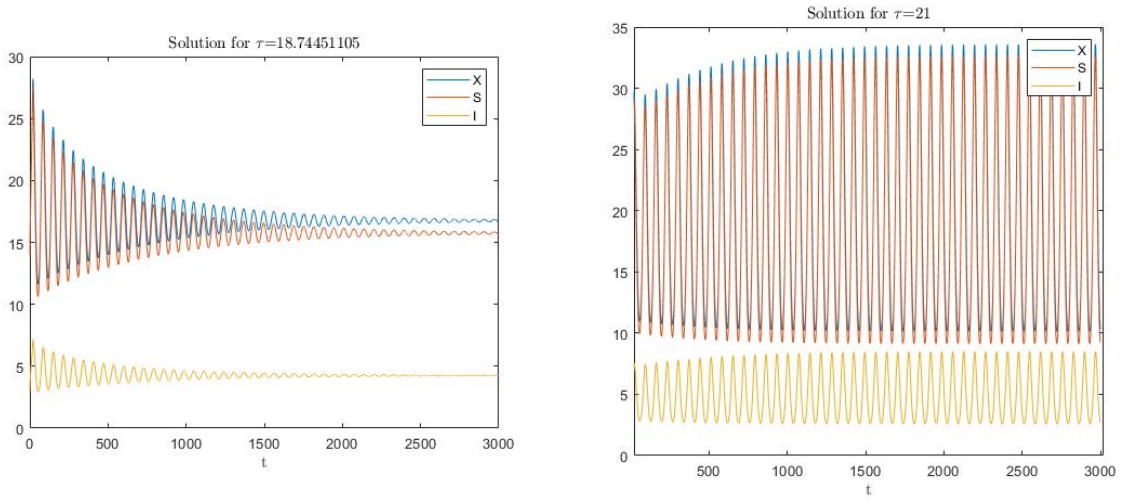
$$b = 2.5,\ d = 0.5,\ \alpha = c = 3,\ m = \beta = 1. \tag{16.3.1}$$

At the positive equilibrium $E_+ = (16.75, 15.75, 4.25)$ the eigenvalues for $\tau = 10$ are :

$$\begin{aligned} \lambda = &-0.0302 \pm 0.1464i,\ -0.1537 \pm 0.6952i,\ -0.1855 \pm 1.2874i,\ -0.1997 \pm 1.8893i,\ -0.2093 \pm 2.4971i, \\ &-0.2177 \pm 3.1089i,\ -0.2257 \pm 3.7237i,\ -0.2335 \pm 4.3408i,\ -0.2412 \pm 4.9596i,\ -0.2487 \pm 5.5800i. \end{aligned} \tag{16.3.2}$$

So the positive equilibrium is stable like in the paper. If we continue the equilibria for higher values of $\tau$ we get a Hopf bifurcation critical value at $\tau_0 = 19.8445$ just like they got in the paper. The first Lyapunov coefficient is equal to $L_1 = -2.7387 \cdot 10^{-4}$ so we have a supercritical hopf bifurcation. We will now plot the solution once for a value $\tau < \tau_0$ and for $\tau > \tau_0$ with initial data $x = 15, s = 14, i = 3$:

Figure 16.1: Solution for $\tau = 18.74451105$ and $\tau = 21$




These figures again confirm the existence of Hopf bifurcation, in the picture to the right we see the solution tending towards the equilibrium $E_+$ and in the picture to the right it tends towards the stable periodic solution.

# 17 SEIR model with bi-linear incidence rate and delayed immunity.

## 17.1 Model

Consider the following model from [13]:

$$\begin{cases} \dot{S}(t) &= b + \gamma I(t) + \varepsilon R(t) - (\mu + p)S(t) - \beta S(t)I(t), \\ \dot{E}(t) &= \beta S(t)I(t) - \mu E(t) - \eta E(t)R(t), \\ \dot{I}(t) &= (\eta + \sigma\beta)E(t) - (\alpha + \mu + \gamma)I(t), \\ \dot{R}(t) &= pS(t-\tau)E(t-\tau)R(t-\tau) - (\varepsilon + \mu + \sigma\beta)R(t) \end{cases} \tag{17.1.1}$$

Where p is the proportion of asymptomatic infection, b is the birth rate, $\gamma$ is the incubation period, $\beta$ is the transmission rate, $\mu$ is the death rate, $\eta$ is the infectious period of symptomatic infection of people, $\sigma$ is the infectious period of asymptomatic infection of people, $\alpha$ is the multiple of the transmissibility while infected cells are created from the viruses and $\varepsilon$ is the latent period of human infection in population no longer infectious due to being fully recovered. The basic reproduction number for the system is defined by:

$$R_0 = \frac{b\beta(\eta+\sigma\beta)}{\mu(\mu+p)(\alpha+\mu+\gamma)}$$

The initial conditions for this model are $S(t)\geq 0, S(0)>0, E(t)\geq 0, E(0)>0, I(t)\geq 0, I(0)>0, R(t)\geq 0, R(0)>0$.

This system is used to model the current COVID-19 pandemic. Looking at the standard model described in (1.2.1) this model uses an extra population for exposed individuals and has the term $pS(t-\tau)E(t-\tau)R(t-\tau)$ such that a delayed immune response and duration of an individual being infectious can be modelled.

## 17.2 Results

We now look at the claims about this model made in [13]. It has three equilibria:

(i) COVID-19 infection free equilibrium:

$$E_0 = \left(\frac{b}{\mu+p}, 0, 0, 0\right) \tag{17.2.1}$$

(ii) COVID-19 infection absent equilibrium:

$$E_1 = \left(\frac{\mu(\alpha+\mu+\gamma)}{\beta}(\eta+\sigma\beta), \frac{b\beta(\eta+\sigma\beta)-\mu(\mu+p)(\alpha+\mu+\gamma)}{\mu\beta(\eta+\sigma\beta)}, \frac{b\beta(\eta+\sigma\beta)-\mu(\mu+p)(\alpha+\mu+\gamma)}{\mu\beta(\alpha+\mu+\gamma)}, 0\right) \tag{17.2.2}$$

(iii) COVID-19 infection present equilibrium: $\overline{E} = (\overline{E}_1, \overline{E}_2, \overline{E}_3, \overline{E}_4)$ where:

$$\begin{cases} \overline{E}_1 &= \dfrac{p(\alpha+\mu+\gamma)b-\beta(\eta+\sigma\beta)(\varepsilon+\mu+\sigma\beta)}{p(\mu+p(\alpha+\mu+\gamma)}, \\ \overline{E}_2 &= \dfrac{(\varepsilon+\mu+\sigma\beta)(\mu+p)(\alpha+\mu+\gamma)}{p(\alpha+\mu+\gamma)b-\beta(\eta+\sigma\beta)(\varepsilon+\mu+\sigma\beta)}, \\ \overline{E}_3 &= \dfrac{(\eta+\sigma\beta)(\varepsilon+\mu+\sigma\beta)(\mu+p)}{p(\alpha+\mu+\gamma)b-\beta(\eta+\sigma\beta)(\varepsilon+\mu+\sigma\beta)}, \\ \overline{E}_4 &= \dfrac{1}{\eta}\left(\dfrac{\beta(\eta+\sigma\beta)(p(\alpha+\mu+\gamma)b-\beta(\eta+\sigma\beta)(\varepsilon+\mu+\sigma\beta))}{p(\mu+p)(\alpha+\mu+\gamma)^2}-\mu\right) \end{cases} \tag{17.2.3}$$

**Theorem 17.1.** *If $R_0<1$, then $E_0$ is globally asymptotically stable.*

**Theorem 17.2.** *If $1<R_0<1+\frac{\beta^2(\eta+\sigma\beta)^2(\varepsilon+\mu+\sigma\beta)}{\mu p(\mu+p)(\alpha+\mu+\gamma)^2}$ then $E_1$ is locally asymptotic stable. If $R_0>1+\frac{\beta^2(\eta+\sigma\beta)^2(\varepsilon+\mu+\sigma\beta)}{\mu p(\mu+p)(\alpha+\mu+\gamma)^2}$ then $E_1$ is unstable.*

The characteristic equation of equilibrium $\overline{E}$ is given by:

$$\Omega(\lambda) = \lambda^4 + x_1\lambda^3 + x_2\lambda^2 + x_3\lambda + x_4 - (x_5\lambda^3 + x_6\lambda^2 + x_7\lambda + x_8)e^{-\lambda\tau}. \tag{17.2.4}$$

Where

$$\begin{aligned}
x_1 &= (\varepsilon+\mu+\sigma\beta)+(\mu+p)+\beta\overline{I}+(\alpha+\mu+\gamma)+\mu+\eta\overline{R}, \\
x_2 &= (\varepsilon+\mu+\sigma\beta)(\mu+p)+(\varepsilon+\mu+\sigma\beta)\beta\overline{I}+(\varepsilon+\mu+\sigma\beta)(\alpha+\mu+\gamma) \\
&+ (\mu+p)(\alpha+\mu+\gamma)+\beta\overline{I}(\alpha+\mu+\gamma)+(\mu+\eta\overline{R})((\varepsilon+\mu+\sigma\beta)+(\mu+p)+\beta\overline{I}), \\
x_3 &= (\varepsilon+\mu+\sigma\beta)(\mu+p)(\alpha+\mu+\gamma)+(\varepsilon+\mu+\sigma\beta)\beta\overline{I}(\alpha+\mu+\gamma) \\
&+ (\mu+\eta\overline{R})((\varepsilon+\mu+\sigma\beta)(\mu+p)+(\varepsilon+\mu+\sigma\beta)\beta\overline{I})+(\eta+\sigma\beta)\beta^2\overline{S}\overline{I}, \\
x_4 &= (\varepsilon+\mu+\sigma\beta)(\eta+\sigma\beta)\beta^2\overline{S}\overline{I}, \\
x_5 &= (\varepsilon+\mu+\sigma\beta), \\
x_6 &= (\varepsilon+\mu+\sigma\beta)(\mu+p)+(\varepsilon+\mu+\sigma\beta)\beta\overline{I}+(\varepsilon+\mu+\sigma\beta)(\alpha+\mu+\gamma)+\mu(\varepsilon+\mu+\sigma\beta), \\
x_7 &= (\varepsilon+\mu+\sigma\beta)(\mu+p)(\alpha+\mu+\gamma)+(\varepsilon+\mu+\sigma\beta)\beta\overline{I}(\alpha+\mu+\gamma) \\
&+ (\mu+\eta\overline{R})((\varepsilon+\mu+\sigma\beta)(\mu+p)+(\varepsilon+\mu+\sigma\beta)\beta\overline{I}) \\
&- (\varepsilon+\mu+\sigma\beta)\eta\overline{R}((\mu+p)+\beta\overline{I}+(\alpha+\mu+\gamma)), \\
x_8 &= (\varepsilon+\mu+\sigma\beta)(\eta+\sigma\beta)\beta^2\overline{S}\overline{I}-(\varepsilon+\mu+\sigma\beta)(\mu+p)(\alpha+\mu+\gamma)\eta\overline{R}.
\end{aligned} \tag{17.2.5}$$

Let $\lambda = \pm\phi_0 i$ be roots to this equation.

**Theorem 17.3.** *$\overline{E}$ is locally asymptotically stable if $\tau = 0$ and $R_0 > 1 + \frac{\beta^2(\eta+\sigma\beta)^2(\varepsilon+\mu+\sigma\beta)}{\mu p(\mu+p)(\alpha+\mu+\gamma)^2}$*

System (17.1.1) is stable when $\tau \in [0, \tau_0)$ and unstable when $\tau > \tau_0$ and there is bifurcation at $\tau = \tau_0$ where:

$$\tau_0 = \frac{1}{\phi_0} \arccos\left(\frac{c_1\phi_0^6 + c_2\phi_0^4 + c_3\phi_0^2 + c_4}{e_1\phi_0^6 + e_2\phi_0^4 + e_3\phi_0^2 + e_4}\right). \tag{17.2.6}$$

for

$$\begin{array}{rcl} c_1 & = & x_1x_5 - x_6,\ c_2 = x_8 + x_2x_6 - x_1x_7 - x_3x_5,\ c_3 = x_3x_7 - x_2x_8 - x_4x_6,\ c_4 = x_4x_8, \\ e_1 & = & x_5,\ e_2 = x_6 - 2x_5x_7,\ e_3 = x_7^2 - 2x_6x_8,\ e_4 = x_8^2. \end{array} \tag{17.2.7}$$

There is no attempt at calculating the normal form for this bifurcation.

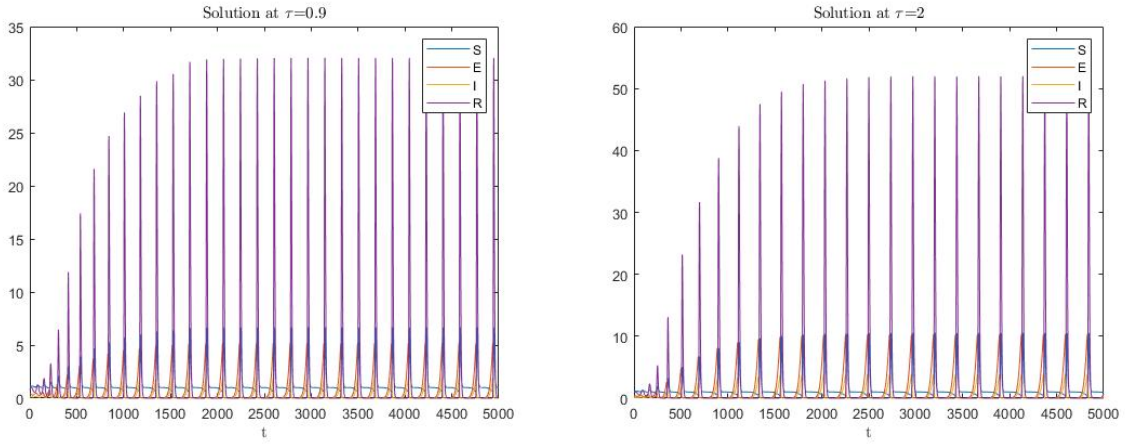
## 17.3 Numerical analysis

The values they propose in the paper for $b, \gamma, \varepsilon, \mu, p, \beta, \eta, \sigma$ and $\alpha$ are $0.5, 0.008, 0.1, 0.0018, 0.5, 0.1923, 0.1, 0.5$ and $0.5$ respectively. Calculating the eigenvalues at the infection present equilibrium $\overline{E} = (\overline{E}_1, \overline{E}_1, \overline{E}_3, \overline{E}_4)$ defined in (17.2.3), taking $\tau = 0.5$ we get:

$$\begin{array}{l} \lambda = 0.0040 \pm 0.0468i, -0.2585, -0.3058, -9.6309 \pm 7.6333i, -10.9546 \pm 21.0382i, \\ -11.7846 \pm 33.8934i, -12.3765 \pm 46.6087i, -12.8350 \pm 59.2668i, -13.2089 \pm 71.8958i, \\ -13.5244 \pm 84.5079i, -13.7972 \pm 97.1091i, \end{array} \tag{17.3.1}$$

So according to the software this equilibrium is unstable, which is in line with the paper because they only got a stable equilibrium at $\tau = 0$. Because there are already eigenvalues with positive real part there is already a periodic solution at $\tau = 0.5$, for lower values of $\tau$ this doesn't change. The paper claims that there occurs Hopf bifurcation at $\tau = 2$. Using DDE23 we can solve the differential equations for $\tau = 0.9$ and $\tau = 2$ like they did in the paper, as initial values we will take $S_0 = 1.1, E_0 = 0.4, I_0 = 0.2$ and $R_0 = 0.8$ as this is close to the equilibrium. Looking at the figure (17.1) we see there is a periodic solution in both cases, so there is no Hopf bifurcation at $\tau = 2$.

Figure 17.1: Solution for $\tau = 0.9$ and $\tau = 2.0$




# 18 SEIR model with nonlinear incidence rate and temporary immunity

## 18.1 Model

Next we look at the following SEIR model found in [14]:

$$\begin{cases} \dot{S}(t) &= \Pi - \dfrac{\beta\beta_E E(r-\tau) + \beta\beta_I I(t-\tau)}{N(t-\tau)} S(t-\tau) - \mu S(t) \\ \dot{E}(t) &= \dfrac{\beta\beta_E E(t-\tau) + \beta\beta_I I(t-\tau)}{N(t-\tau)} S(t-\tau) - (\mu+\sigma+\kappa)E(t) \\ \dot{I}(t) &= \sigma E(t) - (\mu+\alpha+\gamma)I(t) \\ \dot{R}(t) &= \kappa E(t) + \gamma I(t) - \mu R(t) \end{cases} \tag{18.1.1}$$

Here $\Pi$ is the recruitment rate into the population, $\mu$ is the natural death rate, $\sigma$ is the rate at which exposed individuals become infectious, $\kappa$ is the recovery rate of exposed individuals, $\gamma$ is the recovery rate of infected individuals, and $\alpha$ is the rate of disease-induced death.
The initial conditions for this model are given by:

$$S(\theta) = \phi_1(\theta), \quad E(\theta) = \sigma_2(\theta), \quad I(\theta) = \phi_3(\theta), \quad R(\theta) = \phi_4(\theta), \quad \theta \in [-\tau, 0], \tag{18.1.2}$$

With $\phi = [\phi_1, \phi_2, \phi_3, \phi_4] \in C[[-\tau, 0], \mathbb{R}^4_+]$, where $\mathbb{R}^4_+ = \{(S, E, I, R) | S \geq 0, E \geq 0, I \geq 0, R \geq 0\}$.

Apart from the extra population for susceptible individuals this system differs from the standard system (1.2.1) in that it utilizes a nonlinear incidence rate $\frac{\beta\beta_E E(t-\tau)+\beta\beta_I I(t-\tau)}{N(t-\tau)}$ instead of the standard $IS/N$. This incidence rate depends on both the infected and exposed population, so it can be used to model diseases for which individuals can spread the disease even if they aren't sick yet. Examples include influenza and SARS, the current COVID-19 pandemic can also be modelled using this system because exposed individuals can infect others.

## 18.2 Results

We now look at the analysis in [14], the system has two positive equilibria:

(i) Disease-free equilibrium $\varepsilon_0 = (\frac{\Pi}{\mu}, 0, 0, 0)$

(ii) Endemic equilibrium $\varepsilon^* = (S^*, E^*, I^*, R^*)$, with:

$$S^* = \frac{\Pi(k_1 k_2 - \sigma\alpha)}{\mu(k_1 k_2 R_0 - \sigma\alpha)}, \quad E^* = \frac{k_2 \Pi(R_0 - 1)}{k_1 k_2 R_0 - \sigma\alpha}, \quad I^* = \frac{\sigma\Pi(R_0 - 1)}{k_1 k_2 R_0 - \sigma\alpha}, \quad R^* = \frac{\Pi(\kappa k_2 + \sigma\gamma)(R_0 - 1)}{\mu(k_2 k_2 R_0 - \sigma\alpha)} \tag{18.2.1}$$

Where $k_1 = \mu + \kappa + \sigma, k_2 = \mu + \gamma + \alpha$ and $R_0 = \frac{\beta\beta_E k_2 + \beta\beta_I \sigma}{k_1 k_2}$. Because they require the population sizes to be positive $R_0 > 1$ needs to be assumed for the endemic equilibrium.

First they give the following theorem for the stability of the disease-free equilibrium:

**Theorem 18.1.** *The disease-free equilibrium $\varepsilon_0$ is:*

*(i) absolutely stable if $R_0 < 1$,*

*(ii) linearly neutrally stable if $R_0 = 1$,*

*(iii) unstable if $R_0 > 0$.*

They prove this by looking at the eigenvalues of the linearization of the model around $\varepsilon_0$. They then also proceed to prove that for the region $\Omega = \{(S, E, I, R) \in \mathbb{R}^4_+ | S + E + I + R \leq \Pi/\mu\}$ the equilibrium of $\varepsilon_0$ is globally asymptotically stable if $R_0 \leq 1$.

The characteristic equation of the endemic equilibrium is given by:

$$(\lambda + \mu)(\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 + (b_2\lambda^2 + b_1\lambda + b_0)e^{-\lambda\tau}) = 0. \tag{18.2.2}$$

Where

$$\begin{aligned} a_0 &= \mu k_1 k_2,\ a_1 = k_1 k_2 + \mu(k_1 + k_2),\ a_2 = \mu + k_1 + k_2, Q^* = \frac{\beta_1 E^* + \beta_2 I^*}{N^*}, \\ b_0 &= -\mu k_1 k_2 + k_1 k_2 Q^* - \frac{\sigma\alpha Q^*}{R_0}, b_1 = -k_1 k_2 - \frac{\beta_1 \mu}{R_0} + (k_1 + k_2)Q^*, \\ b_2 &= Q^* - \frac{\beta_1}{R_0}. \end{aligned} \tag{18.2.3}$$

This equation has a pair of purely imaginary roots of the form $\lambda = \pm i\omega_0$. The bifurcation critical parameter $\tau_0$ is equal to:

$$\tau_0 = \frac{1}{\omega_0} \arccos\left(\frac{(\omega_0^3 - a_1\omega_0)b_1\omega_0 - (a_2\omega_0^2 - a_0)(b_2\omega_0^2 - b_0)}{(b_0 - b_2\omega_0^2)^2 + (b_1\omega_0)^2}\right). \tag{18.2.4}$$

The stability of the endemic equilibrium is then analysed in the following theorem:

**Theorem 18.2.** *Suppose $R_0 > 1$. Then the endemic equilibrium $\varepsilon^*$ is:*

*(i) absolutely stable for* $\tau \geq 0$ *whenever*

$$\frac{k_1 k_2}{\beta_E k_2 + \beta_I \sigma} < \beta < \frac{2k_1 k_2 - \sigma\alpha}{\beta_E k_2 + \beta_I \sigma}. \tag{18.2.5}$$

*(ii) conditionally stable for* $\tau \in [0, \tau_0)$ *whenever* $\beta > \frac{3k_1 k_2 - \sigma\alpha}{\beta_E k_2 + \beta_I \sigma}$. *System (18.1.1) undergoes a Hopf bifurcation at* $\tau = \tau_0$.

Lastly they compute the normal form of the Hopf bifurcation.

## 18.3 Numerical analysis

The following parameter values are proposed in the paper:

$$\Pi = 160,\ \beta_E = 0.21,\ \beta_I = 0.84,\ \mu = 0.000263,\ \sigma = 2.32,\ \gamma = 1.4,\ \kappa = 1.3,\ \alpha = 0.065. \tag{18.3.1}$$

And $\beta$ is kept as a variable. When $\beta = 2$ we have $R_0 < 1$ so the solution should converge to the disease-free equilibrium $\varepsilon_0$ for all values of $\tau$, plotting the solution for $\tau = 5, 15, 25$ we get the following figures for initial conditions $S(0) = 1.92465 \cdot 10^5$, $E(0) = 30$, $I(0) = 47$ and $R(0) = 4.03998 \cdot 10^5$:

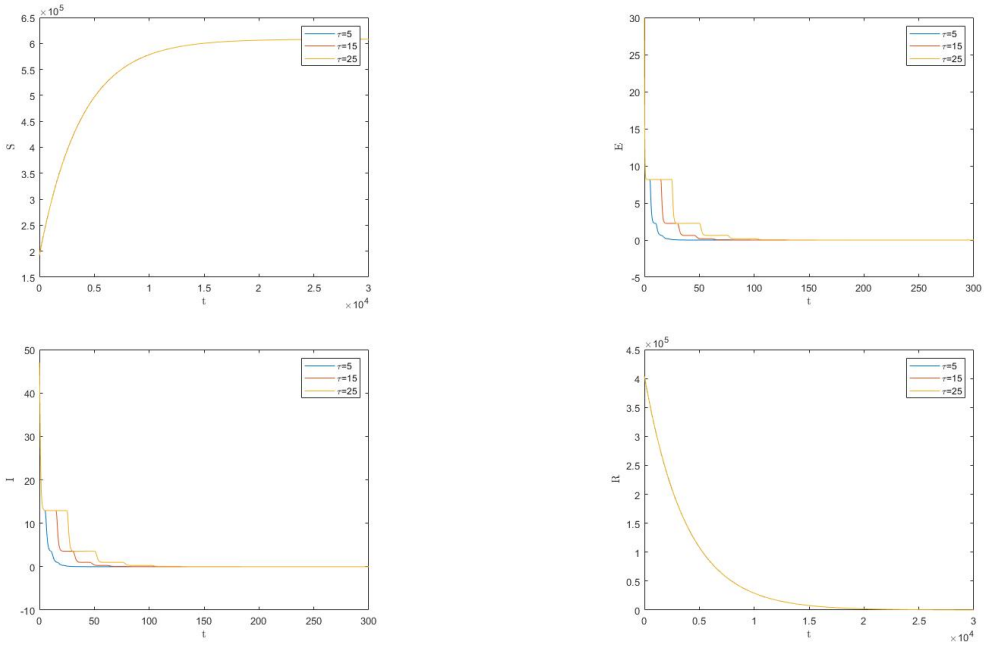



Figure 18.1: Solutions of model (18.1.1) for $\tau = 5, 15, 25$ when $\beta = 2$.

We see that for all three values of $\tau$ the solution tends towards the equilibrium and is therefore stable.

Next for $\beta = 7.2875$ we have $R_0 >$ and the condition for Hopf bifurcation is satisfied. Continuing the equilibrium in DDE-BIFTOOL we get a Hopf bifurcation at $\tau_0 = 25.8558$ just as they got in the paper. The first Lyapunov coefficient of this bifurcation is $L_1 = 2.0951 \cdot 10^{-13}$, this indicates a subcritical Hopf bifurcation where as in the paper they thought it was supercritical. Next we will plot the solutions for $\tau = 15$ and $\tau = 30$:

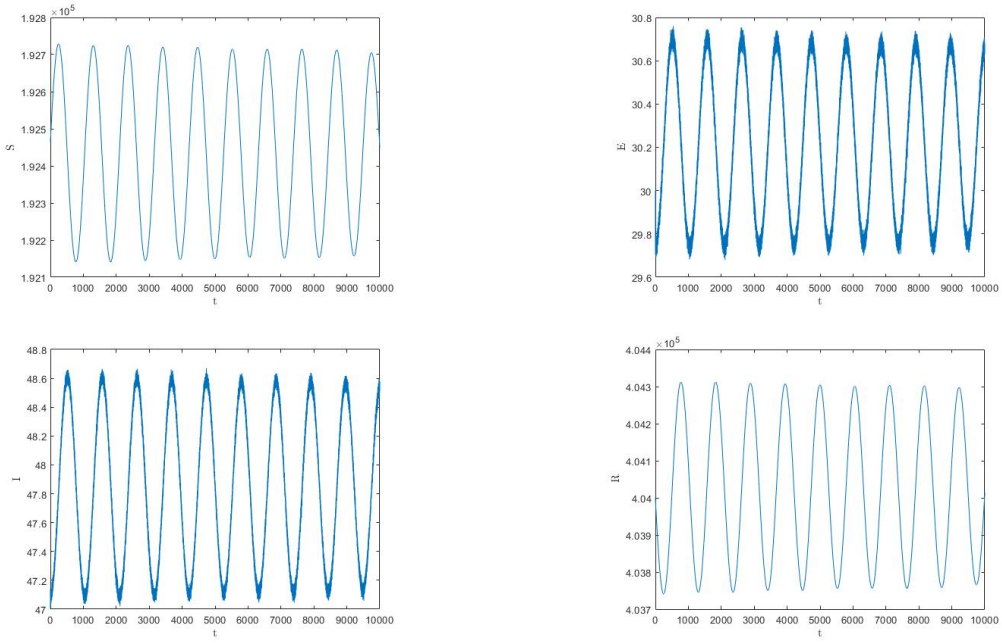



Figure 18.2: Solutions of model (18.1.1) for $\tau = 15$

Here we see the solution getting smaller, so there is no periodic solution yet and it will tend towards the equilibrium.

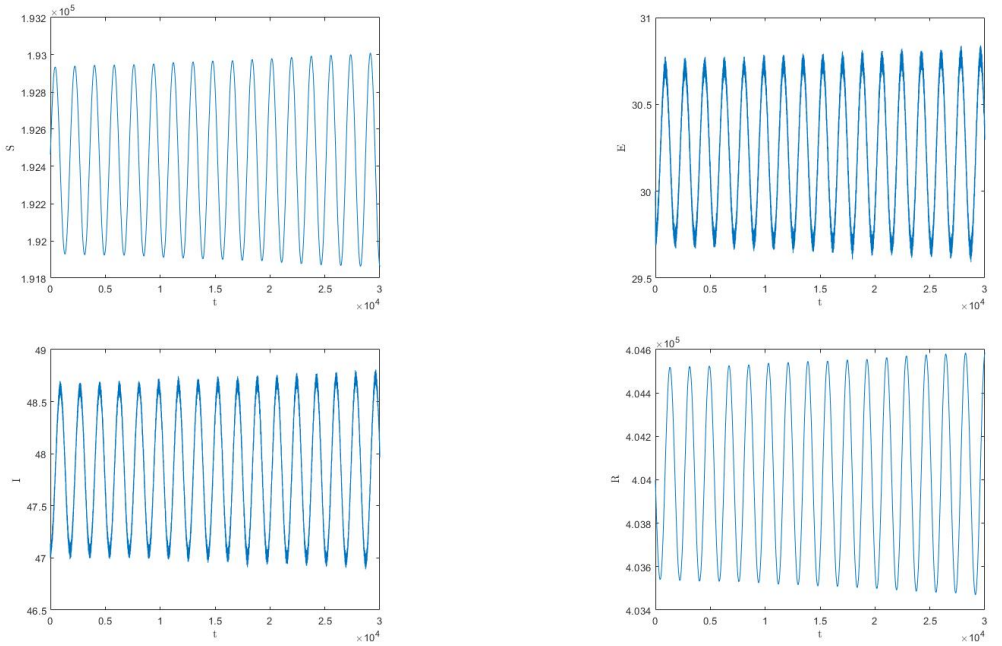



Figure 18.3: Solutions of model (18.1.1) for $\tau = 30$

Here we see the oscillations getting bigger, which again indicates a subcritical Hopf bifurcation.

# 19 SLIR model with bi-linear incidence rate

## 19.1 Model

We consider the following system found in [12]:

$$\begin{cases} \dot{S}(t) &= A-\beta S(t)L(t)+\omega I(t-\tau)-\varepsilon R(t)-\mu S(t), \\ \dot{L}(t) &= \beta S(t)L(t)-(\delta+\gamma+\mu)L(t), \\ \dot{I}(t) &= \gamma L(t)-(\alpha+\mu)I(t)-\omega I(t-\tau), \\ \dot{R}(t) &= \delta L(t)+\alpha I(t)-(\mu-\varepsilon)R(t), \end{cases} \tag{19.1.1}$$

Here $S(t)$, $L(t)$, $I(t)$ and $R(t)$ are the sizes of susceptible, exposed, infectious and recovered populations at time $t$. $\alpha,\beta,\gamma,\mu,\varepsilon,A\in\mathbb{R}$, $t>0$ and $\tau$ indicates the time delay. This model can be used to model computer viruses and normal diseases because it is pretty standard. Differences from the standard epidemic model defined in (1.2.1) are the separate population for susceptible nodes, a term $\varepsilon R$ which allow the transfer from the susceptible population directly into the recovered population and a delayed transfer from the infected population to the susceptible population. This model can be used to model computer viruses in a network, the ability to transfer from susceptible directly into recovered might be because when a computers start to recover updates can be developed to prevent other computer from getting infected.

## 19.2 Results

System (19.1.1) is analysed in [12], it has an unique endemic equilibrium $P_*(S_*,L_*,I_*,R_*)$, where:

$$\begin{aligned} S_* &= \frac{\delta+\gamma+\mu}{\beta},\ L_*=\frac{(\mu-\varepsilon)(\alpha+\mu+\omega)G}{\beta F}, \\ I_* &= \frac{\gamma(\mu-\varepsilon)G}{\beta F},\ R_*=\frac{(\alpha(\delta+\gamma)+\delta(\mu+\omega))G}{\beta F}, \\ F &= \mu(\alpha(\delta+\gamma+\mu-\varepsilon)+\delta(\mu+\omega)+(\mu-\varepsilon)(\gamma+\mu+\omega)), \\ G &= \beta A-\mu(\delta+\gamma+\mu). \end{aligned} \tag{19.2.1}$$

The characteristic equation at this equilibrium is:

$$\lambda^4+\alpha_3\lambda^3+\alpha_2\lambda^2+\alpha_1\lambda+\alpha_0+(\beta_3\lambda^3+\beta_2\lambda^2+\beta_1\lambda+\beta_0)e^{-\lambda\tau}=0. \tag{19.2.2}$$

Where:

$$\begin{aligned} \alpha_0 &= (\alpha_{11}\alpha_{22}-\alpha_{12}\alpha_{21})\alpha_{33}\alpha_{44}+(\alpha_{33}\alpha_{42}-\alpha_{32}\alpha_{43})\alpha_{14}\alpha_{21}, \\ \alpha_1 &= (\alpha_{12}\alpha_{21}-\alpha_{11}\alpha_{22})(\alpha_{33}+\alpha_{44})-(\alpha_{11}+\alpha_{22})\alpha_{33}\alpha_{44}-\alpha_{14}\alpha_{21}\alpha_{42}, \\ \alpha_3 &= \alpha_{11}\alpha_{22}-\alpha_{12}\alpha_{21}+\alpha_{33}\alpha_{44}+(\alpha_{11}+\alpha_{22})(\alpha_{33}+\alpha_{44}), \\ \alpha_3 &= -(\alpha_{11}+\alpha_{22}+\alpha_{33}+\alpha_{44}), \\ \beta_0 &= (\alpha_{11}\alpha_{22}-\alpha_{12}\alpha_{21})\alpha_{44}\beta_{33}+\alpha_{21}(\alpha_{14}\alpha_{42}\beta_{33}+\alpha_{32}\alpha_{44}\beta_{13}), \\ \beta_1 &= \beta_{33}(\alpha_{12}\alpha_{21}-\alpha 11\alpha_{22})-\alpha_{44}\beta_{33}(\alpha_{11}+\alpha_{22})-\alpha_{21}\alpha_{32}\beta_{13}, \\ \beta_2 &= \beta_{33}(\alpha_{11}+\alpha_{22}+\alpha_{44}), \\ \beta_3 &= -\beta_{33}, \\ \alpha_{11} &= -\beta L_*-\mu,\ \alpha_{12}=-\beta S_*,\ \alpha_{14}=\omega,\ \alpha_{21}=\beta L_*,\ \alpha_{22}=\beta S_*-(\delta+\gamma+\mu),\ \alpha_{32}=\gamma, \\ \alpha_{33} &= -(\alpha+\mu),\ \alpha_{42}=\delta,\ \alpha_{43}=\alpha,\ \alpha_{44}=-(\mu-\varepsilon),\ \beta_{13}=\omega,\ \beta_{33}=-\omega. \end{aligned} \tag{19.2.3}$$

And let $i\omega_0$ be a root of this equation. They then formulate the following theorem:

**Theorem 19.1.** *Under the hypotheses $(H_1)$ and $(H_2)$ defined in the paper, the endemic equilibrium is locally asymptotically stable when $\tau\in[0,\tau_0)$ and a Hopf bifurcation occurs at the endemic equilibrium when $\tau=\tau_0$ where:*

$$\tau_0=\frac{1}{\omega_0}\arccos\left(\frac{(\beta_2-\alpha_3\beta_3)\omega_0^6+(\alpha_1\beta_3+\alpha_3\beta_1-\alpha_2\beta_2)\omega_0^4+(\alpha_0\beta_2+\alpha_2\beta_0-\alpha_1\beta_1)\omega_0^2-\alpha_0\beta_0}{(\beta_1\omega_0-\beta_3\omega_0^3)^2+(\beta_0-\beta_2\omega_0^2)^2}\right). \tag{19.2.4}$$

After this they proceed to calculate the normal form of this Hopf bifurcation.

## 19.3 Numerical analysis

In the paper they propose the following values for the parameters:

$$A = 1,\ \beta = 0.35,\ \omega = 0.8,\ \varepsilon = 0.15,\ \mu = 0.35,\ \gamma = 0.05,\ \alpha = 0.35,\ \delta = 0.15. \tag{19.3.1}$$

In the paper they wrote that the steady state is at $P_*(1.5714, 0.6712, 0.0224, 0.5427$ while it is actually at $P_*(1.5714, 0.6981, 0.0233, 0.5643)$. They also say that the Hopf bifurcation critical value is equal to $\tau_0 = 5.3442$ but i found it at $\tau_0 = 6.4588$ with first Lyapunov coefficient equal to $Ł_1 = 5.3256 \cdot 10^{-4}$ which indicates a subcritical Hopf bifurcation where they found a supercritical one. Simulating the solution for $\tau = 5.2482$ and $\tau = 6.5$ we get:

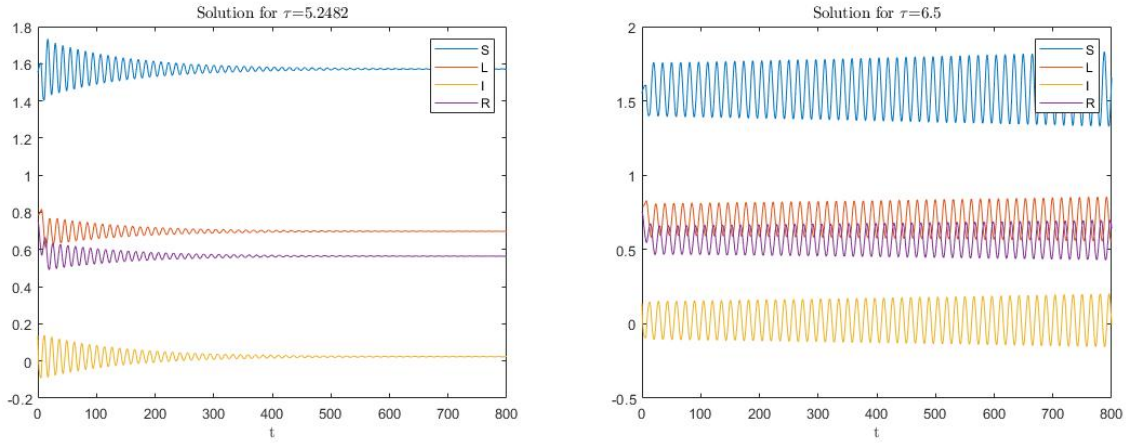



Figure 19.1: Solution of system (18.1.1) for $\tau = 5.2482$ and $\tau = 6.5$.

We can clearly see that in both figures there is no periodic solution or even an unstable one. Now plotting the solution for $\tau = 8 > \tau_0$ we get:

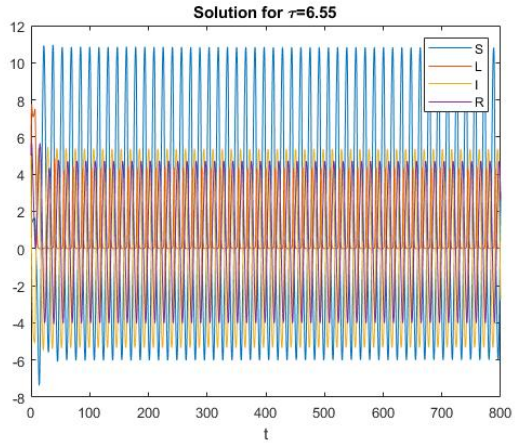



Figure 19.2: Solution for to system (18.1.1) for $\tau = 6.55$.

In this figure we also see a periodic solution after the critical parameter value. In order to further investigate what is happening here we will continue the periodic solution with respect to the time delay, which gives the following figure:

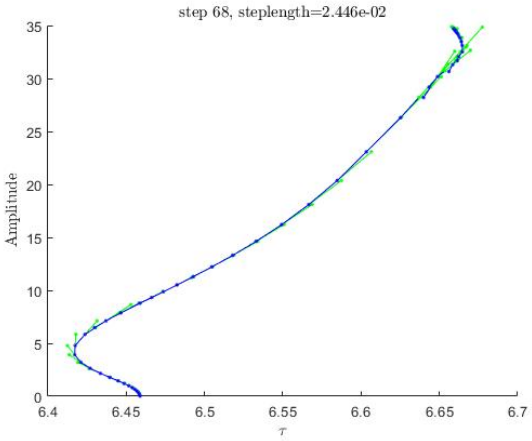



Figure 19.3: Continuation of the periodic solution emanating from the detected Hopf point.

Here we see that there are periodic solutions before the critical parameter value, which confirms a subcritical Hopf bifurcation. As we saw in the simulations there are also periodic oscillations after the critical parameter value. This model is a perfect example of why simulations are not enough to confirm the direction a Hopf bifurcation has, if we would only simulate for $\tau = 5$ and $\tau = 6.55$ we would see the stable solution in (19.1) and then the periodic solution in (19.2) which would indicate a supercritical Hopf bifurcation while normal form calculation using DDE-BIFTOOL detects a subcritical Hopf bifurcation.

# 20 SIQR model with bi-linear incidence rate and a delayed infection rate

## 20.1 Model

In [15] we find the following system:

$$\begin{cases} \dot{S}(t) &= (1-p)b - \beta S(t-\tau)I(t-\tau) - dS(t), \\ \dot{I}(t) &= \beta S(t-\tau)I(t-\tau) - (\delta + d + \alpha_1 + \gamma)I(t), \\ \dot{Q}(t) &= \delta I(t) - (\varepsilon + d + \alpha_2)Q(t), \\ \dot{R}(t) &= \gamma I(t) + pb + \varepsilon Q(t) - dR(t), \end{cases} \tag{20.1.1}$$

Where $S$, $I$, $Q$ and $R$ represent the susceptible, infected, quarantined and recovered populations at time $t$ respectively. The parameter $b$ is the new number of individuals, $p$ is the proportion of individuals who are immunized directly, $\beta$ is the probability of an individual to get infected, $d$ is the natural death rate, $\alpha_1$ and $\alpha_2$ are the death rates due to the virus, $\gamma$, $\lambda$ and $\varepsilon$ are the coefficients of state transmission and $\tau$ is the latent period of the virus. The basic reproduction number of this system is given by:

$$R_0 = (1-p)b\frac{\beta}{d}(\lambda + d\alpha_1 + \gamma). \tag{20.1.2}$$

This model can be used to model computer viruses in a network. Many computer viruses have a latent period delay and a temporary immunity period delay which can be modelled using this system, and computers within a network can be shut off from the network while infected which is why there is a quarantined population. Looking at the standard system (1.2.1) this system is very similar, it only has an extra population for quarantined nodes and a time delay to model the time it takes for a node to be able to infect other nodes.

## 20.2 Results

The model is also analyed in [15]. When $R_0 > 1$ system (21.1.1) has an unique positive equilibrium $D_*(S_*, I_*, Q_*, R_*)$ with:

$$\begin{aligned} S_* &= \frac{\delta + d + \alpha_1 + \gamma}{\beta}, \\ I_* &= \frac{(1-p)b\beta - d(\delta + d + \alpha_1 + \gamma)}{\beta(\delta + d + \alpha_1 + \gamma)}, \\ Q_* &= \frac{\delta I_*}{\varepsilon + d + \alpha_2}, \\ R_* &= \frac{pb + \gamma I_* + \varepsilon Q_*}{d}. \end{aligned} \tag{20.2.1}$$

They define the following parameters:

$$\begin{array}{rcl} a_1 & = & -d,\ a_2 = -(\varepsilon + d + \alpha_1 + \gamma),\ a_3 = \delta,\ a_4 = -(\varepsilon + d + \alpha_2),\ a_6 = \varepsilon,\ a_7 = -d, \\ b_1 & = & -\beta I_*,\ b_2 = -\beta S_*,\ b_3 = -b_1,\ b_4 = -b_2. \end{array} \tag{20.2.2}$$

The characteristic equation of system (21.1.1) at the equilibrium $D_*$ is:

$$\lambda^4 + A_3\lambda^3 + A_2\lambda^2 + A_1\lambda + A_0 + (B_3\lambda^3 + B_2\lambda^2 + B_1\lambda + B_0)e^{-\lambda\tau} = 0. \tag{20.2.3}$$

Where:

$$\begin{array}{rcl} A_0 & = & a_1a_2a_4a_7,\ A_1 = -a_1a_2(a_4 + a_7) - a_4a_7(a_1 + a_2), \\ A_2 & = & a_1a_2 + a_4a_7 + (a_1 + a_2)(a_4 + a_7),\ A_3 = -(a_1 + a_2 + a_4 + a_7), \\ B_0 & = & a_4a_7(a_1b_4 + a_2b_1),\ B_1 = -a_4a_7(b_1 + b_4) - (a_4 + a_7)(a_1b_4 + a_2b_1), \\ B_2 & = & a_1b_4 + a_2b_1 + (a_4 + a_7)(b_1 + b_4),\ B_3 = -(b_1 + b_4). \end{array} \tag{20.2.4}$$

Let (20.2.3) have the purely imaginary roots $\lambda = \pm i\omega_0$. Then the following theorem holds:

**Theorem 20.1.** *If the conditions $(H_1)$-$(H_3)$ defined in [15] hold, then the positive equilibrium $D_*$ is asymptotically stable for $\tau \in [0, \tau_0)$ and system (20.1.1) undergoes a Hopf bifurcation at $\tau = \tau_0$ where*

$$\tau_0 = \frac{1}{\omega_0}\arccos\left(\frac{m_6\omega_0^6 + m_4\omega_0^4 + m_2\omega_0^2 + m_0}{n_6\omega_0^6 + n_4\omega_0^4 + n_2\omega_0^2 + n_0}\right). \tag{20.2.5}$$

*for*

$$\begin{array}{rcl} m_0 & = & -A_0B_0,\ m_2 = A_0B_2 - A_1B_1 + A_2B_0,\ m_4 = A_1B_3 - A_2B_2 + A_3B_1 - B_0,\ m_6 = B_2 - A_3B_3, \\ n_0 & = & B_0^2,\ n_2 = B_1^2 - 2B_0B_2,\ n_4 = B_2^2 - 2B_1B_3,\ n_6 = B_3^2. \end{array} \tag{20.2.6}$$

## 20.3 Numerical analysis

In the paper they propose the following values for the parameters:

$$p = 0.2,\ \beta = 0.1,\ d = 0.01,\ \alpha_1 = 0.01,\ \alpha_2 = 0.02,\ \gamma = 0.5,\ \delta = 0.1,\ \varepsilon = 0.1,\ b = 10. \tag{20.3.1}$$

Using DDE-BIFTOOL we can find an occurrence of Hopf bifurcation at $\tau_0 = 1.3976$, in the paper they found it at $\tau_0 = 1.3265$. The first Lyapunov coefficient of this bifurcation is $L_1 = 0.0028$, so a subcritical Hopf bifurcation. We now simulate the solutions of this system for $\tau = 1.3$ and $\tau = 1.4$, we get the following figures:

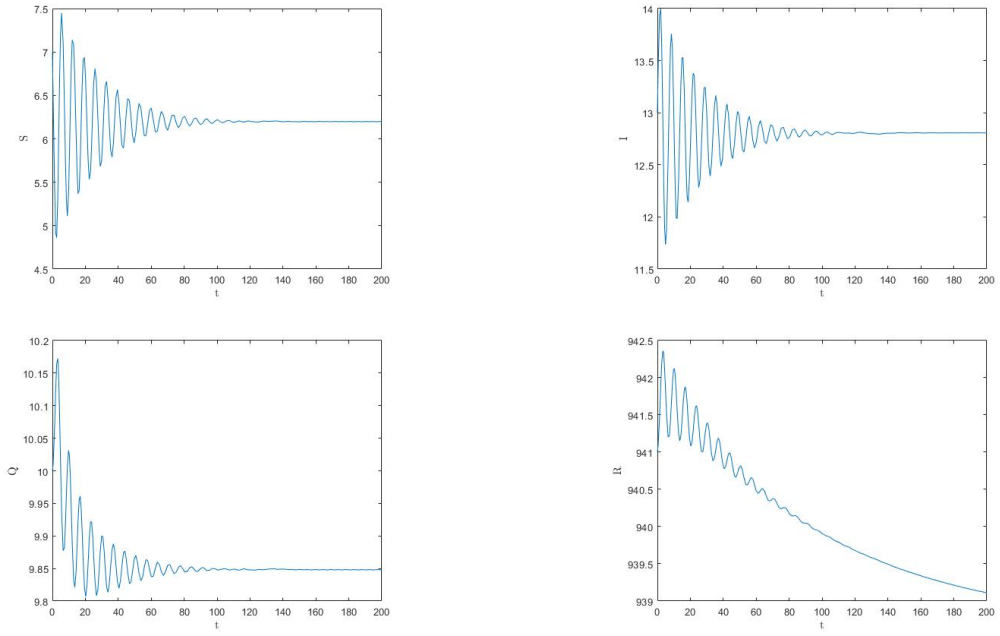



Figure 20.1: Solutions of model (20.1.1) for $\tau = 1.3$.

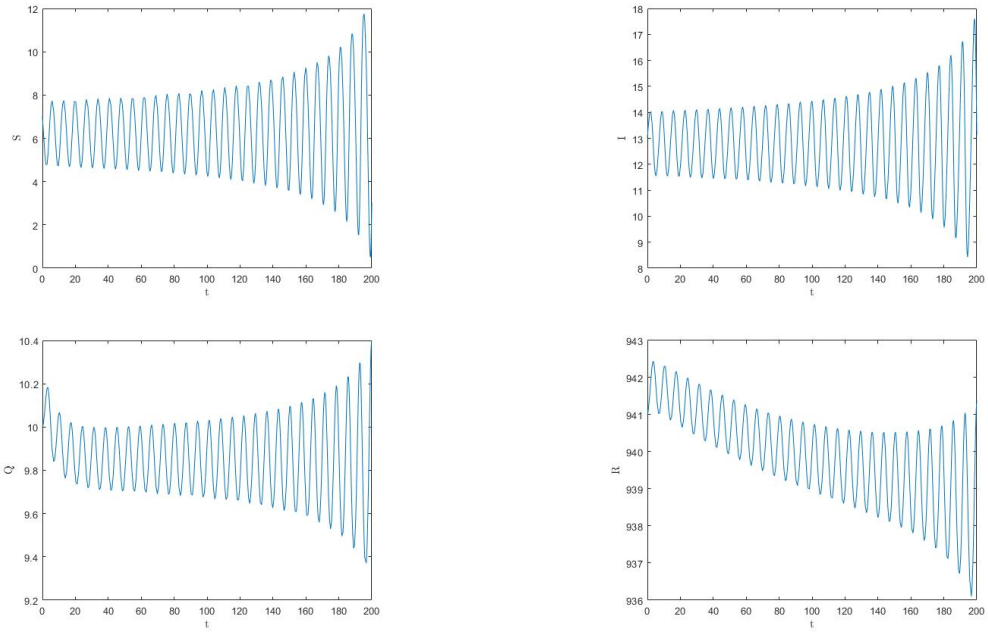



Figure 20.2: Solutions of model (20.1.1) for $\tau = 1.4$.

This again confirms a subcritical Hopf bifurcation. In figure (20.1) we see the solution tend towards the equilibrium and in figure (20.2) we see the solution getting a bigger, so we have an unstable periodic solution. When we continue the periodic solution with respect to $\tau$ we get the following figure:

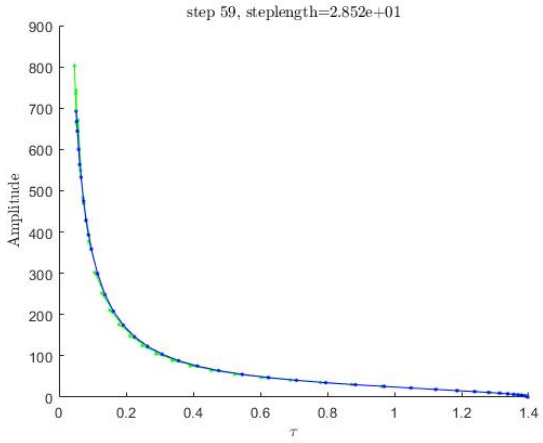



Figure 20.3: Continuation of the periodic solution starting at the critical Hopf point

Here we see the existence of periodic solutions before the critical parameter $\tau_0 = 1.3976$, so this figure confirms the existence of a subcritical Hopf bifurcation.

# 21 SIQR model with noninear incindence rate and delayed infection rate

## 21.1 Model

Consider the following system found in [4]:

$$\begin{cases} \dot{S}(t) &= \Lambda - \dfrac{\beta S(t-\tau)I(t-\tau)}{S(t-\tau)+I(t-\tau)} - dS(t), \\ \dot{I}(t) &= \dfrac{\beta S(t-\tau)I(t-\tau)}{S(t-\tau)+I(t-\tau)} - (c_1+k+d+\mu_1)I(t), \\ \dot{Q}(t) &= kI(t) - (c_2+d+\mu_2)Q(t), \\ \dot{R}(t) &= c_1 I(t) + c_2 Q(t) - dR(t). \end{cases} \tag{21.1.1}$$

Where $S(t)$, $I(t)$, $Q(t)$ and $R(t)$ represent the susceptible, infected, quarantined and recovered populations at time $t$ respectively. $\Lambda$ is the recruitment rate of the population, $d$ is the natural death rate, $\mu_1$ and $\mu_2$ are the extra disease-related death rates and $k$, $c_1$ and $c_2$ are the transfer rates between populations. This model is pretty general and can therefore be used for most kinds of disease which do not have a difference between infection rates of mature and immature populations. Looking at the standard model (1.2.1) this model has an extra quarantined population and instead of the standard incindence rate $\beta SI/N$ this model utilizes the incindence rate $\frac{\beta S(t-\tau)I(t-\tau)}{S(t-\tau)+I(t-\tau)}$ which again accounts for the behavioral change of susceptible individuals as the infectious population grows.

## 21.2 Results

Model (21.1.1) is discussed in [4]. Because the equations for $\dot{Q}$ and $\dot{R}$ are uncoupled from the first two equations the model reduces to:

$$\begin{cases} \dot{S}(t) &= \Lambda - \dfrac{\beta S(t-\tau)I(t-\tau)}{S(t-\tau)+I(t-\tau)} - dS(t), \\ \dot{I}(t) &= \dfrac{\beta S(t-\tau)I(t-\tau)}{S(t-\tau)+I(t-\tau)} - (c_1+k+d+\mu_1)I(t), \end{cases} \tag{21.2.1}$$

This system has a disease-free equilibrium $E_1 = (\Lambda/d, 0)$ and and endemic equilibrium $E_2(S^*, I^*)$, where:

$$\begin{aligned} S^* &= \frac{\Lambda}{\beta + d - (c_1+k+d+\mu_1)}, \\ I^* &= \frac{(\beta - (c_1+k+d+\mu_1))\Lambda}{(c_1+k+d+\mu_1)(\beta+d-(c_1+k+d+\mu_1))}. \end{aligned} \tag{21.2.2}$$

The characteristic equation at the endemic equilibrium is:

$$\lambda^2 + A\lambda + (B\lambda + c)e^{-\lambda\tau} + D = 0 \tag{21.2.3}$$

where

$$\begin{aligned} A &= c_1+k+2d+\mu_1, \quad B = \frac{\beta((I^*)^2+(S^*)^2)}{(S^*+I^*)^2}, \\ C &= (c_1+k+d+\mu_1)\frac{\beta(I^*)^2}{(S^*+I^*)^2} + d\frac{\beta(S^*)^2}{(S^*+I^*)^2}, \quad D = d\frac{\beta(I^*)^2}{(S^*+I^*)^2}. \end{aligned} \tag{21.2.4}$$

Let $\lambda = \omega_\pm i$ be a root of this equation. The following theorem about the stability of the equilibrium $E_2$ holds:

**Theorem 21.1.** *(i) if $A^2 - B^2 - 2D > 0$ and $D^2 - C^2 > 0$ $E_2$ is asymptotically stable for all $\tau > 0$.*

*(ii) if $D^2 - C^2 < 0$, then equilibrium $E_2$ is asymptotically stable when $\tau \in [0, \tau_0^+)$ and unstable when $\tau > \tau_0^+$ and system (21.1.1) undergoes a hopf bifurcation at $\tau = \tau_0$.*

*(iii) if $A^2 - B^2 - 2D < 0$, $D^2 - C^2 > 0$ the equilibrium $E_+$ is stable if:*

$$\tau \in [0, \tau_0^+) \cup [\tau_0^-, \tau_1^+) \cup \ldots [\tau_{k-1}^-, \tau_k^+) \tag{21.2.5}$$

*and unstable when*

$$\tau \in [\tau_0^+, \tau_0^-) \cup [\tau_1^+, \tau_1^-) \cup \ldots [\tau_{k-1}^+, \tau_{k-1}^-), \quad \tau > \tau_k^+. \tag{21.2.6}$$

*And system (21.1.1) undergoes a Hopf bifurcation when $\tau = \tau_j^\pm$.*

*Here $\tau_j^\pm$ is equal to the following:*

$$\tau_j^\pm = \frac{1}{\omega_\pm} \arccos\left(\frac{(C-AB)\omega_\pm^2 - DC}{B^2\omega_\pm^2 + C^2}\right) + \frac{2\pi j}{\omega_\pm}. \tag{21.2.7}$$

After this they calculate the normal form of this Hopf bifurcation.

## 21.3 Numerical analysis

The parameter values they propose are:

$$\Lambda = 10,\ \beta = 0.6,\ d = 0.02,\ c_1 = 0.08,\ k = 0.01,\ \mu_1 = 0.03,\ c_2 = 0.02,\ \mu_2 = 0.04. \tag{21.3.1}$$

When we check this system for Hopf bifurcation we get one at the critical parameter value $\tau_0 = 4.9422$ with a Lyapunov coefficient of $L_1 = -1.9546 \cdot 10^{-4}$ just like they got in the paper. We get the following figures for history $S_0 = 20$, $I_0 = 68$, $Q_0 = 0$ and $R = 0$:

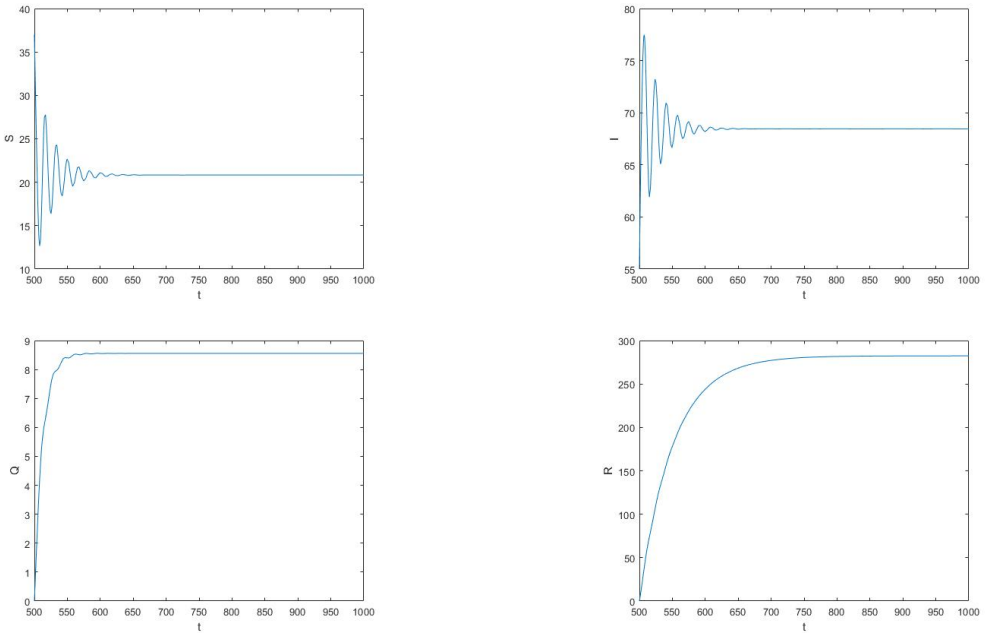



Figure 21.1: Solutions of model (21.1.1) for $\tau = 4$.

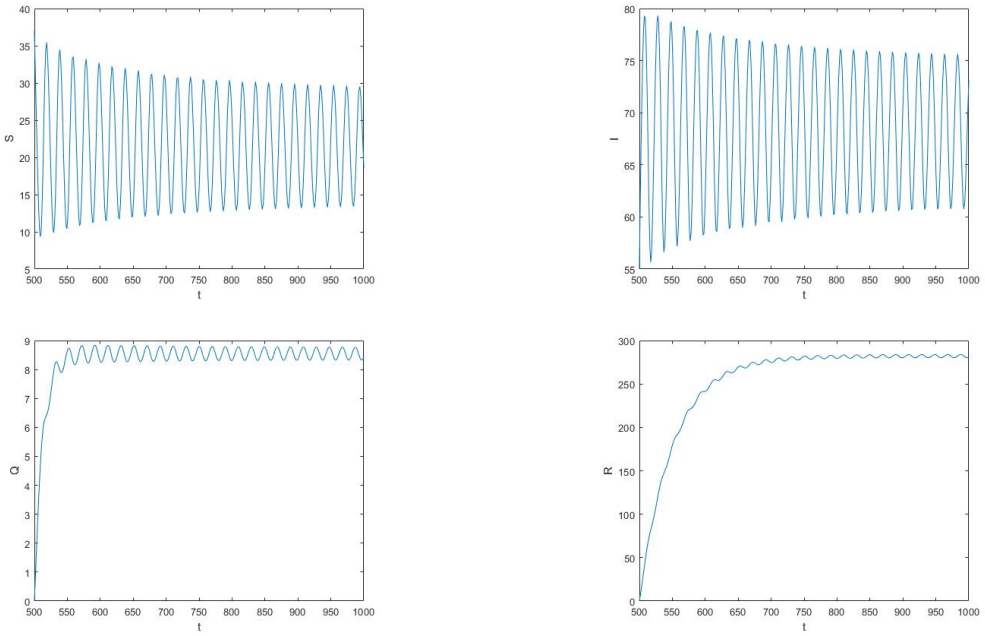



Figure 21.2: Solutions of model (21.1.1) for $\tau = 5$.

We can clearly see the solutions heading towards the equilibrium in (21.1) and in (21.2) we see the solution heading towards a periodic solution. This again confirms the existence of a supercritical Hopf bifurcation. When we continue the periodic solution with respect to $\tau$ we get the following figure:

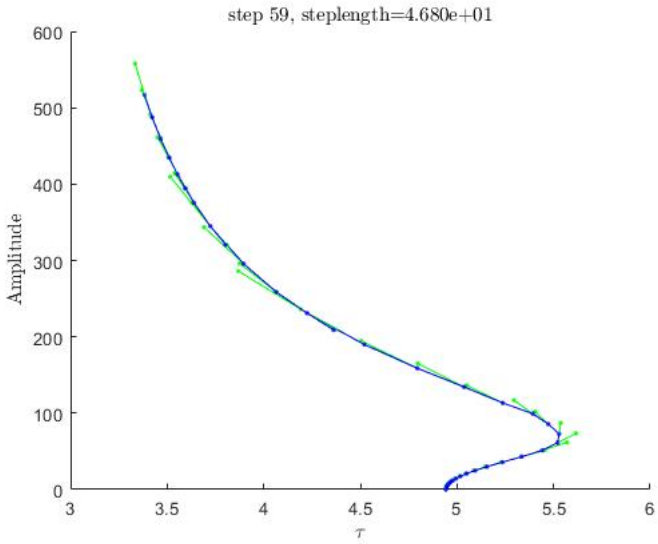



Figure 21.3: Continuation of the periodic solution starting at $\tau_0$

Here we see that even though there are periodic solutions just after the critical parameter value, they do not exist for all $\tau$, but we also have (unstable) periodic solutions that merge with the stable ones near $\tau = 5.5$.

# Part V
# Conclusion

In this thesis we have looked at epidemiological models with time delays. We found that there are many reasons for time delays to be included in the models, and they provide many ways for models to be more accurate. Using time delays does make the models more difficult to analyse and we have found that the solutions include oscillations.

With these oscillations we can expect Hopf bifurcation to occur in a lot of these models. This is especially relevant in epidemiological models because periodic outbreaks of a disease or virus within a computer network are often have to be prevented. Therefore analysing the conditions for which these periodic solutions occur is very important. This is why the notion of Hopf bifurcation is of particular interest. We have looked at the conditions for Hopf bifurcation in delayed differential equations and the formulas to determine the direction of these bifurcations and seen that these conditions and the formulas are very similar to the ones which we defined for ordinary differential equations.

To analyse epidemiological models and determine if Hopf bifurcation is present in these models, we used the DDE-BIFTOOL package for MATLAB. We used this software to analyse 8 different models which can be used to model all kinds of epidemics. We have looked at published research papers discussing the conditions for Hopf bifurcation in these models and applied DDE-BIFTOOL to check the validity of the claims made in these papers by checking their analysis with modern numerical techniques.

We found that in 5 out of 8 cases the analysis was wrong, in two of these cases there was no sign of Hopf bifurcation when analysing them using the software and in the other three cases the direction of the bifurcation was wrong and the parameter value at which these bifurcations occur was also not always consistent with the claims made in the articles. All of these articles had simulations of their models which backed up their analysis but in 5 of these cases this was not sufficient to confirm the results in the paper. The model we discussed in section 19 is of particular interest as we have seen, because there were stable periodic solutions before and after the Hopf bifurcation. So if we would have simulated this model for particular values of the time delay, we could feasibly arrive at the conclusion that a supercritical Hopf bifurcation occurs. Yet using DDE-BIFTOOL we found a subcritical Hopf bifurcation. This illustrates the need for normal form calculation in order to complete Hopf bifurcation analysis done for the models.

In all considered papers, the first Lyapunov coefficient was determined in an outdated way (with a separate computation of the center manifold followed by the normalisation on it), which is also really tedious and labour intensive, so making mistakes in these calculations is relatively easy. Using the explicit formula (10.2.4) for the normal form coefficient, which is also implemented in DDE-BIFTOOL, streamlines this process and reduces chances for mistakes.

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