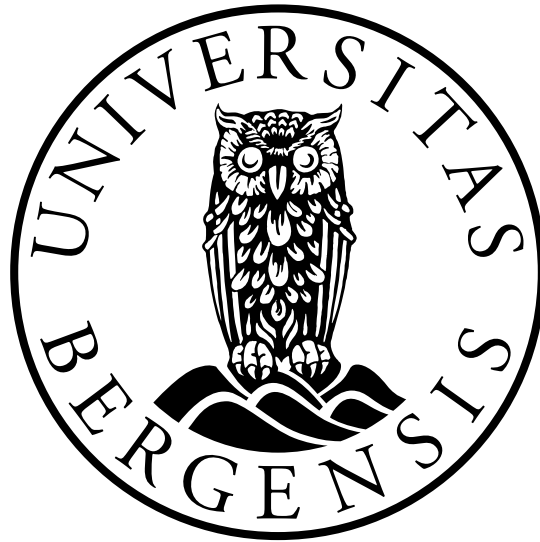# UNIVERSITY OF BERGEN

Department of Mathematics

MASTERS THESIS IN APPLIED MATHEMATICS

Fish louse and treatment options: A mathematical approach

Author: Therese Sjøstrøm Utåker
Supervisor: Guttorm Alendal

August 13, 2018

Contents

Chapter 1

Introduction

In this chapter the main foxus will be what is a fish louse, how the life stages of the louse interact with the host, why louse infection is such a big problemfor the fish farming industry and what kind of measures have been done to stop, or at least slow down, the louse infection.

1.1 Salmon louse

There are different kinds of sea louse but one of the main sea louse that we find in norwegian sea-cages is the salmon louse and from the name it's given that this is a sea louse species that is mainly found on salmon. You can also find this specific type of louse on for example sea trout as well, but it is most commonly found on salmon. The salmon louse is a parasite that lives on the salmon eating the skin causing wounds [1]. These wounds can prevent that the salmon grows as normal and they can also weakens the health of the fish, and in worst case cause death.

The salmon louse life cycle depends on the temperature of the surrounding water, and the warmer the water is, the faster the louse hatches [1]. When the salmon is ready to leave the river and swim to the ocean they are called a smolt and this is the phase where the fish is most susceptible to infection from the sea louse, and especially when they swim past the sea-cages (because of the high rate of infected hosts inside the sea-cages). The salmon is more likely to return to the river if they're infected by few or none sea louse. For the sea trout it is different, because they live along the coast to graze and they are possibly exposed to prolonged infection.

1.1.1 Life stages

The salmon louse has eight stages of life [1] and they are the following:

- Stage I: Naupilius 1

- Stage II: Naupilius 2

- Stage III:: Copepodite

- Stage IV: Chalimus 1

- Stage V: Chalimus 2

- Stage VI: Pre-adult 1

- Stage VII: Pre-adult 2

- Stage VIII: Adult

Stages I-III are the free-living stages of life and this is when the louse swims freely in the water. The critcal stage of all the eight stages is the third stage. When the louse evolves to a copepodite it starts to look for a host to feed on because it will not survive if it can't find a viable host. If the copepodite finds a viable host (the life spawn of a copepodite is from one week to one month, depending on the water temperature), it will change the shell and reach the first of two stages where the louse is attached to the host. This is chalimus 1 and 2 and the louse will feed off the host and the louse will eventually reach the pre-adult stages where the louse moves along the skin of the host. The louse is not fertile before they reach the last stage in the life cycle, the adult stage, but is still mobile and can move around on the fish.

After the louse reaches the adult stage, they can reproduce and new generations of naupilius are released to the water masses. The different stages of life is important and the model used as a basis for this work uses mainly the copepodite stage to describe the free-living louse and the all the stages where the louse is attached to the fish as one. So there is only two stages that is studied, free-living stages and the infection stage.

1.1.2 Finding the spread pattern

To understand how the louse spreads around the coast, and most importantly the sea-cages, we have to undertand how the ocean moves. Since the louse hatches straight into the ocean, the ocean currents will decide if the copepodites will find viable hosts [2].

The ocean currents also vary both horizontally (for example wind and tide) and vertically (weight differences caused by for example rivers that dilute the sea water) and it can therefore be very important to model the ocean currents to understand the local and regional transport [2]. To understand how the louse spreads over distances, one must make model(s) that shows the effect of different currents, and it is important to model the salinity and temperature to understand how the behaviour and growth of the sea louse.

There is at least one questions that is important to ask: how high must the consentration of copepodites be in order to affect the wild salmon. This can be answered by making a more precise model that explains the spread pattern in detail.

1.1.3   Problems caused by the sea louse

Wild salmon is one of the main hosts for the salmon louse, but since the fish in the sea-cages are clumped together the louse will most likely infect the caged fish at a faster rate then the wild fish [3]. This will in turn increase the number of wild fish that gets infected [3]. In other words, the contribution from the caged fish will most likely be higher then the contribution from the wild fish when we look at the exposure to infection. That is one of the reasons that the sea trout is more prone to infection than salmon when it comes to wild fish.

Reaserchers have found that sea louse causes higher levels of stress and that the louse lowers the immunological capacity in the infected hosts. In particular the transistion from attached to mobile stage lowers the immunological capasity [3]. Effects that appear later in the hosts life cycle are decreased growth, decreases the ability to swim, affects the behaviour and increased mortality rate. This can in turn make the fish more vulnerable to predation because of the smaller size. Other natural variations, for example resistance to medicine, makes it harder to see if the wild fish dies of sea louses infection when it returns to river to spawn. Therefore it will be harder to calculate what causes the reduction in the fish population.

All these problems causes huge problems for the companies that owns the sea-cages and the loses caused by sea louse costs around 500 million Norwegian kroner annullally [4] to give one example. This includes direct loses (dead fish), chemicals to counter the infection and extra to work to delouse the fish. The fish also haves a lower weight and size because the louse causes the fish to postpone the maturation [3] and also reduce the potential for reproduction. Finding solutions that can help against these problems is therefore important to continue the reaserch for a cure against the sea louse, or at least help to lower the exposure to infection in the sea-cages and hopefully reduce the number of infected fish.

1.2   The purpose of this thesis

As stated in the previous section there is several problems caused by the sea louse when it infects the fish and it will be important to find measures to control the number of louse that can infect the fish in the sea-cage. The common method to stop further infection by sea louse is to use chemicals [5] and eventually find a vaccine to stop the louse permanently, but the problem is that the louse is attached to the skin of the fish. It is therefore hard to find a vaccine that cures the fish and prevents further infection to the fish in sea-cages. This

thesis will then look at one model that tries to explain the growth and decay of a louse population.

The article that this paper is based upon is: *Critical thresholds in sea lice epidemics: evidence, sensititvity and subcritcal stimation* (2012) [6] and the first steps is to explain the method that will be used in this work, before analysing the system used in the article to see if the results are accurate. The main focus for this paper will be to look at the harvest and treatment variable and how changing this will alter the system to become more or less stable. We will also in the discusion part of the paper look into other types of models to see how they want to stop the sea louse problem, and also use linear control theory to see if this system is even viable in a real life situation.

Chapter 2

Method

In this chapter we will look at the original model that this thesis is based on, and also look at the different mathematical approaches to analyze the system. Chapter 3 will be the analysis of the system that this theises is loks into, and this analysis will use the mathematical methods written in this chapter.

2.1 The original model

The basis for this thesis is to look at the model written by Frazer et al [6], where we will describe the model and how changing the parameters can alter this system. We will in this subsection give the parameters, both the definition and how the parameters relate to each other, and how we can use this system in a real-life situation. All the following data in this section (The original model) is found in [6].

2.1.1 Parameters and variables

There are two variables in this model, P and L, where P is the number of louse on the farmed fish and L is the number of free-living copepodites. These two variables will change over time, depending on different parameters related to for example the natural mortality rate of the louse. These two variables is set to vary in time, but the area where the sea-cage(s) is set to be fixed so there is no change in where the fish are located. The area can for example be 3-4 sea-cages located together with surrounding mountains and ocean.

The parameters is given in the following table:

Parameter	Meaning
F	Number of fish in the sea-cages
L_0	Free-living copepodites from wild fish
μ	Louses natural mortality rate, not caused by harvest or treatment
h = $h_1 + h_2$	Louse mortality rate, caused by harvest (h_1) and treatment (h_2)
γ	mortality rate of free-living copepodites
β	transmission coefficient, captures per copepodites
λ	natality, how many copepodites each female louse produces
ψ	propability for survival to adulthood for attached copepodites

Table 1: Parameters used in this model

All of these parameters is set to be constant in this model, but they could easily be estimated to depend on other variables and parameters. The F parameter stands for the total number of fish in sea-cages within the given area we have, and does not include wild fish. One interesting parameter is the background infection process L_0 and tells us how many copepodites comes from wild fish that surrounds the sea-cages. These wild fishes with contagious pathogens can come from either rivers or ocean currents and L_0 can easily be changed to depend on either of these. Three of the parameters are mortality rate parameters, μ, h and γ, and according to model it is set to one louse death (both from fish in sea-cages and free-living copepodites). The birth rate λ tells us how many copepodites each female louse can birth and both the transmission coefficient, β, and the settlement probability, ψ, can tell us how likely it will be that a copepodite will attach and survive to adulthood. So these parameters are regarded as positive factors for the growth of the louse population. The parameters and variables are connected in the model and how they are connected will be explained in the following subsection.

2.1.2   Summary of the model

The model is written as:

$$\frac{dP}{dt} = \beta\psi\left(L + L_0\right)F - \left(\mu + h\right)P \qquad (2.1)$$

$$\frac{dL}{dt} = \lambda P - \gamma L - \beta L F \qquad (2.2)$$

This is two differential equations describing if (in this case) a population grows or decays over time, through derivatives in the system ($\frac{dP}{dt}$ and $\frac{dL}{dt}$). We will have growth in populations if the derivatives are positive and decay if the derivatives are negative. Since this system is considerd to be a real-life situation, we will have that all the parameters will be positive.

This system has two populations that changes changes over time, but none

of the parameters depends on time and they will therefore stay constant (unless they're changed to depend on time or on the variables P and L that depends on time). We will in this thesis analyze the system withconstant parameters, before changing the harvest and treatment parameter h to depend on the variable P.

The combination of parameters and variables gives a picture of how these types of systems work and onw way is to look at each part of the equations. The following can be read from the equations:

$$\beta\psi\left(L+L_0\right)F = \text{ Change in P caused by number of free-living copepodites that attaches to fish} \tag{2.3}$$

$$(\mu + h)\,P = \text{ Death rates of P} \tag{2.4}$$

$$\lambda P = \text{ Number of copepodites from female louses per unit time} \tag{2.5}$$

$$\gamma L = \text{ Number of free-living copepodites that dies} \tag{2.6}$$

$$\beta L F = \text{ Number of copepodites that attaches itself to a host} \tag{2.7}$$

Since every part of the equations is regarded to be positive, the sign (+ or -) will decide if the parts will give a growth or decay to the system.

Frazer et al. [6] main goal is to find the critical threshold for the value F and they call this the critical stocking level for the number of fish that can be in the sea-cages to avoid an epidemic. To find this threshold they first find the net reproductive level, R_0, and this is set to be the expected number each female louse produces. This value can be read from the equations as the following[6]:

$$R_0 = \frac{\lambda}{\mu + h}\frac{\beta F}{\beta F + \gamma}\psi \tag{2.8}$$

The first factor in R_0 is the natality rate (λ) of a female louse multiplied by the expected lifetime of an adult female louse ($(\mu + h)^{-1}$), and this factor will then be the expected number of copepodites produced by a female louse over a lifetime.

The next factor will be the probability that the copepodites will attach to a host fish instead of dying, and the product of these two factors will together be the total number of louse produced that also will attach to a host fish. The last factor is the probability that they will survive to adulthood.

9

To calculate the critical threshold value for this model, they see that if $R_0 < 1$, the population will then either die out or stay at a low level and not cause an epidemic. But if $R_0 > 1$, the population will grow and can cause an epidemic because each female will produce more than one female offspring. The critical value for the population is when $R_0 = 1$ and by setting this we find that the critical stocking level is:

$$F_x = \frac{\gamma \left(\mu + h\right)}{\beta \left(\lambda\psi - \mu - h\right)} \tag{2.9}$$

By analysing this equation using equilibrium values they found that this system is sensitive to the transmission rate, and they also found out that the system is also sensitive to temperature and salinity levels. The result from these analyses are that the location of the sea-cages can be important to reduce the transmission rate of the system. The only values that we humans easily can control are the number of fish we have in the sea-cages, how much treatment is used and how many fishare harvested.We will see if the system can be controlled by focusing on the mortality rate caused by harvest and treatment parameter h.

2.2 Nondimensionalization

The two equations in this model has several parameters and in the original paper they have estimated and calculated several of the parameters, but instead you can nondimensionalize the equations [7]. By doing that one can make new parameters to include several of the given parameters in Table 1. This change can make experiments easier to handle in simulations and can then be compared to real-life situations to see if the dimensionless parameters give an accurate solution. For example, one can make a 2D-plot of a model with 5 dimensions [7] (but to get the more accurate result, one must conduct several variations in all 5 dimensions).

First we have to find out with of our parameters and variables have dimensions, and that is L, P, t and L_0. They can be re-written as:

$$P = \bar{P}p \tag{2.10}$$

$$L = \bar{L}l \tag{2.11}$$

$$t = T\tau \tag{2.12}$$

$$L = \bar{L}l_0 \tag{2.13}$$

Where the parameters p, l l_0 and τ are the non-dimensionalized parameters and $\bar{P}\bar{L}$ and T are characteristic values. Including these parameter changes to the original equations, we can re-write the equations to:

$$\frac{dp}{d\tau} = \frac{T\beta\psi F\bar{L}}{\bar{P}} \left(l + l_0\right) - T\left(\mu + h\right)p \tag{2.14}$$

$$\frac{dl}{d\tau} = \frac{\lambda \bar{P} pT}{\bar{L}} - T\left(\gamma + \beta F\right) l \tag{2.15}$$

We make the following choices:

$$T\left(\gamma + \beta F\right) = 1 \rightarrow T = \frac{1}{\gamma + \beta F} \tag{2.16}$$

$$\frac{\lambda \bar{P}}{\bar{L}\left(\gamma + \beta F\right)} = 1 \rightarrow \frac{\bar{L}}{\bar{P}} = \frac{\lambda}{\gamma + \beta F} \tag{2.17}$$

Now the equations look the following:

$$\frac{dp}{d\tau} = \frac{T\beta\psi F\lambda}{\left(\gamma + \beta F\right)^2}\left(l + l_0\right) - \frac{1}{\gamma + \beta F}\left(\mu + h\right) p \tag{2.18}$$

$$\frac{dl}{d\tau} = p - l \tag{2.19}$$

The second equation is now much easier to handle, and the following parameters can be chosen to make the first equation easier as well:

$$\kappa = \frac{\beta\psi\lambda F}{\left(\gamma + \beta F\right)^2} \tag{2.20}$$

$$\alpha = \frac{\mu}{\gamma + \beta F} \tag{2.21}$$

where we also changed the harvest and treatment parameter h to be the following

$$H = \frac{h}{\mu} \tag{2.22}$$

The end result is the following two equations:

$$\frac{dp}{d\tau} = \kappa\left(l + l_0\right) - \alpha\left(1 + H\right)) p \tag{2.23}$$

$$\frac{dl}{d\tau} = p - l \tag{2.24}$$

These are the two equations we will use in our calculations, with two variables p and l and down to three parameters: H, κ and α.

2.3 Classification of equilibrium points

In this section we describe how to find where there is constant stable solutions to our system, in other words where the equilibrium points are located. We will also describe the way of finding the eigenvalues and eigenvectors and use them to say if the solution we have found will be stable or not.

2.3.1 Linearization

To find the eigenvalues to the system we first consider that our system is a nonhomogenous system (systems that includes parts without the variables, in this system it's κl_0) and we will aslo introduce a value Hp so we will have a nonlinear system, and we need to make the system "easier" to work with. The method to make nonlinear and nonhomogenous systems "easier" is called linerazation[8], where we move the equilibrium point to the origin and therefore elimante the nonhomogenous part (κl_0 and also make the nonlinear system linear. The equilibrium point is assumed to be (δ, ξ), and then we introduce new variables as

$$u = p - \delta \tag{2.25}$$

$$v = l - \xi \tag{2.26}$$

Since the values δ and ξ are constants and

$$p = u + \delta \tag{2.27}$$

$$l = v + \xi \tag{2.28}$$

we can get the following

$$\frac{du}{d\tau} = \frac{p - \delta}{d\tau} = \frac{dp}{d\tau} = f(u + \delta, v + \xi) \tag{2.29}$$

$$\frac{dv}{d\tau} = \frac{l - \xi}{d\tau} = \frac{dl}{d\tau} = g(u + \delta, v + \xi) \tag{2.30}$$

This equals to

$$\frac{du}{d\tau} = f(u + \delta, v + \xi) \tag{2.31}$$

$$\frac{dv}{d\tau} = g(u + \delta, v + \xi) \tag{2.32}$$

This is used to eliminate the nonlinear terms and we will be left with the best linear approximation for the problem. By using the Taylor approximation of the equations (and neglecting the terms that would give nonlinear terms), one is left with the following terms

$$f(u + p_0, v + l_0) \sim f(\delta, \xi) + \left[\frac{\partial f}{\partial p}(\delta, \xi)\right] p + \left[\frac{\partial f}{\partial l}(\delta, \xi)\right] l \tag{2.33}$$

$$g(u + p_0, v + l_0) \sim g(\delta, \xi) + \left[\frac{\partial g}{\partial p}(\delta, \xi)\right] p + \left[\frac{\partial g}{\partial l}(\delta, \xi)\right] l \tag{2.34}$$

So using the fact that $f(\delta, \xi) = g(\delta, \xi) = 0$, one will get the following matrix that is called the Jacobian matrix corresponding to the linearized system:

$$\begin{bmatrix} \frac{du}{d\tau} \\ \frac{dv}{d\tau} \end{bmatrix} = \begin{bmatrix} \frac{\partial f}{\partial p}(\delta, \xi) & \frac{\partial f}{\partial l}(\delta, \xi) \\ \frac{\partial g}{\partial p}(\delta, \xi) & \frac{\partial g}{\partial l}(\delta, \xi) \end{bmatrix} \begin{bmatrix} u \\ v \end{bmatrix} \tag{2.35}$$

2.3.2 Equilibrium points

We want a system that doesn't change over time, no deacy or growth, and for that to happen for our system we have to have that:

$$\frac{dp}{d\tau} = 0 \tag{2.36}$$

$$\frac{dl}{d\tau} = 0 \tag{2.37}$$

This will be true for our system if:

$$\kappa\,(l + l_0) - \alpha\,(1 + H))\,p = 0 \tag{2.38}$$

$$p - l = 0 \tag{2.39}$$

We will have a system in equilibrium if equations (2.38) and (2.39) are satisfied at the same time [8]. The corresponding values for p and l will then be the equilibrium points for the system and the next step is to classify the different equilibrium points.

By classifying the equilibrium points we can find out if the system will be stable or unstable over time or not. The general solution of the equations and are

$$p = l \tag{2.40}$$

Using this in equation and we get the following

$$p = \frac{\kappa l_0}{\alpha\,(1 + H) - \kappa} \tag{2.41}$$

In our system we will change the values for H value to see if this changes the system to reach equilibrium faster and then it is interesting to see what happens around the equilibrium points.

The next step is therefore to classify the equilibrium points. We can rewrite the equations (2.23) and (2.24) to the following

$$\begin{bmatrix} \dot{p} \\ \dot{l} \end{bmatrix} = \begin{bmatrix} -\alpha\,(1 + H) & \kappa \\ 1 & -1 \end{bmatrix} \begin{bmatrix} p \\ l \end{bmatrix} + \begin{bmatrix} \kappa l_0 \\ 0 \end{bmatrix} \tag{2.42}$$

The following matrix is the Jacobin matrix [9] found in section 2.3.2:

$$\tilde{J} = \begin{bmatrix} -\alpha\,(1 + H) & \kappa \\ 1 & -1 \end{bmatrix} \tag{2.43}$$

The Jacobian matrix (also called the coefficient matrix for linear systems) is useful to classify the equilibrium points because we can look for straight-line solutions that solves

$$\tilde{A}V = \omega V \tag{2.44}$$

Here we have that ω equals to a scalar called eigenvalues to the coefficient matrix $\tilde{A}$ and these vaules tells ut what will happen around the equilibrium point(s). The eigenvalues correspond to vectors called eigenvectors and is noted as V in the equation. To use this to calculate the eigenvalues and eigenvectors, we first need to re-write equation

$$\omega V = \omega I V = \omega \begin{bmatrix} 1 & 0 \\ 0 & 1 \end{bmatrix} V \Rightarrow \left(\tilde{A} - I\omega \right) V = 0 \tag{2.45}$$

We know from algebraic theory that if we want the last statement to be true for a nonzero solution for V, we need that the determinant of the coefficient matrix to be equal to zero. This means that:

$$det\left(A - I\omega\right) = 0 \tag{2.46}$$

This is called the characteristic equation and it will give the characteristic polynomial of the solution

$$\omega^2 + \left(\alpha\left(1 + H\right) + 1\right)\omega + \alpha\left(1 + H\right) - \kappa = 0 \tag{2.47}$$

How the eigenvalues are defined for each case will in turn tell us how stable the system is near the equilibrium point(s), and the classification of the equilibrium points is the next section.

2.3.3   Classification of the equilibrium points

After finding the equilibrium points and linearization of the system we can classify the equilibrium points by find the eigenvalues and the corresponding eigenvectors. The Jacobian matrix can be used to find the different eigenvalues by the methods written in section 2.3.1 and 2.3.2.

We will get a set of eigenvalues for each situation (for the nonlinear system we will get two sets of eigenvalues) and the following is found in book written by Jordan and Smith [9] to classify the eigenvalues. For real values of ω_1 and ω_2 we have that:

$$\omega_1 < \omega_2 < 0 \rightarrow \text{ Stable node} \tag{2.48}$$

$$0 < \omega_1 < \omega_2 \rightarrow \text{ Unstable node} \tag{2.49}$$

$$\omega_1 < 0 < \omega_2 \rightarrow \text{ Saddle point, always unstable} \tag{2.50}$$

For the complex eigenvalues we will have that the eigenvaules are the following:

$$\omega = \eta \pm i\zeta \tag{2.51}$$

where η stands for the real part of the eigenvalue and ζ real numbers corresponding to the imaginary part of the eigenvalue. The classification will then be the following

$$\eta < 0 \rightarrow \text{ Stable spiral} \tag{2.52}$$

$$\eta > 0 \rightarrow \text{ Unstable spiral} \tag{2.53}$$

$$\eta = 0 \rightarrow \text{ Centre} \tag{2.54}$$

By knowing how the behaviour of the system is around the equilibrium points it will be easier to draw a phase portrait over the system.

This can show how the system will behave for different initial conditions and will give a picture of how the solution will be. This is useful for our system where we look at how the treatment and harvest value (H) will affect the system and also if we can pressure the system to go faster towards equilibrium be increasing the complexity of this value. Instead of calculating for many possible variables, one can use a phase portrait to give a picture over the system and how it changes over time.

2.4   MatLab code(s)

The solver used to find numerical solutions is for this system is ode45 in MatLab [10]. This solver solves system like the one used in this thesis where you specify the initial conditons and uses that to calculate the solution. The method the program uses with this solver is the explicit Runge-Kutta formula called the Dormand-Prince pair. This is also a single-step solver and this means that the solver only needs the immediate preceding time step $y\,(t_{n-1})$ to calculate the solution we want $y\,(t_n)$.

Chapter 3

Analysis and results

The following chapter will be dedicated to analysis of our model and we will have three different cases for how we decide the mortality rate caused by havest and treatment: 1) no harvest and treatment in the system (H = 0), 2) it will be constant over time (H = H_0) and 3) there is a linear dependence between H and the number of louse found on fish (H = H_0p).

For each case we will first show how the equilibrium points will be defined, following that we fill find the eigenvalues and eigenvectors and finally we will show a plot over the system. For every case we will use the nondimensional system we found in the method chapter and adjust the H value:

$$\frac{dp}{d\tau} = \kappa \left(l + l_0\right) - \alpha \left(1 + H\right) p \tag{3.1}$$

$$\frac{dl}{d\tau} = p - l \tag{3.2}$$

The parameters will be estimated to fit the solutions so it will be clear where the equilibrium point(s) are located and also so we can see how the phase portrait around the equilibrium point will be located. In the original model they have estimated the parameters using real-life data [6] and this could also be a possibility in regards to how the model is written in this thesis.

3.1   Case 1: No treatment and Harvest

This case represents when we have that H = 0 and there is no mortality rate caused by harvest and treatment. For this case we have that the nondimensional equations are the follwing:

$$\frac{dp}{d\tau} = \kappa \left(l + l_0\right) - \alpha p \tag{3.3}$$

$$\frac{dl}{d\tau} = p - l \tag{3.4}$$

To find the equilibrium point of the case we will use the method described in the method chapter. We write this at the following

$$\frac{dp}{d\tau} = \frac{dl}{d\tau} = 0 \tag{3.5}$$

This gives us that:

$$l = p \tag{3.6}$$

$$p = \frac{\kappa l_0}{\alpha - \kappa} \tag{3.7}$$

For the sake of this analyze, we will include both the negative and positive equilibrium solutions and from the equation we see that:

$$\alpha > \kappa \rightarrow p > 0 \tag{3.8}$$

$$\alpha < \kappa \rightarrow p < 0 \tag{3.9}$$

To find the eigenvalues and the corresponding eigenvectors we have to find the Jacobian matrix of the system by using the method of linearzation. Following the procedure from the method chapter, we have that the Jacobian matrix for this system will be the following:

$$\tilde{J} = \begin{bmatrix} -\alpha & \kappa \\ 1 & -1 \end{bmatrix} \tag{3.10}$$

This will be the same as the coefficient matrix because the system we have for H = 0 is a linear system. We will then get the following linearized system:

$$\begin{bmatrix} \frac{du}{d\tau} \\ \frac{dv}{d\tau} \end{bmatrix} = \begin{bmatrix} -\alpha & \kappa \\ 1 & -1 \end{bmatrix} \begin{bmatrix} u \\ v \end{bmatrix} \tag{3.11}$$

This we can use in the characteristic equation to find the eigenvalues and this will give us the following:

$$det \begin{bmatrix} -\alpha - \omega & \kappa \\ 1 & -1 - \omega \end{bmatrix} = 0 \rightarrow \omega^2 + (\alpha + 1)\omega + \alpha - \kappa = 0 \tag{3.12}$$

Solving this equation gives the eigenvalues:

$$\omega_{1,2} = \frac{1}{2}\left[-(\alpha + 1) \pm \sqrt{(\alpha + 1)^2 + 4(\kappa - \alpha)} \right] \tag{3.13}$$

By looking at section 2.3.3, we have the following two situations:

$$\alpha > \kappa \rightarrow \text{ stable node} \tag{3.14}$$

$$\alpha < \kappa \rightarrow \text{ unstable saddle point} \tag{3.15}$$

17

Inserting the eigenvalues back into the characteristic equation we can calculate eigenvectors corresponding to the eigenvalues. The eigenvectors will tell us how the orientation around the equilibrium points will be, and they will therefore be useful for each case that we look upon in this thesis. The eigenvectors are for this case:

$$
\begin{bmatrix} -\alpha - \frac{1}{2}\left[-(\alpha+1) \pm y\right] & \kappa \\ 1 & -1 - \frac{1}{2}\left[-(\alpha+1) \pm y\right] \end{bmatrix} \begin{bmatrix} V_1 \\ V_2 \end{bmatrix} = \begin{bmatrix} 0 \\ 0 \end{bmatrix} \tag{3.16}
$$

where:

$$
y = \sqrt{(\alpha+1)^2 + 4(\kappa - \alpha)} \tag{3.17}
$$

So this will give us four different eigenvalues (two for each case depending on what α and κ will be as found above). The function to find the eigenvectors can be written as:

$$
V_1 = \left(1 + \frac{1}{2}\left[-(\alpha+1) \pm \sqrt{(\alpha+1)^2 + 4(\kappa-\alpha)}\right]\right) V_2 \tag{3.18}
$$

What will this mean for the original equation? Changing from the non-dimensionlized parameters to the original parameters one can see that the following correlation is apparent:

$$
\alpha > \kappa \rightarrow \frac{\mu}{\gamma + \beta F} > \frac{\beta \psi \lambda F}{(\gamma + \beta F)^2} \rightarrow \mu > \frac{\beta \psi \lambda F}{(\gamma + \beta F)} \tag{3.19}
$$

The meaning of this is that if there is to be a stable solution where there are no growth in neither the number of lice on farmed fish or number of free-living copepodites from lice on wild fish ($\frac{dP}{dt} = \frac{dL}{dt} = 0$), then the louse mortality rate caused by natural causes (not by harvest or treatment) must be larger then the rate of the probability that a copepodite will attach to a host fish ($\frac{\beta F}{\gamma + \beta F}$) times the probability that an attached copepodite will survive to adulthood, ψ, and also times the natility to the louse λ. This is in accordance with Frazer [6].

Another way to look at this is:

$$
\frac{\beta F}{\gamma + \beta F} \cdot \frac{\lambda}{\mu} \cdot \psi < 1 \rightarrow R_0 < 1 \tag{3.20}
$$

This equation is a form of net reproduction value, R_0, from the original model where the factor $\frac{\lambda}{\mu}$ will be the natility of the louse times multiplied with the expected life time of the louse μ^{-1}. The reproductive rate will in a case where it's below 1 mean that every female louse will reproduce less louse then that dies from natural causes and the louse population will in this case most likely slowly die out or come to an level of endemic abundance.

3.1.1 Phase portrait

After calculating all the eigenvalue and eigenvectors can we now draw a plot that shows how the system behaves around the equilibrium point and this is called a phase portrait or diagram for the system [**?**]. By using the ode45 solver in MatLab we get the following figure for the first situation where $\alpha > \kappa$:
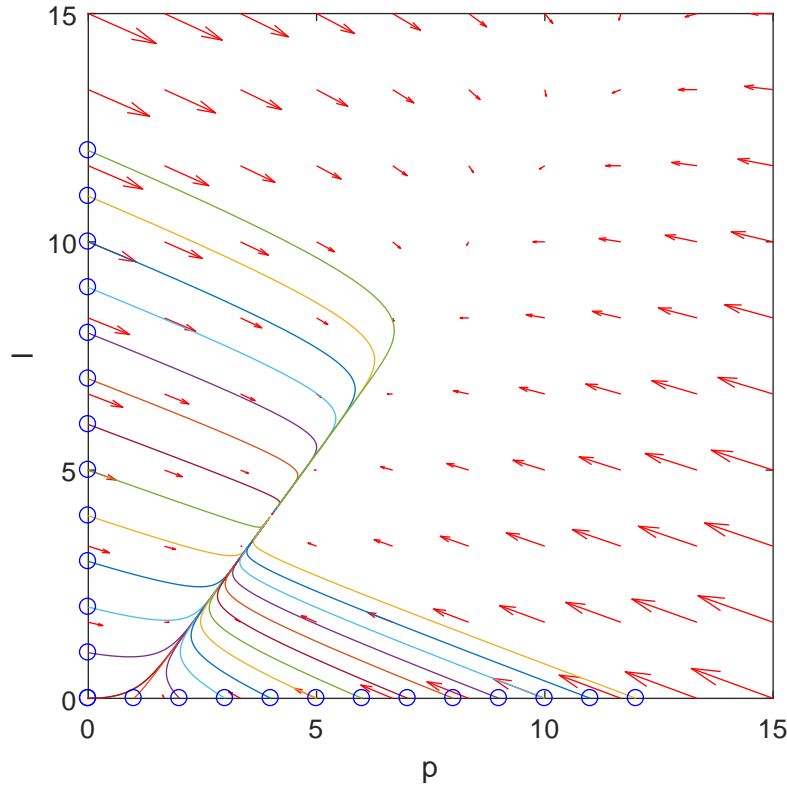

Figure 3.1: $\alpha = 3$, $\kappa = 2$, $H_0 = 0$ and $l_0 = 2$

For figure 3.1 we have ploted the equilibrium point that we will get the following conditions: $\alpha = 3$, $\kappa = 2$, $H_0 = 0$ and $l_0 = 2$. This means that we have ploted the first situation for case where there is no harvest or treatment that is affecting the mortality rate for the louse ($H = 0$), and we also have that $\alpha > \kappa$ so we have a stabil node as a solution for this equilibrium point that is given as (p,l) = (4,4) (from $\frac{dp}{d\tau} = \frac{dl}{d\tau} = 0$).

The way we see that this is a stable node from the plot is the curves form around the equilibrium point and they all curve to the solution. This means that the system will tend to go towards equilibrium along an eigenvector and the louse population (both the free-living copepodites and the louse on farm fish) will remain at a constant level until it is disturbed from other sources then

the natural mortality rate and natality.

The eigenvectors for this case shows that we will get a decrease in the population as time progresses and the system will remain stable under these conditions or go towards zero as we want it to do eventually. The louse population will either die out or stay at an endemic level to the population is disturbed in some way, in the case where it is only controlled by the fact that the natural mortality rate is higher then the reproduction of new louse. Where the endemic level will be depends on what starting values one chooses, but as long as $\alpha > \kappa$ the louse population will never exponentially grow.

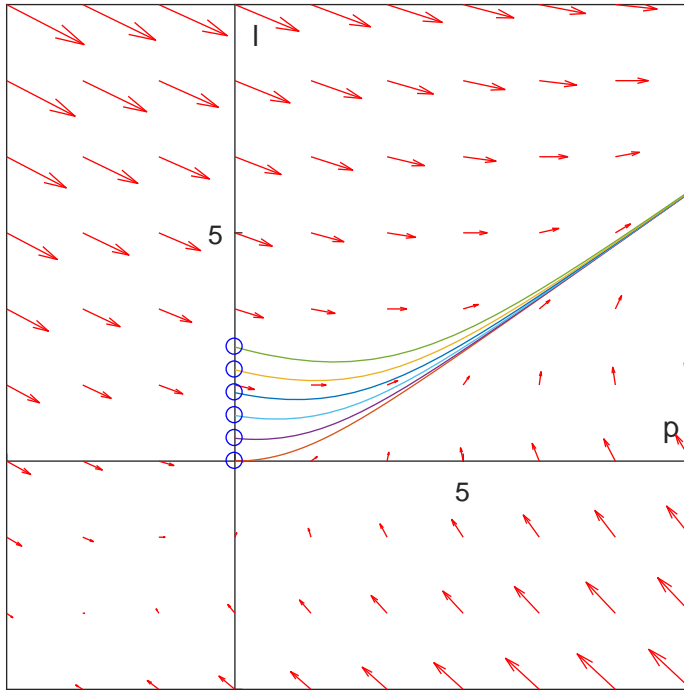The next figure 3.2 will show how the phase portrait will look for the situation where $\alpha < \kappa$:

Figure 3.2: $\alpha = 1$, $\kappa = 2$, $H = 0$ and $l_0 = 2$

Figure 3.2 shows the system with the following conditions: $\alpha = 1$, $\kappa = 2$, $H = 0$ and $l_0 = 2$. There is still no harvest or treatment that affects the louses mortality, but now we have that $\alpha < \kappa$. The solution will be an unstable saddle point and we can see it from the figure that the curves for different initial conditions that they tend to increase. The equilibrium point (p,l) = (-4,-4) is in the negative part of the plot, but it is interesting to see what happens if you start with a positive p and l value and the tendency is that the solutions

follow the unstable eigenvector. From both the eigenvectors and the plots we will see that the populations will just grow exponentially from small values until it will become a pandemic. This means that if we have condtions where $\alpha < \kappa$ and no harvest or treatment, there will be an exponentially growth in the louse populations until it will become uncontrollable.

3.2 Case 2: Constant treatment and harvest effort

In this case we have that the mortality rate caused by harvest and treatment will be constand and this will mean that $H = H_0$, where H_0 is a positive constant. We will use the same approach as for $H = 0$ and we will have that nondimensional equations are the following:

$$\frac{dp}{d\tau} = \kappa\,(l + l_0) - \alpha\,(1 + H_0)\,p \tag{3.21}$$

$$\frac{dl}{d\tau} = p - l \tag{3.22}$$

This will give us the equilibrium point defined as

$$l = p \tag{3.23}$$

$$p = \frac{\kappa l_0}{\alpha\,(1 + H_0) - \kappa} \tag{3.24}$$

As for $H = 0$ we have to define what will give a postive and negative value for P and we find this to be:

$$\alpha\,(1 + H_0) > \kappa \rightarrow p > 0 \tag{3.25}$$

$$\alpha\,(1 + H_0) < \kappa \rightarrow p < 0 \tag{3.26}$$

After linearizing the system and finding the Jacobian matrix given as:

$$\tilde{J} = \begin{bmatrix} -\alpha\,(1 + H_0) & \kappa \\ 1 & -1 \end{bmatrix} \tag{3.27}$$

We can use the characteristic equation the same way as we did for $H = 0$ to find the following eigenvalues and eigenvectors:

$$\omega_{1,2} = \frac{1}{2}\left[-\alpha\,(1 + H_0) \pm \sqrt{(\alpha\,(1 + H_0))^2 + 4\,(\kappa - (\alpha\,(1 + H_0)))}\right] \tag{3.28}$$

$$V_1 = \left(1 + \frac{1}{2}\left[-\alpha\,(1 + H_0) \pm \sqrt{(\alpha\,(1 + H_0))^2 + 4\,(\kappa - (\alpha\,(1 + H_0)))}\right]\right)V_2 \tag{3.29}$$

From the eigenvalues we can find that by looking at the how they are defined from the equation above and the classification for eigenvalues that:

$$\alpha\left(1+H\right) > \kappa \rightarrow \text{ stable node} \tag{3.30}$$

$$\alpha\left(1+H\right) < \kappa \rightarrow \text{ unstable saddle point} \tag{3.31}$$

This will also be apparant in the following section when we look at the phase portrait for the this system.

As for the case where H $=$ 0, we see this correlation for H $=$ constant when we change back to the original parameters in the equation:

$$\frac{\beta F}{\gamma + \beta F} \cdot \frac{\lambda}{\mu + h} \cdot \psi < 1 \tag{3.32}$$

The difference between when H $= H_0$ and when H $=$ 0 is that is easier to get the net reproductive value R_0 smaller since the expected lifetime of the louse now depends both on the mortality rate caused by natural causes, μ, and the mortality rate caused by harvest and treatment h. This means that by including the mortality rate caused by harvest and treatment, it is easier to control the net reproductive value to make it so the louse population will either die out or come down to an acceptable level where it doesn't lead to an epidemic with exponential growth of the louse population.

3.2.1 Phase portrait

First we will look at the phase portrait when $p > 0$ and this will be the following:
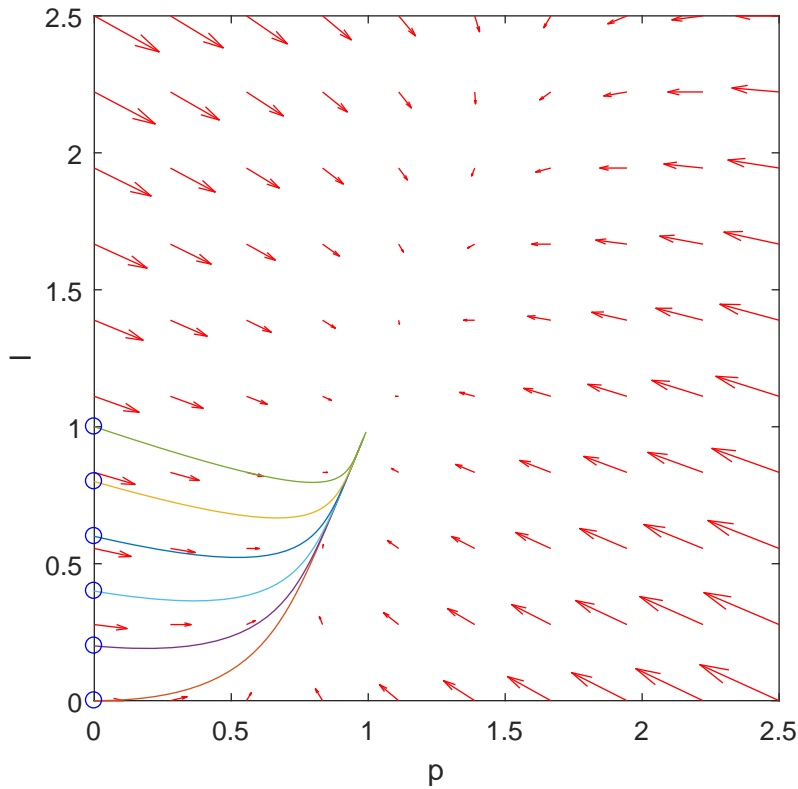
Figure 3.3: $H = $ constant and$\alpha \left(1 + H_0\right) > \kappa$

Figure 3.3 shows the situation with the following conditions: $\alpha = 1$, $\kappa = 2$, $H_0 = 1$ and $l_0 = 2$. We see the same tendency as for figure 3.1, but there is some big differences between these two figures. The equilibrium point is given as (p,l) = (1,1) and from the eigenvectors we find that system will decrease and go towards a stable solution for every initial value we start with. The difference between figure 3.1 and figure 3.3 is not that visible from just looking at them, but by including the constant harvest and treatment H we see that reaching the equilibrium point will be closer to origin of the coordinate system. This means for the same conditions as for no harvest and treatment will give a much lower louse populations with just including a constant flow of either treatment (by medicine or other options), including harvest or a combination of both. This is valid as long as $\alpha \left(1 + H_0\right) > \kappa$.
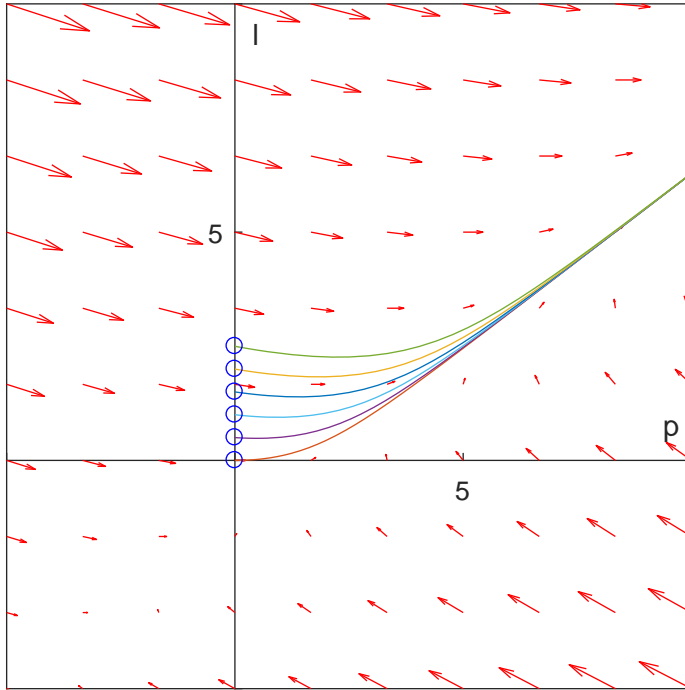
The next figure 3.4 will show will for the case when $p < 0$:

Figure 3.4: $H = $ constant and $\alpha\left(1 + H_0\right) < \kappa$

Figure 3.4 shows the situation with the following conditions: $\alpha = 0.5$, $\kappa = 4$, $H_0 = 1$ and $l_0 = 2$. For this situation we have that the equilibrium point at (p,1) $= \left(-\frac{8}{3}, -\frac{8}{3}\right)$ and we have that $\alpha\left(1 + H_0\right) < \kappa$. The results from this situation will be similar to the result in figure 3.2, but the difference here is that we have included a small parameter H_0 that can help us going from this situation with an unstabil saddle point where we have potentially an exponentiell growth to a stabil solution where the louse population either dies out are stays stable.

The difference between figure 3.1 and figure 3.3 is that we see that by including a small value H_0 the equlibrium point will have a smaller value and this means that the system will go faster towards becoming a smaller and stable population. The number of louse on fish P and free-living copepodites L will remain stable over time, and the only way to cause and epidemic is when there is for example outside pressure from infected wild fish that introduces new copepodites to the system.

3.3 Case 3: Scaled treatment and harvest effort

Here we have that the harvest and treatment constant is no longer a constant, but it also depends on how many louse we find on the farmed fish, and our

24

equations become nonlinear differential equations. The treatment and harvest will therefore increase when the infection levels increases, and this is written as $H = H_0 p$ (H_0 is regarded still as a positive constant).

For this case the nondimensionalized equations are the following

$$\frac{dp}{d\tau} = \kappa \left(l + l_0\right) - \alpha \left(1 + H_0 p\right) p \tag{3.33}$$

$$\frac{dl}{d\tau} = -l + p \tag{3.34}$$

The procedure to find the equilibrium points is still the same, $\frac{dp}{d\tau} = \frac{dl}{d\tau} = 0$, and by using this for our equations we get

$$l = p \tag{3.35}$$

$$\kappa p + \kappa l_0 - \alpha \left(1 + H_0 p\right) p = 0 \tag{3.36}$$

The last equations can be re-written to become the following

$$p^2 - \frac{1}{\alpha H_0} \left(\kappa - \alpha\right) p - \frac{\kappa l_0}{\alpha H_0} = 0 \tag{3.37}$$

This must be solved as a normal second degree polynomial and the solution is found to be

$$p = \frac{1}{2\alpha H_0} \left[\left(\kappa - \alpha\right) \pm \sqrt{\left(\kappa - \alpha\right)^2 + 4\kappa\alpha H_0 l_0}\right] \tag{3.38}$$

The solutions where p is positive, $p > 0$, is given as follows:

$$p = \frac{1}{2\alpha H_0} \left[\left(\kappa - \alpha\right) + \sqrt{\left(\kappa - \alpha\right)^2 + 4\alpha\kappa H_0 l_0}\right] \tag{3.39}$$

The corresponding value for p negative, $p < 0$, is given as follows:

$$p = \frac{1}{2\alpha H_0} \left[\left(\kappa - \alpha\right) - \sqrt{\left(\kappa - \alpha\right)^2 + 4\alpha\kappa H_0 l_0}\right] \tag{3.40}$$

We will use the method of linearzation to make the system easier to work with, but it is important to remember that by doing this we have to only look at our system around the equilibrium point(s). This is because this is just an estimation around the equilibrium point(s) and it will not give an accurate solution further away from the points.

For the two other cases, H = 0 and H = H_0, we had linear nonhomogenous equations so we made an estimation by using the method of linearization to find the homogenous solution to the system. The Jacobian matrix for the two other cases we have in this thesis will be equal to coefficient matrix $\tilde{A}$ because it was a system of linear differential equations, but for the nonlinear system it is not that easy.

We have to calculate the Jacobian matrix (before we insert the equilibrium value) as follows:

$$\tilde{J} = \begin{bmatrix} \frac{d\partial f}{dP}(P_0, L_0) & \frac{d\partial f}{dL}(P_0, L_0) \\ \frac{d\partial g}{dP}(P_0, L_0) & \frac{d\partial g}{dL}(P_0, L_0) \end{bmatrix} = \begin{bmatrix} -\alpha(1 + 2H_0 p) & \kappa \\ 1 & -1 \end{bmatrix} \tag{3.41}$$

After we insert the positive p value for the equilibrium points we have:

$$\tilde{J} = \begin{bmatrix} -\alpha(1 + 2H \frac{1}{2\alpha H} \left[(\kappa - \alpha) + \sqrt{(\kappa - \alpha)^2 + 4\alpha\kappa H_0 l_0} \right]) & \kappa \\ 1 & -1 \end{bmatrix} \tag{3.42}$$

By doing some small algebraic calculations, it can be written as

$$\tilde{J} = \begin{bmatrix} -\kappa - \sqrt{(\kappa - \alpha)^2 + 4\alpha\kappa H_0 l_0} & \kappa \\ 1 & -1 \end{bmatrix} \tag{3.43}$$

Now we can calculate the eigenvalues and eigenvectors for this system and we will use the characteristic equation again to find them. We will then calculate the eigenvalues as follows:

$$det \begin{bmatrix} -\kappa - \sqrt{(\kappa - \alpha)^2 + 4\alpha\kappa H_0 l_0} - \omega & \kappa \\ 1 & -1 - \omega \end{bmatrix} = 0 \tag{3.44}$$

Before calculating the eigenvalues, we will define a parameter S to make the calculations easier to read:

$$S = \sqrt{(\kappa - \alpha)^2 + 4\alpha\kappa H_0 l_0} \tag{3.45}$$

The eigenvalues for the positive p value will then be:

$$\omega_{1,2} = \frac{1}{2} \left[-(1 + \kappa + S) \pm \sqrt{(1 + \kappa + S)^2 - 4S} \right] \tag{3.46}$$

We know for a fact that

$$1 + \kappa + S > \sqrt{(1 + \kappa + S)^2 - 4S} \tag{3.47}$$

This then results in only negative eigenvalues and we will therefor have a stable node as a solution. It can also be a spiral here (where there is a complex solution for the eigenvalues), but that is only when $\kappa < 0$ and is outside our domain.

For the cases where we get negative p values, the calculations will be the same and we will find that the eigenvalues will be:

$$\omega 3, 4 = \frac{1}{2} \left[-(1 + \kappa - S) \pm \sqrt{(1 + \kappa - S)^2 + 4S} \right] \tag{3.48}$$

so here we will have that

$$1 + \kappa - S < \sqrt{(1 + \kappa - S)^2 - 4S} \tag{3.49}$$

This means that we get one positve eigenvalue and one negative eigenvalue, $\omega_3 < 0 < \omega_4$, and we will get unstable saddle point as the solution for negative p value. The next step is to find out how the eigenvectors will behave for this case. The steps are similar for H $= 0$ and H $= H_0$ where the we find that the eigenvectors for $p > 0$ are given by:

$$V_1 = \left(1 + \frac{1}{2}\left[-(1 + \kappa + S) \pm \sqrt{(1 + \kappa + S)^2 - 4S}\right]\right)V_2 \tag{3.50}$$

It will almost be the same for the situation when $p < 0$ and it will be:

$$V_1 = \left(1 + \frac{1}{2}\left[-(1 + \kappa - S) \pm \sqrt{(1 + \kappa - S)^2 + 4S}\right]\right)V_2 \tag{3.51}$$

3.3.1 Phase portraits

The next figure for this case will show the two equilibrium points we found in our calculations above.
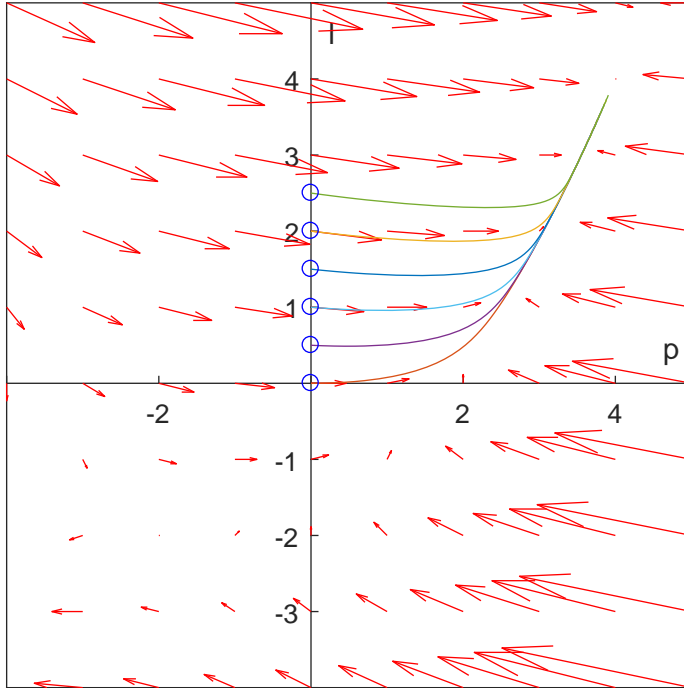

Figure 3.5: $H = Hp$

27

For the last figure 3.5 we show the solution for the nonlinear case and the parameters will be the following: $\alpha = 1$, $\kappa = 1$, $H_0 = 0.1$ and $l_0 = 2$. The figure shows that we have to equilibrium points where one is in the positive quadrant where p and l is larger than zero and one in the negative quadrant. The following plot shows that the equilibrium point with positive p will give us a stable solution and the other equilibrium point will be an unstable saddle point. This will be valid for all parameters (since all parameters are positive, $\alpha = \kappa = H_0 = l_0 => 0$).

The plot shows that if we start with a positive p/l value we will achieve a stable solution and the number of louse found on the fish/free-living copepodites will therefore remain constant and decrease down to equilibrium point if we get an increased starting value. The other equilibrium point is in the negative quadrant and it will not be of any interest for a real-life situation.

3.4 Equilibrium points as a function of H

Since in this thesis it is the louse mortality rate caused by harvest and treatment parameter, H_0, we have chosen to focus on, it would be interesting to see how the louse population will change as H_0 changes for each case. The plot shows that the p_1 function is for both the cases $H = 0$ and $H = H_0$ (by including H = 0) and p_2 and p_3 correspond to $H = H_0 p$ for positive and negative p value, and these cases are plotted against each other in figure 3.6. The equations are the following:

$$p_1 = \frac{\kappa l_0}{\alpha (1 + H_0) - \kappa} \tag{3.52}$$

$$p_2 = \frac{1}{2\alpha H} \left(\kappa - \alpha + \sqrt{(\kappa - \alpha)^2 + 4\alpha\kappa H_0 l_0} \right) \tag{3.53}$$

$$p_3 = \frac{1}{2\alpha H} \left(\kappa - \alpha - \sqrt{(\kappa - \alpha)^2 + 4\alpha\kappa H_0 l_0} \right) \tag{3.54}$$
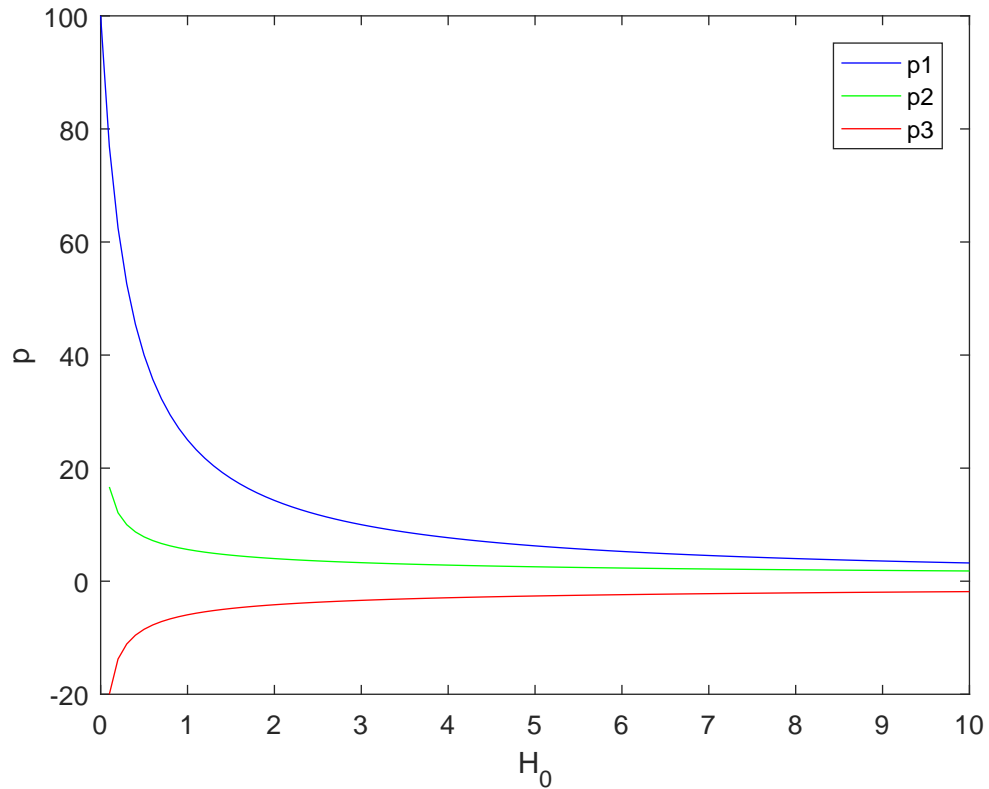
The plot will be the following

28

Figure 3.6: P ploted as a function of H

We know that for the equilibrium solution that p = 1, so the free-living copepodites will depend on the same parameters and give the same outcome as for p. We see that the all three graphs will go towards zero, but the difference between the graphs is how fast the graphs goes toward zero. The plot also shows that the H = H_0p function will give us smaller equilibrium points than the same for H = 0 and a constant H_0, but the only thing that we have to look out for that is that if we have zero harvest and treatment (H = 0) we cannot use this function as the function depends on $\frac{1}{H}$ and this will go towards infinity so it's not defined for H = 0. We can also see that from the equation for this case that the system will become the system that is for H = 0 for this situation.

Chapter 4

Discussion

Here there will be a discussion around the different cases found and explained in the analysis chapter and what these results mean for real life situations. There will also be section about other models and how they compare to the model in this paper. We will also look at if we can control the system and also if we can observe the system at all.

4.1 What does the analyze show

The model in this paper is the same as the one in [6], but with one difference. We have chosen to vary the harvest and treatment factor H to see what happens to the system, where the ultimate goal is to reach stability faster and therefore control the system faster. The first change to H was to make it zero and by doing so is equal to no harvest and treatment involved in the system, and there will be no directly affect from humans on the system.

By looking at the variables p and l over time when H = 0, we can see that the system will depend only on the natural survival rate of the louse. This system will not be of a any interest to us if we look at from a real life perspective because we use fish in sea-cages as food, and by removing the harvest factor we will not get any fish. We can also harvest fish as a means to control the louse population, and this will be studied in a later section. It's still good to use this in our model to compare the results to the other cases and therefore show that increasing the complexity of H will in return give us a system with increased stability.

For the next case we choose H to be constant over time. This will be similar to the situation they have analyzed in the original paper, but instead of focusing on critical threshold value of F parameter (the number of fish in the sea-cage(s)), we focus on making a stable solution by changing the H value. The results shows that the stability of this equation is higher because by including

the parameter H = H_0 versus H = 0 , the equilibrium point has a lower value and also the solution curves goes faster towards the equilibrium solution.This is also the exact same equation that is used in the original model, but the main focus here is not the critical values for the number of farm fish in the sea-cages. Instead by focusing on how much treatment we have to use to acheive a stable solution, we can find out how the direct human factor can alter the system to become stable (there is other factors we human can change, like how many fish there is in the sea-cages (F)). However, the focus is not how much treatment and harvest we have to use, but instead on how little treatment and harvest we have to do to acheive a stable system. This system does show that without control over the population with treatment and harvest, it is easier for the louse to cause an epidemic.

In the last case we introduce a linear dependence between the harvest and treatment parameter and the variable p, that is the number of louse on the fish. This dependence makes our system become a nonlinear system and it was shown that for p and l larger then zero we will a stable solution for all parameters. This is an important result because it shows that as long as we can somehow count the number of fish louse in the sea-cages, we can administrate a minimum amount of treatment and harvest as much as we want.

The only problem is, how can we count the number of louse that is on the fish? Maybe it will be possible if we can analyze the spread pattern for the louse and use this to calculate how many louse will infect a given sea-cage. It was suggested by the Institute of Marine Research that if we can make a precise spread pattern model that includes all the factors that affect the fish populations, it can be possible to come with more precise treatment option [2]. According to the Institute of Marine Research [2] it is the louses behaviour that makes it difficult to know precisely how the spread pattern will be, because sometimes the spread can be over 100 km from the start point and in other situations they can only spread $\sim 20 - 30$km. It can therefore be viable to find out more about the louse spread and therefor also find better spots for the sea-cages.

4.2   Linear Control Theory

The next step is to see if one can control this system and for the purpose of this thesis we will still look at the nondimensionalized parameters and see how changing the H will affect if the system will be more or less controllable and also if it is possible to observe the system at all. To find out if our system is either observable or controllable, or both, we have to define the state equation and measurement equation and then we can define what will make the system observable or/and controllable.

The state eqaution can be written as [11]

$$\dot{x} = Ax(t) + Bu(t) \tag{4.1}$$

And the measurement equation is listed here

$$y(t) = Cx(t) + Du(t) \tag{4.2}$$

The variables for both the state equation and the measurement equation are listed here[11]:

Variable	Dimension	Name
x(t)	n× 1	State vector
$\dot{x}$	n×1	Time derivative of the vector x(t)
A	n× n	System matrix
B	n × r	Input matrix
u(t)	r × 1	Input vector
y(t)	p ×1	Output vector
C	p × n	Output matrix
D	p × r	Matrix, couples input and output

This system of equations can then tell us what kind of output we will get from a set of inputs and how the relation between the two can be measured, and this can be used to find if we can control our system and also if it is observable.

The definition for a controllable system is a system where we can do whatever we want to the system under a control input and a system is observable if we can see what happens inside the system under observation at every time [12].

The state equation for our system is as before

$$\dot{x} = \begin{bmatrix} \dot{p} \\ \dot{l} \end{bmatrix} = \begin{bmatrix} -\alpha(1+H) & \kappa \\ 1 & -1 \end{bmatrix} \begin{bmatrix} p \\ l \end{bmatrix} + \begin{bmatrix} \kappa l_0 \\ 0 \end{bmatrix} \tag{4.3}$$

The definition for controllability of the system is if the rank(B AB) = n. where (B AB) is called the controllability matrix [11]. The definition for controllability is that for a given state equation where we start with a given initial condtion x(0) = x_0, there will exist a control function u(t) such that the equation will go towards a given point x(T) = x_T in a given number of steps n [12].

In regards to our system will the steps be given as the dimensions of the matrix $\tilde{A}$ and is in our case 2. We will therefore have a controllable system if the rank of controllability matrix is equal to two. We know that

$$B = \begin{bmatrix} \kappa l_0 \\ 0 \end{bmatrix} \tag{4.4}$$

$$A = \begin{bmatrix} -\alpha\left(1+H\right) & \kappa \\ 1 & -1 \end{bmatrix} \tag{4.5}$$

This means that

$$\begin{bmatrix} B & AB \end{bmatrix} = \begin{bmatrix} \kappa l_0 & -\alpha\kappa l_0\left(1+H\right) \\ 0 & \kappa l_0 \end{bmatrix} \tag{4.6}$$

This means that rank(B AB) = 2 (since they columns are independent of each other) and the system will then be controllable for all variables.

The next step is to see if the system is observable and the definition is the following [12]:

$$rank \begin{bmatrix} C \\ CA \end{bmatrix} = n \tag{4.7}$$

This means that if we have an observable system, we have that we start with a n-dimension initial vector $x(0)$ as an input to give us the output function. This vector has n unknown components and we will have to do n measurements to determine x(0). By doing this we will have to do two measurements to find the initial input vector for our system and it follows from equations that we have the definition of observability [12].

To find out if our system is observable, we need to define the measurement equation. First, we will say that we can measure the output of both the number of lice on farmed fish in an area (P) and the number of free-living copepodites from lice on farmed fish (L). We can define this is

$$y\left(t\right) = \begin{bmatrix} 1 & 1 \end{bmatrix} \begin{bmatrix} p \\ l \end{bmatrix} \tag{4.8}$$

where

$$C = \begin{bmatrix} 1 & 1 \end{bmatrix} \tag{4.9}$$

and this gives us

$$rank = \begin{bmatrix} C \\ CA \end{bmatrix} = \begin{bmatrix} 1 & 1 \\ -\alpha\left(1+H\right)+1 & \kappa-1 \end{bmatrix} \tag{4.10}$$

The rank of this is equal to two as long as

$$-\alpha\left(1+H\right)+1 \neq \kappa-1 \tag{4.11}$$

The next case is what if we can only measure the number of lice on farmed fish in an area (P) and not the number of free-living copepodites from lice on farmed fish (L). In other words

$$y\left(t\right) = \begin{bmatrix} 1 & 0 \end{bmatrix} \begin{bmatrix} p \\ l \end{bmatrix} \tag{4.12}$$

This gives is that

$$rank = \begin{bmatrix} C \\ CA \end{bmatrix} = \begin{bmatrix} 1 & 0 \\ -\alpha\left(1+H\right) & \kappa \end{bmatrix} \tag{4.13}$$

The rank of this will equal 2 as long as $\kappa \neq 0$.

The linear control theory shows us at it is not necessary to know how many free-living copepodites (L), and this is good because this parameter is much harder to track then the number of parasites on farm fish (P). The fact that the system still is controllable and observable makes the original model viable under these circumstances and this means that the Anderson-May model used in [6] can be a good model to track the growth and decline in the louse population in farmed fish in the free-living copepodites.

4.3   Main result from the original model

The main result from [6] is as stated in the method chapter:

$$R_0 = \frac{\lambda}{\mu + h} \frac{\beta F}{\beta F + \gamma} \psi \tag{4.14}$$

And they use this to find the critical threshold for number of fish in farm [6], in other words when $R_0 = 1$. written mathematically:

$$F_x = \frac{\gamma (\mu + h)}{\beta (\lambda \psi - \mu - h)} \tag{4.15}$$

Saying that if the number of fish in the farm exceeds this value, the reproductive value will be larger than one and we will most likely have a louse epidemic. The reproductive value was found also in our anallyze and and we found that to get stable solutions for the two first cases (H = 0 and H = H_0) we needed that:

$$\alpha > \kappa \tag{4.16}$$

$$\alpha (1 + H) > \kappa \tag{4.17}$$

where the first equation equals to H = 0 and the second equation equals to H = H_0 and we found that by changing the parameters back to the same used in the orignal model we found respectively

$$\frac{\beta F}{\gamma + \beta F} \cdot \frac{\lambda}{\mu} \cdot \psi < 1 \tag{4.18}$$

$$\frac{\beta F}{\gamma + \beta F} \cdot \frac{\lambda}{\mu + h} \cdot \psi < 1 \tag{4.19}$$

The only difference between these two equations is that $\mu \rightarrow \mu + h$ and this means that including an extra parameter h to the reproductive value found when there is no harvest and treatment affecting the system. This means that by increasing the mortality rate of the louse, μ, with a parameter h we will also

increase the possibility to avoid an epidemic by lowering the value of R_0. Then we will have the number of fish in sea-cages(F) will be lower than the critical threshold value (F_0) and we will avoid an epidemic according to the original model.

We said in the introduction that the most common treatment method is by using anti-parasitic medicine to remove the louse from the fish, but what will happen to the louse when it is exposed to the medicine over time? The louse can develop a resistance to the medicines used and these traits can be inherited from one louse to the next generations, and that is when it becomes difficult to use the same medicine to treat the louse problem [13].

Resistance to medicine is real problem when it comes to treating the louse problem because one has to switch the medicine out with a new type or administrate higher doses of medicine to treat the louse problem [13]. This can in turns cause double and tripple resistance to several types of medicine and makes it even harder to remove the louse. This can possibly affect our model in the way that louse mortality rate caused by harvest and treatment will be lowered and that can in turn make the reproductive value to increase. The next step will then be to look at the other parameters in equation and see if we can lower the value by changing some of the other parameters.

The other parameters in this equation is estimated to be constant (in the original model they used figures from real-life sea-cages to get the values used in the model [6]), but it is possible to change these values so we can lower the reproductive value in other ways. By looking at the reproductive value without harvest and treatment, we see that there are potentially 6 other parameters that can alter the equation: λ, ψ, γ, F, μ and β. The following subsections will explain how the equation can change by varying some of the parameters.

4.3.1 β: The transmission rate

To reduce the value of the reproductive value R_0 by changing the β, we have to decrease the parameter value. One way is to try find preventive measures to slow the transmission rate. One non-medical method is to build a louse skirt [14] that will catch the sea louse before it can infect the fish in the sea-cages [15]. The louse will then in theory never reach the fish and the transmission rate will decrease.

A model made by Anderson and May [16] looked into how the transmission of a pathogen occurs and they came up with a main conclusion: if one can calculate the host density threshold, N_t, that is the threshold where below the pathogen cannot invade a population of susceptible individuals, one can control the pathogen such that no epidemic can happen. This only works if there exists such a threshold. A threshold for the introduction of a disease is the minimum population size of susceptible hosts necessary for the disease to increase. The

problem by neglecting the transmission rate in for example human diseases is not that visible, because it's most likely not fatale enough to ruin the population, but for animals it can be fatale and therefore it is important to try and calculate the transmission rate and also how the transmission occurs.

4.3.2 ψ: Probability that an attached copepodite survives to adulthood

To lower the probability that an attached copepodite survives to adulthood, one must find ways to stop the louse from reaching the next stage in the life cycle. The mathematical model used in the paper by C.W Revie et. al.[17] has two different conditions to predict likely effects of different treatment strategies: development rates of the lice growth and the mortality of the lice (both from the different life stages of the lice and also external infection pressure). The authors of the model found out that the most sensitive time period to administrate treatment (with regards to survivability) is when the louse evolves from one mobile stage to the next mobile stage, by using parameters such as background infection rate to calculate the survivability rate. The authors also found out that administrating many treatments early in the life cycle is shown to be the best option to control the system [17].

This means that administrating early in the life cycle can in theory lower the parameter ψ, but the system doesn't include all the factors that affect the louse population because of the complexity of such a system [17]. It can still tell us that if we still want to use medicin to treat the louse problem, we should administrate many treatments in the early stages of the louse life cycle. This will therefor depend on when we find out that the fish is infected to best calculate the best time to administrate the medicine.

By doing as explained in the model above, we will also in theory lower the probability that the louse survives to produce new llices. This means that if we can make a connection between ψ and λ, we can reduce the number of louse produced by each female louse. This will be to take the model in this paper further, but it would be interesting to see what will happen to the reproductive value R_0 if we can include several life stages in the probability value ψ.

4.3.3 F: Number of farm fish in the sea-cages

In [6] they assume that this value is constant (with the criteria that it has to be smaller then F_0), but how can we decide how many fish we should have in the sea-cage? As the model found for the transmission rate we have reasons to want to control the host density. One model tries to see what will happen if we should remove the fish with louse infection and how this will affect the population. The model [18] describes several processes to reduce the louse population and one of them is to remove the fish based on the infection status. Targeting the host based on the infection status (for example taking large fish, that is older and

can be further down the infection rate or have more lice than a smaller fish) can be very effectful because it can reduce the parasites reproduction rate.

The problem is that it can be impossible to target the uninfected fish in a real-life situation and the same was found for the other host population processes [18]. It will be too difficult to know if the results we will get comes from the removal of fish or from some other aspect that happens under these processes. Another aspect to fishing out the host is to remove the community around the host fish, for example removal of predators that attack the host fish. The result from this article [18] is that fish-driven change in fish community structure should reduce the parasite abundance and if there is an increase in the parasite abundance, one should target other hosts for the parasites that are released from predation to reduce the louse population

This shows that it can be possible to lower the value of F to lower the reproductive value, but this will not be that interesting to do for someone owning a system of sea-cages. We wish to have the highest number of fish in the sea-cages possible, but also the control the number of sea louse to a level such that there is no risk for an epidemic. The possibility is that we can target the fish based on the infection status and therefor reduce the possibility for furthur infection to uninfected hosts.

By including some of the changes to the parameters we can make the reporductive value in theory, but this system is also filled with unkowns like how louses behaviour. We don't know how the louse behaves and can then be difficult to see if the changes we make to the parameters will ultimately give a realistic model over a real-life situation. Using models like this is still important because they can give us a picture over how a system such as this one will more, and by doing experiments we can maybe see a result corresponding to the model.

4.4 The way forward

The model in this thesis is a simplified model of a possible real-life situation and one step is to include different variations of the parameters as listed above. One of the problems is that we don't know how the louse spreads and it can therefore be difficult to stop the increase of the louse population if we don't have detailed information on how the louse behaves. It's still important to make models like this one, because they can be used to get a theoretic values for how many fish we can have in sea-cage and still not have an louse epidemic. By incorporating the changes found in the previous section, one can in theory reach a lower reproductive value.

Bibliography

[1] Havforskningsinstituttet. Generell biologi, 2017. URL `https://www.imr.no/temasider/parasitter/lus/lakselus/90682/nb-no`.

[2] Havforskningsinstituttet. Hvordan spres lakselusa?, 2017. URL `https://www.imr.no/temasider/parasitter/lus/lakselus/90683/nb-no`.

[3] Havforskningsinstituttet. Effekter av lakselus på vill laksefisk, 2018. URL `https://www.imr.no/temasider/parasitter/lus/lakselus/90684/nb-no`.

[4] Havforskningsinstituttet. Lakselus, 2016. URL `https://www.imr.no/temasider/parasitter/lus/lakselus/nb-no`.

[5] Havforkningsinstituttet. Vaksineutvikling, 2018. URL `https://www.imr.no/temasider/parasitter/lus/lakselus/vaksineutvikling/nb-no`.

[6] L. Neil Frazer, Martin Krkošek, and Alexandra Morton. Critical thresholds in sea lice epidemics: evidence, sensitivity and subcritical estimation. *Proceedings of the Royal Society of London B: Biological Sciences*, 279(1735): 1950–1958, 2012. ISSN 0962-8452. doi: 10.1098/rspb.2011.2210. URL `http://rspb.royalsocietypublishing.org/content/279/1735/1950`.

[7] Ira M. Cohen, David R. Dowling, and Pijush K. Kundu. *Fluid Mechanics*. Academic Press, 5 edition, 2012.

[8] Paul Blanchard, Robert L. Devaney, Glen R. Hall, and Brian Persaud. *Differential Equations*. Brooks/Cole, Cengage Learning, 2011.

[9] D. W. Jordan and P. Smith. *Nonlinear ordinary differential equations*. Oxford University Press, 4 edition, 2007.

[10] MathWorks. Ode45, 2018. URL `https://se.mathworks.com/help/matlab/ref/ode45.html`.

[11] S. Simrock. Control theory. *Deutsches Elektronen-Synchrotron (DESY),Conference*, pages 73–130, 2008. URL `https://cds.cern.ch/record/1100534/files/p73.pdf`.

[12] Rutgers EDE. Controllability and observability, n.d. URL `http://www.ece.rutgers.edu/~gajic/psfiles/chap5traCO.pdf`.

[13] Havforskningsinstituttet. Resistens hos lakselus, 2018. URL `https://www.imr.no/temasider/parasitter/lus/lakselus/resistens_hos_lakselus/nb-no`.

[14] Mattilsynet. Fakta om lakselus og lakselusbekjempelse, 2016. URL `https://www.mattilsynet.no/fisk_og_akvakultur/fiskehelse/fiske_og_skjellsykdommer/lakselus/fakta_om_lakselus_og_lakselusbekjempelse.23766`.

[15] Global Salmon Initiative. Non-medicinal approaches to sea lice management, 2018. URL `https://globalsalmoninitiative.org/en/what-is-the-gsi-working-on/biosecurity/non-medicinal-approaches-to-sea-lice-management/`.

[16] R. M. Anderson and R. M. May. Population biology of infectious diseases: Part i. *Nature: International journal of science*, 280, 1979. URL `http://www.nature.com.pva.uib.no/articles/280361a0.pdf`.

[17] G. Gettinby, L. Kelly, C. W. Revie, C. Robbins, and J. W. Treasurer. A mathematical model of the growth of sea lice, lepeophtheirus salminis, populations on farmed atlantic salmon, salmo salar l., in scotland andits use in the assessment of treatment strategies. *Journal of Fish Diseases*, pages 603–613, 2005.

[18] K. D. Lafferty, F. Micheli, and C. L. Wood. Fishing out marine parasites? impacts of fishing on rates of parasitism in the ocean. *Ecology Letters*, 2010.