Aalto University
School of Science

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Analysis of a predator-prey system with a shared disease

Master´s thesis submitted in partial fulfillment of the requirements for the degree of Master of Science in Technology in the Degree Programme in Engineering Physics and Mathematics.

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Supervisor: Professor Harri Ehtamo

Instructor: Research fellow Eva Kisdi

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Aalto University School of Science	ABSTRACT OF THE MASTER'S THESIS

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Title: Analysis of a predator-prey system with a shared disease

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Abstract:

This thesis studies a predator-prey model with a shared disease. In addition to the prey the predator has an alternative food source which it consumes. Both species are vulnerable to a pathogen that spreads not only from prey to prey but also from predator to predator. Predators can get infected when consuming infected preys and preys can get infected from environmental transmission.

Properties of the model and its behaviour are studied. This includes the basic reproduction, the next-generation matrix and the stability of equilibria. Factors affecting the properties of the equilibria are identified. Consumption rate, the rate at which the predator consumes prey with, is studied and critical consumption rate, where prey or predator population ceases to exist, is identified.

Evolution of the consumpion rate is studied within the adaptive dynamics framework. Without the presence of the pathogen the consumption rate increases indefinitely, but introducing the pathogen to the system can change the course of evolution and make a decrease in the consumption rate possible.

The evolution of the consumption rate is explored further with help of trade-offs. Taking into account that increase in consumption rate might be costly to the predator provides alternative outcomes. Two trade-offs that are studied are trade-off between consumption rate and the consumption of the alternative food source and trade-off between consumption and death rate. These trade-offs can exist concurrently or independently.

Having both of the trade-offs in place we can identify situations where we have a branching point. In such a situation the consumption rate evolves first to this brancing point, where two different branches arise and disruptive evolution takes place. The result is two different types of predator, one using only the alternative food source and one using only the prey as its source of food.

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Tiivistelmä:

Tässä diplomityössä tutkitaan saalis-saalistajamallia, jossa molempiin lajeihin tarttuu yhteinen tartuntatauti. Saalislajin lisäksi saalistajalla on käytössään vaihtoehtoinen ravinnonlähde. Molemmat lajit ovat haavoittuvaisia tartuntataudille, joka ei leviä vain lajinsisäisesti, vaan myös niiden välillä. Saalistaja voi saada taudin syödessään sairastunutta saalista ja saaliseläimet voivat saada tartunnan ympäristön saastumisen seurauksena.

Mallin ominaisuuksia ja käytöstä tutkitaan. Tähän sisältyvät esimerkiksi peruslisääntymisluvun, seuraavan sukupolven matriisin määrittäminen ja tasapainotilan stabiilisuuden määrittäminen. Tasapainotilan ominaisuuksiin vaikuttavat tekijät tunnistetaan. Kulutustahtia, nopeutta jolla saalistaja saalistaa saalista, tutkitaan ja kriittinen kulutustahti, jossa saalis- tai saalistajapopulaatio kuolee, tunnistetaan.

Kulutustahdin evoluutiota tutkitaan adaptiivisen dynamiikan keinoin. Ilman tartuntatautia evoluutio johtaa kulutustahdin loputtomaan kasvuun. Jos systeemiin lisätään tartuntatauti, voidaan eräissä tapauksissa havaita, että evoluution seurauksena on kulutustahdin lasku.

Evoluutiota tutkitaan lisää ominaisuuksien vaihtoehtoisuuden avulla. Kulutustahdin kasvattaminen voi olla saalistajalle kallista muiden ominaisuuksien kannalta. Vaihtoehtoisuutta tutkitaan kulutustahdin ja vaihtoehtoisen ravinnonlähteen käytön välillä ja kulutustahdin ja kuolleisuuden välillä. Nämä voivat esiintyä niin yhdessä kuin erikseenkin.

Jos molemmat vaihtoehtoisuudet esiintyvät, voidaan havaita tilanne, jossa on haarautumispiste. Tällaisessa tapauksessa kulutustahdin evoluutio johtaa ensin haarautumispisteeseen. Tämän pisteen ympäristössä ilmestyy kaksi erilaista kulutustahtia, jotka kokevat eriyttävää evoluutiota. Lopulta saalistaja lajiutuu yhteen lajiin, joka käyttää vain vaihtoehtoista ravinnonlähdettä ja yhteen, joka saalistaa vain saaliseläintä.

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1 Preface

Completing this thesis has been the climax of my studies in Aalto University. I hope it represents my abilities and strengths well in and outside of my expertise.

I would like to thank my instructor Eva Kisdi for introducing me to this topic and for her guidance in completing my thesis. My supervisor Harri Ehtamo I want to thank not only for his insights into this thesis but also for guiding me with my studies in general.

I would also like to thank my family, especially my parents, for supplying me with the skills and motivation I need for my studies and life itself. Especially I want to thank my spouse Lotta for everything.

2 General predator-prey model

For a two-species system for which the growth rates of the species are functions of the population sizes can be described with equations

$$\dot{x} = xf(x, y) \tag{1}$$
$$\dot{y} = yg(x, y). \tag{2}$$

These equations form what is called a Kolmogorov model for the predator-prey system.[4] Kolmogorov postulated some general conditions for realistic predator-prey interaction. Assuming that $f(x, y)$ and $g(x, y)$ are continuously differentiable we can impose the following conditions.[31] Let x be the population size for the prey, and y for the predator.

First,

$$f_y < 0, \tag{3}$$

where subscript means derivative with respect to that variable. This condition means that the rate of increase for the prey decreases when more predators are present. Second,

$$g_y < 0, \tag{4}$$

meaning that the rate for the predator decreases when more predators are present. These are the two conditions that can be accepted in a very general setting.

The third condition is

$$g(\overline{x}, 0) > 0, \tag{5}$$

which means that the growth rate of the predator is positive in such a position where there are no predators and prey is at an equilibrium. What is implied is that the prey alone has a positive equilibrium.

These three conditions don't guarantee the existence of a single equilibrium. Conditions

$$f_x < 0 \tag{6}$$
$$g_x > 0 \tag{7}$$

would guarantee this. With (3)-(5) we know there exists a positive equilibrium with both species. When we add conditions (6) and (7) we can deduct that the equilibrium is unique.[31]

The problem with these conditions is that they may not always hold. Kolmogorov already noticed that in small enough populations the rate of increase may decrease or become negative if the population density is too low. This is one example of the phenomena known as Allee effect.[7]

Another set of conditions could also be used. They are

$$xf_x + yg_y < 0 \tag{8}$$
$$xg_x + yg_y > 0. \tag{9}$$

These two conditions combined with the first three guarantee the existence of a single positive equilibrium in this model.

The model studied in this thesis has basis on a predator-prey model with functions

$$f(x, y) = v_1(x) - \mu_1 - \phi y \tag{10}$$
$$g(x, y) = v_2(y) - \mu_2 + e\phi x, \tag{11}$$

where v_1 and v_2 are functions describing an influx of new individuals, the so called birth rate functions. ϕ defines the amount of predation and e describes how much of the prey biomass (or some other suitable unit) is converted into predator biomass. Because ϕ is a positive constant condition 3 holds. 4 holds if $v_2(y)$ is assumed to be a decreasing function of y.

3 Epidemiological models

The basic epidemiological SIR model was presented by W. O. Kermack and A. G. McKendrick in 1927.[19]. The model is

$$\dot{S} = -\beta SI \tag{12a}$$
$$\dot{I} = \beta SI - \gamma I \tag{12b}$$
$$\dot{R} = \gamma I. \tag{12c}$$

In this model S represents susceptibles that can be infected with the disease. I are the infected and R are the recovered individuals that have suffered the infection and cannot be infected anymore. β is a factor describing the infectivity rate of the disease in the population. γ is the recovery rate from the disease.

This model is very simplified and it doesn't include deaths or births. What is used in this thesis is a variant of this model. The model used is

$$\dot{S} = -\beta \frac{SI}{N} + \gamma I \tag{13a}$$
$$\dot{I} = \beta \frac{SI}{N} - \gamma I \tag{13b}$$

that is, the recovered individuals return to the susceptible class. This means there is no immunity from the disease. There is also another important difference. The basic SIR-model uses so called law of the mass action where every individual mixes with every other individual with a constant rate. This law of the mass action was first proposed by chemists Guldberg and Waage on reversible chemical reactions in 1864.[36][35][17]

In our model, however, individuals mix with a constant rate independent from the size of the whole population.

The simple epidemiological model presented in (13) is supplemented with cross-species epidemiological interaction and the previously mentioned predator-prey dynamics.

4 The model

This model was first presented by M. G. Roberts and J. A. P. Heesterbeek in 2013.[27] The system is a predator-prey system where both species share a common pathogen. The main result of their study was showing that there exists a range of model parameters where the two species coexists only when the pathogen is present.

The system is described as a system of differential equations. We let N_1 be the density, number of animals or biomass of class prey. Correspondingly N_2 is the amount of predators. Now some of these individuals might be infected with the disease and we denote these with I_1 and I_2. The rest of the individuals are healthy and are denoted by $S_i = N_i - I_i$ for $i = 1, 2$. These are called susceptibles.

$$\dot{N}_1 = v_1(N_1)N_1 - \mu_1 N_1 - \alpha_1 I_1 - \phi(S_1 + qI_1)(S_2 + pI_2) \tag{14a}$$
$$\dot{N}_2 = v_2(N_2)N_2 - \mu_2 N_2 - \alpha_2 I_2 + e\phi(S_1 + qI_1)(S_2 + pI_2) \tag{14b}$$
$$\dot{I}_1 = \beta_1 \frac{S_1 I_1}{N_1} - \mu_1 I_1 - \alpha_1 I_1 - \phi q I_1(S_2 + pI_2) + \kappa S_1(I_1 + rI_2) \tag{14c}$$
$$\dot{I}_2 = \beta_2 \frac{S_2 I_2}{N_2} - \mu_2 I_2 - \alpha_2 I_2 + cq\phi I_1 S_2 \tag{14d}$$

Here $v_i(N_i)$ is a general birth function. Roberts and Heesterbeek used a function of type $V_i(N_i) = \rho_i e^{-k_i N_i}$ in their calculations. Another choice could be to take $V_i(N_i) = \frac{B_i}{N_i}$ leading to a constant influx of individuals. With only one species and a constant death rate μ, this assumption leads to a stable population with $N = \frac{B}{\mu}$. What we must assume about the birth function is that it has a negative derivative everywhere. This might not make sense in every situation. For example if there are too few individuals, they might not be able to meet and mate. The assumption is however important for the results that follow. For the prey the birth rate function can be thought of as a representation of its food source. Similarly for the predator, the birth rate function describes the alternative food sources for it.

As suggested earlier μ_i is the death rate of type i individuals.

The prey is eaten by the predator with rate $\phi(S_1 + qI_1)(S_2 + pI_2)$. ϕ denotes the rate at which the predator hunts the prey. q denotes how the infection in prey affects the catch rate. Sick prey could be easier to catch, in which case

$q > 1$ or not as attractive, in which case $q < 1$. Similarly p tells how infection in predator affects hunting. In this case $p \leq 1$ because it is unlikely that infection would enhance the predator's abilities to hunt. e tells us how much of the prey biomass is converted to predator biomass. Only about 10 % (the ten percent rule) of the amount of energy is actually transferred through a food chain.[32] Thus usually $e \approx 0.1$ when N_i is measured as biomass. κ describes the strength of environmental transmission. This means that infected predators and prey leave infected material in the environment, from which susceptible prey can get infected. r describes how much the predators spread the infection through this mechanism compared to the prey. c describes how much the infected prey causes predators to get infected through consuming.

The disease dynamics in this model are assumed to be of type SIS. There is no recovered state. The infected individuals have an increased death rate of α_i and susceptibles are infected with a rate of β_i. The infections are not modelled here with mass action but with standard incidence, where the number of contacts per unit of time per individual are independent from the size of the population.[11] This is often thought to be a more suitable approach when modeling animals in herds.[10] The model actually has no recoveries at all. The usual recovery rate γ in equations 13 is zero in a sense. Infected individuals remain infected until they die.

4.1 Next-generation matrix

We can find the next-generation matrix (NGM) for this system. It can be used to derive the basic reproduction number and concerns the situation where the disease is rare. NGM is defined as

$$K = -T\Sigma^{-1} \tag{15}$$

where T is a matrix containing epidemiological transimission terms and Σ is a matrix containing epidemiological transition terms.[34] We have only two states-at-infection, infected prey and infected predator, and it is impossible to get from one to the other. Therefore Σ is a diagonal matrix containing the rates with which an individual is removed from an infective state. We have

$$\Sigma = -\begin{pmatrix} \mu_1 + \alpha_1 + \phi q N_2 & 0 \\ 0 & \mu_2 + \alpha_2 \end{pmatrix} \tag{16}$$

For transmission we have a more complicated matrix

$$T = \begin{pmatrix} \beta_1 + \kappa N_1 & r\kappa N_1 \\ c\phi q N_2 & \beta_2 \end{pmatrix} \tag{17}$$

From (15) we get

$$K = -T\Sigma^{-1} = \begin{pmatrix} \frac{\kappa N_1 + \beta_1}{q\phi N_2 + \alpha_1 + \mu_1} & \frac{r\kappa N_1}{\alpha_2 + \mu_2} \\ \frac{cq\phi N_2}{q\phi N_2 + \alpha_1 + \mu_1} & \frac{\beta_2}{\alpha_2 + \mu_2} \end{pmatrix} \tag{18}$$

From this matrix we can study the basic reproduction numbers for the species. If we assume that the prey and the predator live in isolation from each other, the predator doesn't feed on the prey and $\phi = 0$. With this assumption we get basic reproduction numbers for the species as the eigenvalues of the next-generation matrix.[34] For the prey we have

$$R_0^1 = \frac{\beta_1 + \kappa N_1}{\mu_1 + \alpha_1} \tag{19}$$

and for the predator

$$R_0^2 = \frac{\beta_2}{\mu_2 + \alpha_2}. \tag{20}$$

It is worth noticing that $\phi = 0$ means ecological isolations. However there is still epidemiological connection through the environment when $\kappa \neq 0$. With total isolation κ would be zero too, and basic reproduction number of the prey would simplify. As it should, the next-generation matrix shows how κ governs the flow from predator to prey and ϕ governs the flow from prey to predator.

In an epidemiological setting, the increased death rate can vary a lot. In some cases there are no or almost no increased deaths from the disease. In such case we can use approximation $\mu \gg \alpha$. With this approximation we can say

$$R_0^1 \approx \frac{\beta_1 + \kappa N_1}{\mu_1} \tag{21}$$

and for the predator

$$R_0^2 \approx \frac{\beta_2}{\mu_2}. \tag{22}$$

For some other diseases increased death rate can be remarkable, even much greater than the normal death rate. Then we, of course, can not use the approximation above.

4.2 Stability of a steady state

Roberts and Heesterbeek study these steady states by reordering the model equations. By exchanging equations 14b and 14c they get a Jacobian of form

$$J = \begin{pmatrix} H & D \\ 0 & C \end{pmatrix} \tag{23}$$

when linearized for a stable state with only one species. Here H is a matrix that controls the epidemiological stability, that is the stability of the system with regards to the pathogen. C is a matrix that deals with ecological stability. That means that stability exists when the other species is introduced to the steady state. D is also a matrix, but it does not have a special meaning.

5 Simplified versions

With only two of the three elements present, the system reduces to a much simpler system. Much of the work presented in this section has already been covered by Roberts and Heesterbeek.

5.1 Prey and predator

Without the pathogen the system reduces to a normal predator-prey system. The differential equations governing this are

$$\dot{N}_1 = v_1(N_1)N_1 - \mu_1 N_1 - \phi N_1 N_2 \tag{24a}$$

$$\dot{N}_2 = v_2(N_2)N_2 - \mu_2 N_2 + e\phi N_1 N_2 \tag{24b}$$

This system has four kind of different steady states. One trivial state with no individuals, one for each species only and one with both species. The stability of the steady states can be examined with the help of the Jacobian of the system. In this case we have

$$J = \begin{pmatrix} v_1(\overline{N}_1) + v_1'(\overline{N}_1)\overline{N}_1 - \mu_1 - \phi\overline{N}_2 & -\phi\overline{N}_1 \\ e\phi\overline{N}_2 & v_2(\overline{N}_2) + v_2'(\overline{N}_2)\overline{N}_2 - \mu_2 + e\phi\overline{N}_1 \end{pmatrix} \tag{25}$$

For the trivial steady state we have $\overline{N}_1 = \overline{N}_2 = 0$ and

$$J = \begin{pmatrix} v_1(0) - \mu_1 & 0 \\ 0 & v_2(0) - \mu_2 \end{pmatrix} \tag{26}$$

A steady state is stable only if the real parts of the eigenvalues of the Jacobian matrix are all negative. Thus this steady state is stable when $v_1(0) \leq \mu_1$ and $v_2(0) \leq \mu_2$. This means that the death rate with infinitesimal population must be greater than birth rate.

For the steady state with prey only we have $\overline{N}_2 = 0$ and $v_1(\overline{N}_1) = \mu_1$. In this case the Jacobian is

$$J = \begin{pmatrix} v_1'(\overline{N}_1)\overline{N}_1 & -\phi\overline{N}_1 \\ 0 & e\phi\overline{N}_1 + v_2(0) - \mu_2 \end{pmatrix} \tag{27}$$

From this matrix we get eigenvalues $\overline{N}_1 v_1(\overline{N}_1)'$ and $e\phi N_1 - \mu_2 + v_2(0)$. The first is negative if and only if the derivate of the birth function is negative. This is exactly the assumption that was made earlier. The second condition is $e\phi N_1 + v_2(0) \leq \mu_2$. This means that the death rate for the predator must be greater than the birth rate and the influx from hunted prey.

For the steady state with only the predator we have similar to the previous case, $\overline{N}_1 = 0$ and $v_2(\overline{N}_2) = \mu_2$. The Jacobian is now

$$J = \begin{pmatrix} -\phi\overline{N}_2 - \mu_1 + v_1(0) & 0 \\ e\phi\overline{N}_2 & v_2'(\overline{N}_2)\overline{N}_2 \end{pmatrix} \tag{28}$$

From this matrix we get eigenvalues $\overline{N}_2 v_2(\overline{N}_2)'$ and $-\phi N_2 - \mu_1 + v_1(0)$. The first is negative if and only if the derivate of the birth function is negative. This is the exact assumption that was made earlier. The second condition

is $v_1(0) \leq \mu_1 + \phi\overline{N}_2$. This means that the death rate for the prey and its outflux due to predation must be greater than the birth rate.

The steady state with both species has $v_1(\overline{N}_1) = \mu_1 + \phi\overline{N}_2$ and $v_2(\overline{N}_2) = \mu_2 - e\phi\overline{N}_1$. This steady state has the Jacobian

$$J = \begin{pmatrix} \overline{N}_1 v_1'(\overline{N}_1) & -\phi\overline{N}_1 \\ e\phi\overline{N}_2 & \overline{N}_2 v_2'(\overline{N}_2) \end{pmatrix} \tag{29}$$

From this matrix we see that the trace is negative. This is follows from the fact that the birth rate functions are decreasing. We also see that the determinant is positive. These together tell us that whenever a positive equilibrium exists, it is stable.

5.2 Prey and pathogen

Lets consider a situation without the predator. The system then reduces to

$$\dot{N}_1 = v_1(N_1)N_1 - \mu_1 N_1 - \alpha_1 I_1 \tag{30}$$

$$\dot{I}_1 = \beta_1 \frac{S_1 I_1}{N_1} - \mu_1 I_1 - \alpha_1 I_1 + \kappa S_1 I_1. \tag{31}$$

The steady state of this solves to

$$\overline{I}_1 = \frac{v_1(\overline{N}_1)\overline{N}_1 - \mu_1\overline{N}_1}{\alpha_1} \tag{32}$$

and

$$\overline{I}_1 = \overline{N}_1(1 - \frac{\mu_1 + \alpha_1}{\beta_1 + \kappa\overline{N}_1}). \tag{33}$$

We can see that this steady state exists only if

$$v_1(\overline{N}_1) > \mu_1 \tag{34}$$

and

$$\frac{\mu_1 + \alpha_1}{\beta_1 + \kappa \overline{N}_1} < 1 \tag{35}$$

Condition (34) tells us that at the steady state, the input of individuals is greater than the normal death rate when $\alpha_1 > 0$. By studying this at the population level $N_1 = 0$, we can see if the addition of the pathogen adds so much pressure that it kills the whole population. This way we can see that for the species and pathogen to coexist we need to have

$$\frac{\mu_1 + \alpha_1}{\beta_1 + \kappa \overline{N}_1} < 1 \tag{36}$$

Condition (35) is exactly the condition where $R_0^1 > 1$.

The stability of this steady state is determined by the Jacobian (23). Roberts and Heesterbeek give

$$H = \begin{pmatrix} \overline{N}_1 v_1'(\overline{N}_1) + \alpha_1 \overline{I}_1 / \overline{N}_1 & -\alpha_1 \\ \kappa \overline{I}_1 & -\beta_1 \overline{I}_1 / \overline{N}_1 \end{pmatrix} \tag{37}$$

and

$$C = \begin{pmatrix} v_2(0) + e\phi(\overline{S}_1 + q\overline{I}_1) - \mu_2 & -e\phi(1-p)(\overline{S}_1 + q\overline{I}_1) - \alpha_2 \\ cq\phi\overline{I}_1 & \beta_2 - \mu_2 - \alpha_2 \end{pmatrix} \tag{38}$$

H was the matrix that controls epidemiological stability. Roberts and Heesterbeek give the results that this state is stable whenever $\beta_1 > \alpha_1$ which is a result from $R_0^1 > 1$. C controls the ecological stability. No simple condition for stability of this matrix can be found.

5.3 Predator and pathogen

Consider a situation without the prey. The situation is very much similar to the one with prey and pathogen. The system reduces to

$$\dot{N}_2 = v_2(N_2)N_2 - \mu_2 N_2 - \alpha_2 I_2 \tag{39}$$

$$\dot{I}_2 = \beta_2 \frac{S_2 I_2}{N_2} - \mu_2 I_2 - \alpha_2 I_2 \tag{40}$$

Now we have a little simpler system because there is no environmental transmission involved. We can solve this steady state analytically and get

$$\frac{\overline{S}_2}{\overline{N}_2} = 1 - \frac{\overline{I}_2}{\overline{N}_2} = \frac{\mu_2 + \alpha_2}{\beta_2} = \frac{1}{R_0^2} \tag{41}$$

for the ratio of susceptibles.

It should be clear that the ratio cannot be greater than one and thus a condition for the existence of this state is that $R_0^2 > 1$.

We can also present this equation as

$$\frac{\overline{I}_2}{\overline{N}_2} = \frac{v_2(\overline{N}_2) - \mu_2}{\alpha_2}. \tag{42}$$

Another thing we have to consider is the increased death from infected individuals. It is possible that this effect drives the predator species extinct. By studying this effect near zero we can deduce from $I_2\alpha_2 + N_2\mu_2 < v_2(N_2)N_2$, a condition

$$\frac{v_2(0) - \mu_2}{\alpha_2} > 1 - \frac{1}{R_0^2} \tag{43}$$

for the survival of the species.

We can present for the predator and the pathogen, as we did for the prey and the pathogen, the same matrices. Roberts and Heesterbeek give

$$H = \begin{pmatrix} \overline{N}_2 v_2'(\overline{N}_2) + \alpha_2 \overline{I}_2/\overline{N}_2 & -\alpha_2 \\ 0 & -\beta_2 \overline{I}_2/\overline{N}_2 \end{pmatrix} \tag{44}$$

and

$$C = \begin{pmatrix} v_1(0) + \phi(\overline{S}_2 + p\overline{I}_2) - \mu_1 & -\phi q(\overline{S}_2 + p\overline{I}_2) - \alpha_1 \\ r\kappa \overline{I}_2 & \beta_1 - \mu_1 - \alpha_1 - \phi q(\overline{S}_2 + p\overline{I}_2). \end{pmatrix} \tag{45}$$

Eigenvalues for H are now easy to study. We get eigenvalues $-\frac{\overline{I}_2}{\overline{N}_2}\beta_2$ and $\frac{\overline{I}_2}{\overline{N}_2}\alpha_2 + \overline{N}_2 v_2'\left(\overline{N}_2\right)$. We can see that the first one is always negative. Thus the epidemiological stability of the state depends on the second eigenvalue. If we were to have a birth rate function $v_2 = \frac{B_2}{N_2}$ the condition would solve to $\overline{I}_2\alpha_2 < B_2$.

6 Critical consumption rate

Roberts and Heesterbeek present the existence of a critical value of ϕ. This is the value ϕ_c such that for any $\phi > \phi_c$, a steady state with predator and prey can not exist without the pathogen. They show that there exists at least some parameter values, for which a steady state exists with the pathogen, but not without it.

We can analyze ϕ_c further. It is evident that the value of ϕ_c depends on the parameters of the model. We can easily see that the dependence is only on the non-epidemiological parameters and the birth rate functions, which may have multiple parameters.

We can calculate ϕ_c for arbitrary parameter values by dividing functions (24a) and (24b) with N_1 and N_2, setting them equal to zero, and solving N_1 and N_2. By changing parameter ϕ we can examine when one or both of these steady state values become zero and negative.

A straigthforward way to solve ϕ_c for the prey is to take the two equations and set $N_1 = 0$. Then we can solve these for ϕ and N_2 to get

$$\phi_{c,prey} = \frac{v_1(0) - \mu_1}{v_2^{-1}(\mu_2)}, \tag{46}$$

where v_2^{-1} is the inverse of v_2 when it exists. For (46) to be meaningful $v_1(0)$ must be finite.

It is assumed that $v_1(0) > \mu_1$. This means that in order to have a positive $\phi_{c,prey}$ we must have $v_2(0) > \mu_2$ meaning that the predator survives on an alternative food source alone.

A critical consumption rate might exist for the predator too. Similarly we

get

$$\phi_{c,predator} = \frac{\mu_2 - v_2(0)}{e v_1^{-1}(\mu_1)} \tag{47}$$

for the predator.

$\phi_{c,predator}$ is now positive if and only if $v_2(0) < \mu_2$. This simply means that the predator must hunt enough prey to stay alive since the alternative food source is not enough.

It is now clear that only one species can and must have ϕ_c, because the existence of $\phi_{c,prey}$ requires $v_2(0) > \mu_2$ and the existence of $\phi_{c,predator}$ requires $v_2(0) < \mu_2$. This means that there does not exist such a combination of other parameters that guarantees an equilibrium with both species for every ϕ.

6.1 Exponentially decaying birth rate

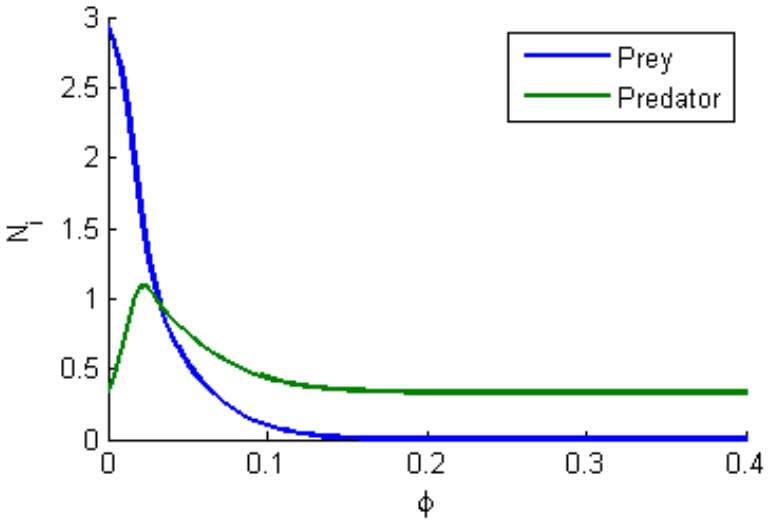

Figure 1: We can find ϕ_c by examining the system with different values of ϕ. Here we use parameter values $\alpha_1 = 0.02$, $\alpha_2 = 0.2$, $\mu_1 = 0.2$, $\mu_2 = 0.05$, $\rho_1 = 0.25$, $\rho_2 = 0.25$, $k1 = 0.0744$, $k2 = 2.7489$ and $e = 2.6185$. The graph shows the steady state values for both species.

The critical value of ϕ exists for birth rate functions of type $v_i(N_i) = \rho_i e^{(-k_i N_i)}$

where $\rho_1 > \mu_1$. From (46) we get

$$\phi_{c,prey} = k_2 \frac{\rho_1 - \mu_1}{\log \frac{\rho_2}{\mu_2}}. \tag{48}$$

It is worth noticing that the value of $\phi_{c,prey}$ does not depend on e or k_1. We also have a linear dependence on $\rho_1 - \mu_1$, which is the growth rate of the prey in the positive limit at zero without the predator. In the divisor of $\phi_{c,prey}$ we have $\log \frac{\rho_2}{\mu_2}$, which can be written as $\log \rho_2 - \log \mu_2$.

Figure 1 shows how we can determine the $\phi_{c,prey}$. Somewhere around $\phi = 0.15$ the prey population ceases to exists. This is the $\phi_{c,prey}$. Using equation (48) we can calculate the exact value. For parameter values $\alpha_1 = 0.02$, $\alpha_2 = 0.2$, $\mu_1 = 0.2$, $\mu_2 = 0.05$, $\rho_1 = 0.25$, $\rho_2 = 0.25$, $k1 = 0.0744$, $k2 = 2.7489$ and $e = 2.6185$ this is $\phi_{c,prey} = 0.150$.

For the predator we have

$$\phi_{c,predator} = k_1 \frac{\mu_2 - \rho_2}{e \log(\frac{\rho_1}{\mu_1})}. \tag{49}$$

6.2 Constant births

If the birth rate function is of type $v_i(N_i) = \frac{B_i}{N_i}$, there would not be a ϕ_c. This can be seen from equation (24a), which becomes

$$\frac{B_1}{N_1} = \mu_1 + \phi N_2. \tag{50}$$

If N_1 were to approach zero now, we would observe ϕ or N_2 approaching infinity. This effect is due to the constant influx of prey in the model.

Simimarly the influx of predators is constant, and thus they cannot go extinct.

6.3 Linearly decreasing birth rate

The birth rate function can also be of type $v_i = V_i - c_i N_i$ for $N_i < \frac{v_i}{c_i}$ and $v_i = 0$ for $N_i \geq \frac{v_i}{c_i}$. This choice of birth rate function leads us to a logistic

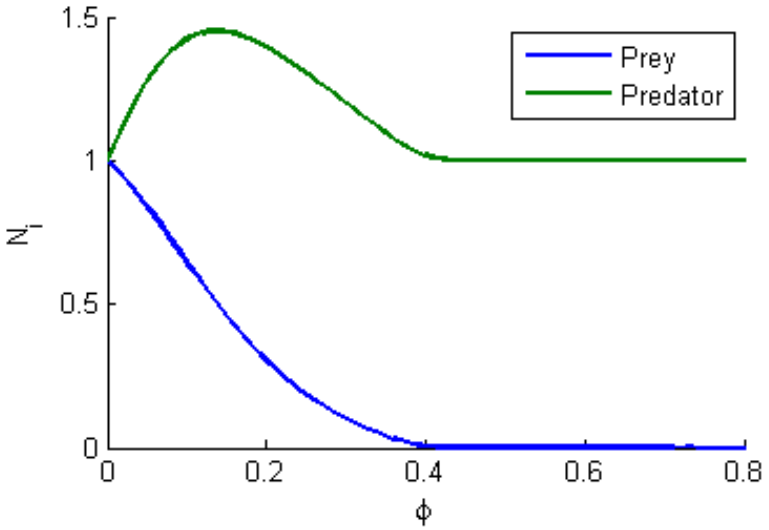

Figure 2: We can find ϕ_c by examining the system with different values of ϕ. Here we use parameter values $\alpha_1 = 0.02$, $\alpha_2 = 0.2$, $\mu_1 = 0.2$, $\mu_2 = 0.05$, $V_1 = 0.6$, $V_2 = 0.5$, $c_1 = 0.4$, $c_2 = 0.4$ and $e = 2.6185$. The graph shows the steady state values for both species.

equation.[14] We can again solve the equations 24a and 24b for ϕ to get

$$\phi_{c,prey} = c_2 \frac{V_1 - \mu_1}{V_2 - \mu_2}. \tag{51}$$

$\phi_{c,prey}$ now only depends on the constant c_2 and the differences $V_i - \mu_i$, which are assumed to be positive.

Figure 2 shows how the steady state values of both species change with the parameter ϕ. From the figure we can see that $\phi_{c,prey}$ is about 0.4. This is the same that equation (51) gives.

For the predator we have

$$\phi_{c,predator} = c_1 \frac{\mu_2 - V_2}{V_1 - \mu_1}. \tag{52}$$

6.4 Other cases

The three aforementioned birth-rate functions are commonly used in modeling. It is possible for the prey and the predator to have different type of functions for their respective birth-rates. For example the predator might have an exponentially decreasing birth-rate function, whereas the prey would have a constant number of births per time unit.

6.5 Critical consumption rate as a bifurcation

What happens at the critical consumption rate is a transcritical bifurcation.[33] The normal form for the transcritical bifurcation is

$$\frac{dx}{dt} = rx - x^2. \tag{53}$$

If the birth rate function is $v_i(N_i) = V_i - C_i N_i$ then we have equation

$$\frac{dN_1}{dt} = (V_1 - \mu_1 - \phi N_2)N_1 - c_1 N_1^2 = rx - c_1 x^2 \tag{54}$$

for the prey. To be precise, we should not treat N_2 as a constant because it depends on ϕ. But in this case approximating it with a constant works.

Figure 2 shows how the prey becomes extinct at $\phi = 0.4$. In this situation $r = 0$ in the system, and a transcritical bifurcation happens. When r is positive the system is biologically meaningful, the positive equilibrium is stable and the equilibrium at zero unstable. When r is negative we have an unstable negative equilibrium and a stable equilibrium at zero.

The exponentially decreasing birth rate gives a transcritical bifurcation too. This can be confirmed by studying the Taylor expansion of it. We get

$$\frac{dN_1}{dt} \approx (\rho_1 - \mu_1 - \phi N_2)N_1 - k_1\rho_1 N_1^2 = rx - k_1\rho_1 x^2. \tag{55}$$

The situation is similar for the predator, $-\phi N_2$ is just replaced with $+e\phi N_1$. As long as the critical consumption rates for the prey and the predator are not the same, a transcritical bifurcation happens. We saw earlier that in any case, both critical values can't exist simultaneously.

7 Properties of an equilibrium

The predator-prey-pathogen system may or may not have an equilibrium which is or is not stable.

7.1 Population sizes

As can be seen from figures 1 and 2 increasing consumption rate does not mean that the predator population grows in size in the equilibrium. We can study the effect of ϕ on equilibrium sizes with implicit differentiation. Let population sizes be functions of ϕ. Then at an equilibrium we have

$$v_1(N_1(\phi))N_1(\phi) - \mu_1 N_1(\phi) - \phi N_1(\phi)N_2(\phi) = 0 \tag{56a}$$
$$v_2(N_2(\phi))N_2(\phi) - \mu_2 N_2(\phi) + e\phi N_1(\phi)N_2(\phi) = 0. \tag{56b}$$

Dividing (56a) by N_1 and (56b) by N_2 and using implicit differentiation we get

$$v_1'(N_1(\phi))N_1'(\phi) - \phi N_2'(\phi) - N_2(\phi) = 0 \tag{57a}$$
$$v_2'(N_2(\phi))N_2'(\phi) + e\phi N_1'(\phi) + eN_1(\phi) = 0. \tag{57b}$$

We can solve this system of equations for N_1' and N_2'. In the next part the arguments are not written out. We get

$$N_1' = \frac{v_2'(N_2)N_2 - e\phi N_1}{e\phi^2 + v_1'(N_1)v_2'(N_2)} \tag{58}$$

and

$$N_2' = e\phi\frac{-v_1'(N_1)N_1 - \phi N_2}{e\phi^2 + v_1'(N_1)v_2'(N_2)}. \tag{59}$$

We can see that N_1' is always negative since the birth rate functions are decreasing and N_1 and N_2 are assumed to be positive equilibrium values. N_2' is more complicated. N_2' is positive whenever $-v_1'(\overline{N}_1)\overline{N}_1 > \phi\overline{N}_2$. This holds at least at $\phi = 0$ which means that when starting from a situation of no

predation, the equilibrium population size of the predator increases initially with the consumption rate.

Earlier we saw that with with certain assumptions the model has a critical consumption rate where the prey goes extinct. It is clear that at this point the equilibrium value of the predator is equal to that where the consumption rate is zero. From (59) we can see that the derivative of N_2 is negative at the critical consumption rate, which is to be expected. We can expect to find a unique global maximum for the equilibrium value of the predator.

The birth rate of the prey might be such that the critical consumption rate does not exist. One example of a situation like this is when the birth rate function for the prey is (50). Now for the predator we have $\overline{N}_2 = \frac{B_1}{\phi \overline{N}_1} - \frac{\mu_1}{\phi}$.

$$N_2' = e\phi \frac{-v_1'(N_1) - \phi N_2}{e\phi^2 + v_1'(N_1)v_2'(N_2)} = \frac{e\phi\mu_1}{e\phi^2 + v_1'(N_1)v_2'(N_2)} \tag{60}$$

which is always positive. In this case the predator population keeps growing with the consumption rate. The population does not grow indefinitely, however. It has a limit when the consumption rate approaches infinity. To see why this is, we must study the equilibrium values at infinity. When the consumption rate is high enough, practically every prey dies because of the predation. This gives us the approximation

$$N_1 \approx \frac{B_1}{\phi N_2}. \tag{61}$$

We can now solve the equilibrium value of the predator implicitly from the equation

$$\overline{N}_2 = \frac{eB_1}{\mu_2 - v_2(\overline{N}_2)}. \tag{62}$$

If we were to have $v_2(N_2) = \frac{B_2}{N_2}$, the solution would be

$$\overline{N}_2 = \frac{eB_1 + B_2}{\mu_2}. \tag{63}$$

We can do this with respect to any parameter. Letting population sizes be

functions of e, we find that

$$N_1' = \frac{-\phi^2 N_1}{e\phi^2 + v_1'(N_1)v_2'(N_2)} \tag{64}$$

and

$$N_2' = v_1'(N_1)N_1' \tag{65}$$

We can see that $N_1' < 0$ and $N_2' > 0$. These results are as expected. The better the predator is in turning prey into offspring, the more it gains in population sizes and thus decreases the prey population.

With death rates, we find that increasing μ_1 decreases both populations and increasing μ_2 decreases predator population, but increases prey population.

These properties carry on to infected equilibriums in some extent. The parts regarding other factors than the consumption rate also hold for the cases with trade-offs.

7.2 Proportion of infected

The proportion of infected individuals is interesting. All susceptible individuals die at the same rate μ, but the infected have additional death rate α. It is clear that higher α drives the number of infected individuals down. Both of these factors have to be compared to birth-rates, which produce only healthy individuals. In addition to this birth-death mechanism, there is also predation and environmental transmission acting in the system. In the end, the final ratio of infected individuals is a complex result of these mechanisms.

In the following examples the exponentially decreasing birth-rate function is used.

Predation is dependent on parameters p, q, e and ϕ. ϕ increases the removal of preys. This has the effect of lowering the ratio of infected prey in population. The effect of ϕ is very much dependent on p and q. When studying the ratio of infected prey, q is important. The higher the q the more infected prey are removed due to predation. This effect is shown in figure 3. The figure shows how the ratio of infected individuals changes in the equilibrium when ϕ varies. It can be seen that change in q only affects significantly the ratio in prey. The higher the q, the faster the decrease.

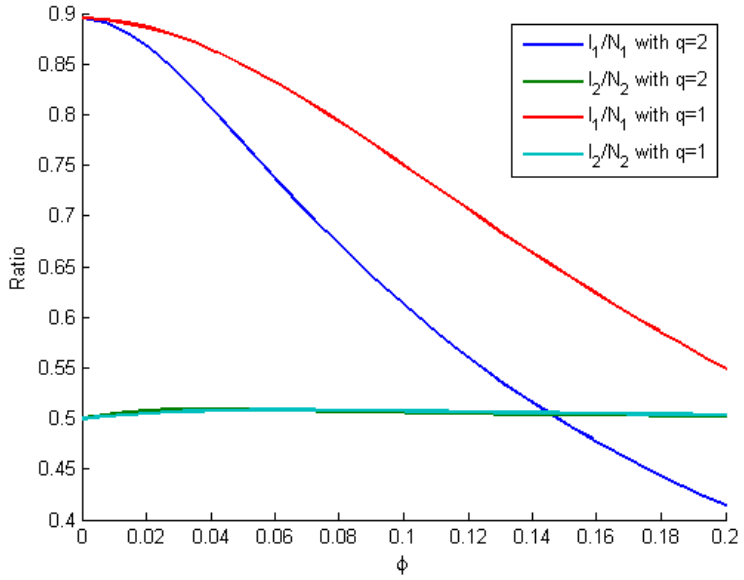

Figure 3: Parameter values used here are $\alpha_1 = 0.02$, $\alpha_2 = 0.2$, $\beta_1 = 0.209$, $\beta_2 = 0.6$, $\mu_1 = 0.2$, $\mu_2 = 0.1$, $\kappa = 1$, $\rho_1 = 0.25$, $\rho_2 = 0.25$, $k_1 = 0.0744$, $k_2 = 2.7489$, $c = 0.1$, $p = r = 1$ and $e = 1.2$. The ratio of the infected in prey population decreases faster with a higher value of q.

Changing the value of p has the same kind of an effect as q. It has no other effect on the equilibrium ratio,s other than on the ratio of infected prey. r relates the output of infectious material to the environment of prey and predators. If $r > 1$, the predators output relatively more material to the environment than the prey. κ then describes the strength of the whole environmental transmission. It should be clear that increasing these factors increases the ratio of infected prey. They should, however, have no other than secondary effects on the ratio of infected predators since predators can not get infected through the environment. The relation of the ratio of infected prey to e, however, is somewhat more complicated. When e is low, the predator population does not grow so much from predation, and the predator population settles for a smaller value. Thus the prey gets hunted less and the ϕN_2 is effectively smaller than it would be for a larger e. Thus the ratio of infected prey decreases with increasing e, regardless of q. This effect is demonstrated in figure 5.

The previous example shed light on the ratio of the infected in the prey population. The predator population was only a little or not at all affected. Important factors for the predator are c, which describes how the infection

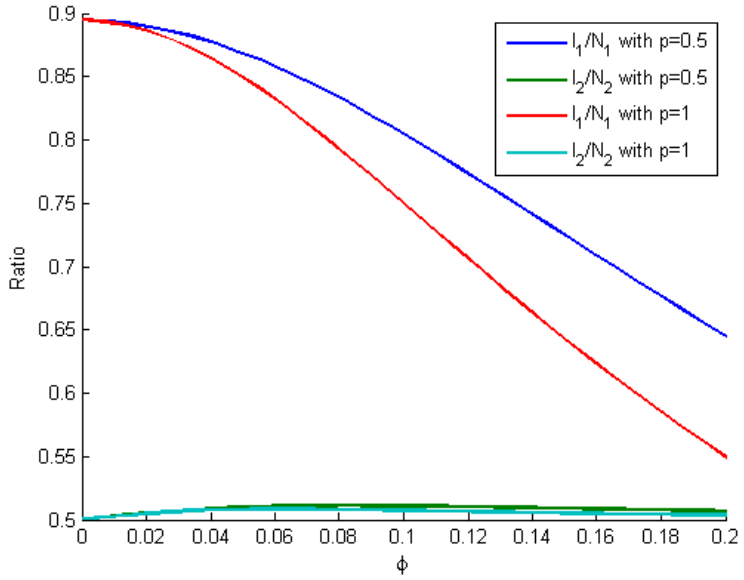

Figure 4: Parameter values used here are $\alpha_1 = 0.02$, $\alpha_2 = 0.2$, $\beta_1 = 0.209$, $\beta_2 = 0.6$, $\mu_1 = 0.2$, $\mu_2 = 0.1$, $\kappa = 1$, $\rho_1 = 0.25$, $\rho_2 = 0.25$, $k_1 = 0.0744$, $k_2 = 2.7489$, $c = 0.1$, $q = r = 1$ and $e = 1.2$. The ratio of the infected in prey population decreases faster with a higher value of p.

spreads through consuming infected prey, and possibly e.

Figure 6 shows how, quite naturally, the ratio of infected predators increases with c. The effect is strengthened by a high q, which tells how much infected prey are being preyd upon. When studying e, it can be seen that this does not affect the ratio of infected predators much. Other than c, parameters α, β and μ determine the equilibrium value of infected predators.

8 Adaptive dynamics

Adaptive dynamics (AD) is a framework, which has been developed since 1990s and is used to study evolution. It uses evolutionary game theory to implement and to study ecological systems realistically. The foundation of adaptive dynamics is laid in papers by Metz et al.[25] Dieckmann and Law[9], Metz et al.[24] and Geritz et al.[16] [3]

Adaptive dynamics investigates the evolution of a single or multiple traits that individuals carry. The first assumption in adaptive dynamics is that the

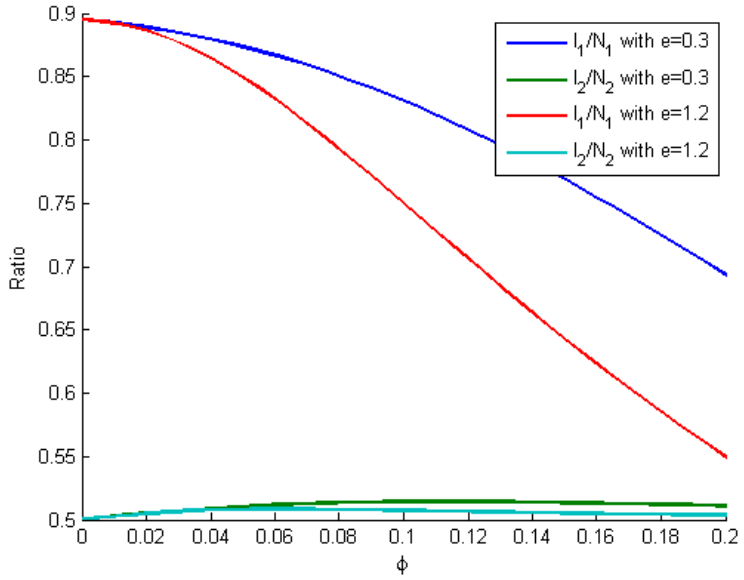

Figure 5: Parameter values used here are $\alpha_1 = 0.02$, $\alpha_2 = 0.2$, $\beta_1 = 0.209$, $\beta_2 = 0.6$, $\mu_1 = 0.2$, $\mu_2 = 0.1$, $\kappa = 1$, $\rho_1 = 0.25$, $\rho_2 = 0.25$, $k_1 = 0.0744$, $k_2 = 2.7489$, $c = 0.1$ and $q = p = r = 1$. The ratio of the infected in prey population decreases faster with a higher value of e.

evolution is slow compared to population dynamics. Therefore the population is always at an equilibrium, when a new mutant is introduced. When a mutant is introduced, its fate is determined by its initial growth rate. The initial growth rate is called *invasion fitness*.[3] The evolution is assumed to progress in small, but discrete steps. The trait of the mutant is thus always close to that of the resident population's trait value.

The following is the formal representation of adaptive dynamics by Geritz et al.[16]

Let E_x be the environment in a population with only traits x. In our model this environment would mean the amount of infected prey and predators, which is determined by the trait of the predator. Also let $r(x, E_x)$ be the long-term exponential growth rate for the population. In an equilibrium we have

$$r(x, E_x) = 0. \tag{66}$$

We then want to study what happens when a mutant with trait y is in-

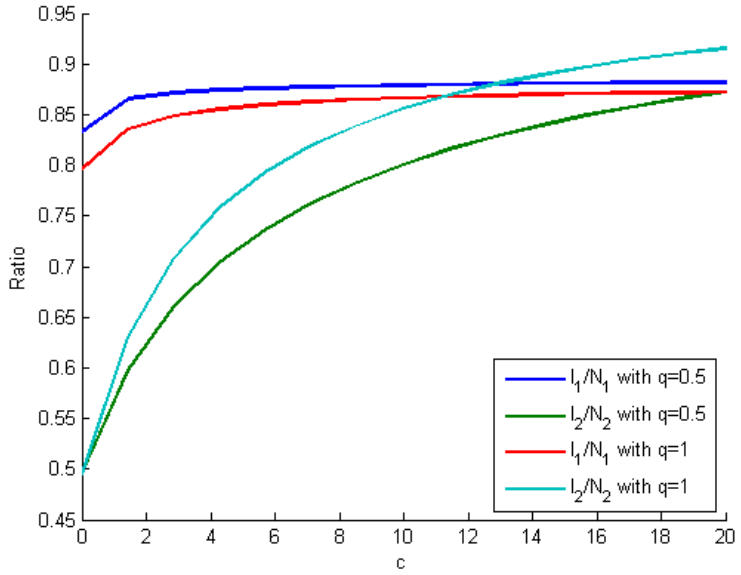

Figure 6: Parameter values used here are $\alpha_1 = 0.02$, $\alpha_2 = 0.2$, $\beta_1 = 0.209$, $\beta_2 = 0.6$, $\mu_1 = 0.2$, $\mu_2 = 0.1$, $\kappa = 1$, $\rho_1 = 0.25$, $\rho_2 = 0.25$, $k_1 = 0.0744$, $k_2 = 2.7489$, $e = 0.3$ and $p = r = 1$. The ratio of infected in prey population decreases faster with higher value of e.

troduced into this population. The exponential growth rate, fitness, of the mutant is

$$s_x(y) = r(y, E_x). \tag{67}$$

The environment is a function of x only because the size of the mutant population in the beginning is so small it does not affect the environment yet. Now, depending on the sign of $s_x(y)$ the mutant either spreads ($s_x(y) > 0$) or dies out ($s_x(y) < 0$). We can define the local fitness gradient as

$$D(x) = \left[\frac{\partial s_x(y)}{\partial y}\right]_{y=x}. \tag{68}$$

When $D(x) > 0$ ($D(x) < 0$) only mutants with $y > x$ ($y < x$) can invade the resident population. The interesting trait values are those that have $D(x) = 0$. These are called evolutionary singular strategies. The full story about what can happen at an evolutionary singular strategy is presented in Geritz et al.[16] One thing that can happen is that no other strategy

can invade it and the evolution stops. With certain conditions a dimorphic population with two adapting traits can arise.

8.1 Properties of a singular strategy

The singular strategy is evolutionarily stable if

$$\frac{\partial^2 s_\phi(\phi_{mut})}{\partial \phi_{mut}^2} < 0. \tag{69}$$

In our model in the absence of the pathogen we have always $\frac{\partial^2 s_\phi(\phi_{mut})}{\partial \phi_{mut}^2} = 0$ because the fitness depends on ϕ_{mut} linearly making the second derivative zero.

The singular strategy is convergence stable if

$$\frac{\partial^2 s_\phi(\phi_{mut})}{\partial \phi^2} > \frac{\partial^2 s_\phi(\phi_{mut})}{\partial \phi_{mut}^2} \tag{70}$$

Without the pathogen we had $\frac{\partial^2 s_\phi(\phi_{mut})}{\partial \phi_{mut}^2} = 0$ making the condition

$$\frac{\partial^2 s_\phi(\phi_{mut})}{\partial \phi^2} > 0. \tag{71}$$

This cannot be calculated analytically, because even the equilibrium values cannot be solved other than numerically.

9 Evolution of consumption rate

The evolution of traits is complicated and hard to study in nature.[2] Currently the most popular view about predator-prey coevolution sees it as an arms race.

In our model we only have the predator as an evolutionary agent. The prey does not evolve at all. Models that give similar predictions for the evolution of the trait as our model without the pathogen have been presented by Schaffer and Rosenzweig.[30][28]

Some examples on how trade-offs in traits are used to control the evolution have been presented by Brodie[6] and Kraaijeveld and Godfray[20]. Evolution with trade-offs is considered in later sections.

9.1 Without pathogen

In section 6 it was shown that for a certain type of birth-rate functions, there exists a threshold value for consumption rate ϕ. It is convenient to use adaptive dynamics to study if the evolution of the consumption rate brings it over the critical value driving the prey species into extinction.

Let ϕ be the trait the evolution of which we study. In a situation without the pathogen the population reaches a stable equilibrium as long as $\phi < \phi_c$. The stability of this equilibrium can be seen from 29. The steady state values $\overline{N}_1$ and $\overline{N}_2$ can be solved from (24a) and (24b). Then the growth rate of and individual with a trait value of ϕ_{mut}, can be solved from (24b) with $\phi = \phi_{mut}$ and $N_i = \overline{N}_i$. We get for the change of the mutant population M per time unit

$$\frac{dM}{dt} = v_2(\overline{N}_2)M + e\phi_{mut}\overline{N}_1 M - \mu_2 M. \tag{72}$$

It is clear that (72) is an increasing function of ϕ_{mut}. This leads us to see that the selection gradient $D(x)$ from (68) is always positive. Evolutionarily this means that if there is nothing stopping the increase of the consumption rate ϕ, it will continue evolve to higher values. Eventually this leads to $\phi > \phi_{c,prey}$ in situations that have the critical value, and therefore to the extinction of the prey. The extinction of the prey then stops the increase in the consumption rate as consumption becomes meaningless. Even in the model with constant births, the prey is practically driven to extinction, because the equilibrium value of N_1 decreases with increasing ϕ. When ϕ approaches infinity, N_1 approaches zero.

If we have $v_2(0) < \mu_2$ and thus $\phi_{c,predator}$ exists, we don't see prey going extinct.

9.2 With pathogen

A similar process happens with the pathogen. In this case we have a structured population and the system is therefore a bit more complicated. We can,

of course, reach all the similiar situations as before by choosing pathogen related parameters, so that the effect of the pathogen disappears.

We have to study the eigenvalues of the matrix for the growth of the mutant. For a rare mutant we have initially the following system of differential equations

$$\begin{aligned} \frac{ds}{dt} &= -cq\phi_{mut}I_1 s - \frac{\beta_2}{N_2+n}(i+I_2)s - \mu_2 s \\ &\quad +e\phi_{mut}(S_1+qI_1)(s+pi) + v_2(N_2+n)(i+s) \qquad (73a) \\ \frac{di}{dt} &= cq\phi_{mut}I_1 s + \frac{\beta_2}{N_2+n}(i+I_2)s - \mu_2 i - \alpha_2 i. \qquad (73b) \end{aligned}$$

Linearizing this system around $(s,i) = (0,0)$ we get a linear system

$$\frac{dM}{dt} = AM, \qquad (74)$$

where $M = [s,i]^T$ and

$$A = \begin{pmatrix} a_m & b_m \\ c_m & d_m \end{pmatrix}. \qquad (75)$$

where

- $a_m = v_2(\overline{N}_2) + e\phi_{mut}(\overline{S}_1 + q\overline{I}_1) - \mu_2 - cq\phi_{mut}\overline{I}_1 - \frac{\beta_2\overline{I}_2}{\overline{N}_2}$
- $b_m = v_2(\overline{N}_2) + ep\phi_{mut}(\overline{S}_1 + q\overline{I}_1)$
- $c_m = cq\phi_{mut}\overline{I}_1 + \frac{\beta_2\overline{I}_2}{\overline{N}_2}$
- $d_m = -\alpha_2 - \mu_2$

Whether or not the mutant can invade depends now on the eigenvalues of this matrix. We can, however, study the invasion fitness by calculating only the trace and determinant. For the equilibrium of a linearized system to be stable we must have $trace(A) < 0$ and $det(A) > 0$.[18]

Figure 7 shows the characterization of equilibria by trace and determinant of the linear system. There are two ways to leave the upper west area, which is stable. We can leave it by going down to the saddle area, or by going right to the unstable foci. What we are studying is a linear system of two variables,

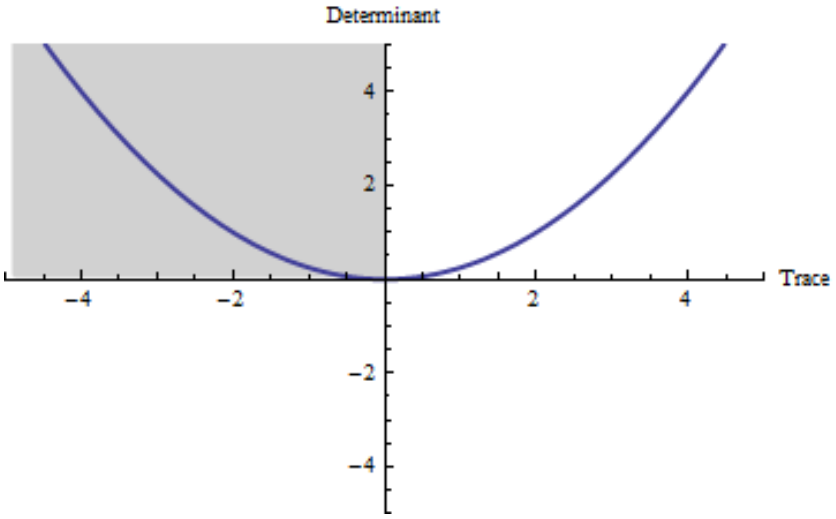

Figure 7: The trace-determinant plane and the classification of equilibria. The grey area is the stable area. The area above the curve has foci and thus cannot be achieved in this biological system.

infected mutants and susceptible mutants. These can be only positive or zero. It is then clear that the system cannot have foci. This is usually the case in biological settings because we are dealing with non-negative population levels.

Thus the only way for the system to transform from stable to unstable is for the determinant to change its sign. This doesn't mean that a change in the sign of the determinant necessarily means a change in eigenvalues. We could have a positive trace and think that having a change of the determinant means no change in system dynamics. But we must remember that the determinant crosses zero when changing from positive to negative and this induces a bifurcation in the system.

If we can show that the determinant being positive implies that the trace is negative, we can see that the determinant is a valid fitness proxy. Because we have $d_m < 0$ to have $a_m d_m$ positive, we must have $a_m < 0$. Now, if this is true, the trace of the matrix is negative. We can see that $b_m c_m > 0$ since both b_m and c_m are positive. The effect of this product is negative on the determinant and if we have a positive determinant, we must have positive product $a_m d_m$. This was the same condition that implied that the trace is negative. Thus, we can conclude that the determinant is a proper fitness proxy.

We can study simpler cases of this easier than the whole system. If the

pathogen does not harm the predator in any way, we have $\alpha_2 = 0$ and $p = 1$. In this case we have eigenvalues

$$\lambda_1 = -\frac{1}{\overline{N}_2}(cq\phi_{mut}\overline{I}_1\overline{N}_2 + \mu_2\overline{S}_2 + (\beta_2 + \mu_2)\overline{I}_2) \tag{76a}$$

$$\lambda_2 = v_2(\overline{N}_2) - \mu_2 + e\phi_{mut}(\overline{S}_1 + q\overline{I}_1). \tag{76b}$$

The first of these is always negative and the second one is positive whenever $\phi_{mut} > \phi$. This leads to uncontrolled increase in the consumption rate similarly as before. We can conclude that a harmless pathogen can't negate the increase in consumption rate.

With $\alpha_2 > 0$ or $p < 1$ we have a more complicated system. It is possible that the pathogen transmitted through consumption reduces the predators capability and increases death rate so much that at some point, increasing consumption rate would not be good for the predator. The pathogen offers more possibilities for the system. We can study the whole system with the help of the determinant

$$det(A) = a_m d_m - b_m c_m. \tag{77}$$

We can calculate $det(A)$ and see that it is a polynomial of ϕ_{mut} of second degree. (69) gives the condition for a singular strategy to be evolutionarily stable. Now, using (77) as our invasion fitness we get

$$\frac{\partial^2 s_\phi(\phi_{mut})}{\partial \phi_{mut}^2} = -2cepq\overline{I}_1(\overline{S}_1 + q\overline{I}_1). \tag{78}$$

This is either zero or negative meaning that the singular strategy is always an ESS. Having (78) equal to zero means having a degenerate case with a vertical line in the pairwise invadability plot. This happens when c, e, p or q equals zero or in the absence of the pathogen. However in the absence of the pathogen, we don't even have a singular strategy. The same goes for some of the other cases. If we have $e = 0$, the predator achieves nothing from consuming the prey and if there is a possibility of getting infected from consuming, the evolutionary drives should be towards zero.

Figure 8 shows the equilibrium values for predator and prey with different values of ϕ with chosen parameters. What is interesting is that the total

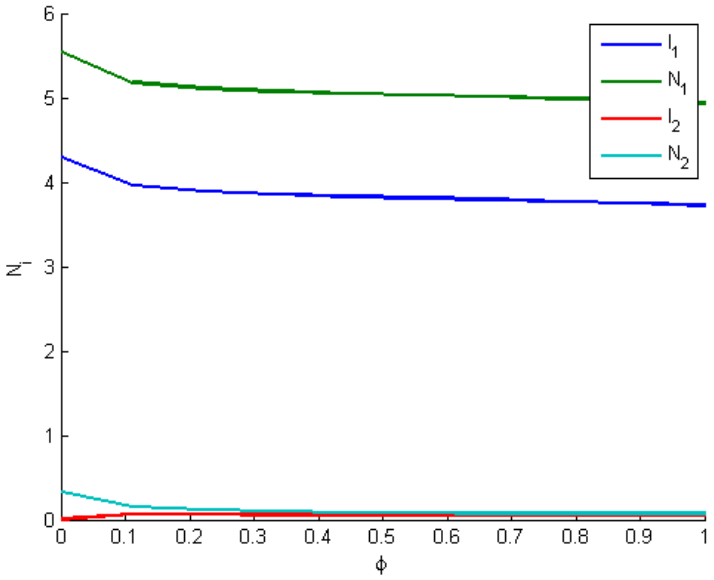

Figure 8: Parameter values used here are $\alpha_1 = 0.02$, $\alpha_2 = 0.2$, $\beta_1 = 0.209$, $\beta_2 = 0.2$, $\mu_1 = 0.15$, $\mu_2 = 0.1$, $\kappa = 0.1$, $\rho_1 = 0.25$, $\rho_2 = 0.25$, $k_1 = 0.0744$, $k_2 = 2.7489$, $e = 0.1$, $q = 0.3$, $p = 0.05$, $r = 1$ and $c = 0.95$. Equilibrium values are for sick and healthy predator and prey with different values of ϕ.

amount of predators decreases with the consumption rate. This is a consequence of both the disease spreading to the predator through prey and the total amount of prey decreasing, which affects the consumption. Keeping this in mind it would seem unwise for the predator to increase its consumption rate. This is something that could be seen in a situation without the pathogen, too. With high enough consumption the predator population decreases. In that case the evolutionary drive was for higher consumptions anyway, because a single predator wants what is best for it, not what is best for the population.

Figure 9 shows the pairwise invadability plot of the situation. Combining the information from figures 8 and 9 it is possible to determine what happens in this case. With any given original value of ϕ, the population goes to a steady state. Then with successive mutations and invasions ϕ decreases to zero. At zero the infected part of the predator population vanishes. Effectively the prey and predator population start to live in isolation. Predator could still infect prey through environmental transmission if only there were any infected predators. As figure 8 shows, with $\phi = 0$ there are no sick predators. This is due to the fact that we have $R_0^2 = \frac{\beta_2}{\mu_2+\alpha_2} = \frac{0.2}{0.1+0.2} = \frac{2}{3} < 1$. In prey

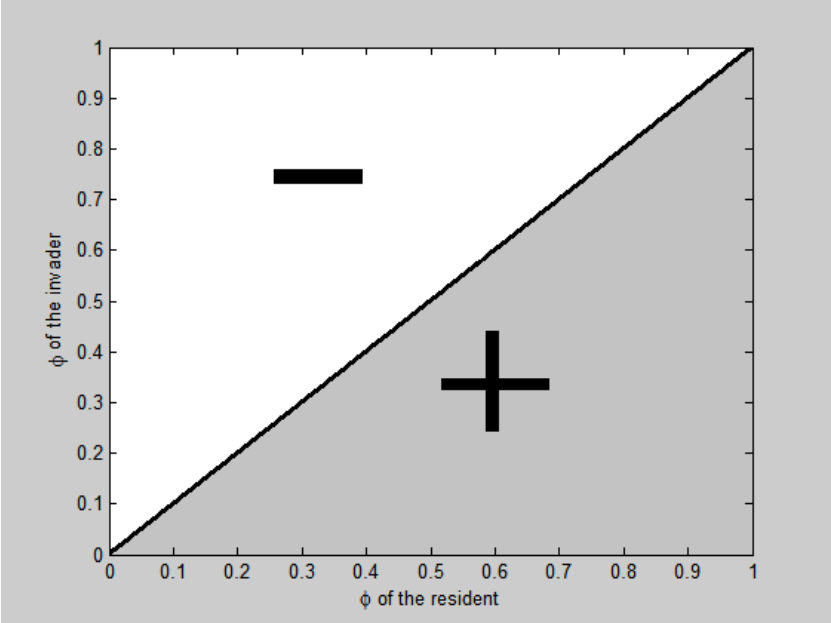

Figure 9: The pairwise invadability plot for the system with a pathogen related to figure 8. Parameter values used here are $\alpha_1 = 0.02$, $\alpha_2 = 0.2$, $\beta_1 = 0.209$, $\beta_2 = 0.2$, $\mu_1 = 0.15$, $\mu_2 = 0.1$, $\kappa = 0.1$, $\rho_1 = 0.25$, $\rho_2 = 0.25$, $k_1 = 0.0744$, $k_2 = 2.7489$, $e = 0.1$, $q = 0.3$, $p = 0.05$, $r = 1$ and $c = 0.95$. The direction of evolution is always negative.

population the disease persists. When the consumption rate is positive, the infection in the predator population is maintained with incoming infection from the prey population.

Seeing this model can have both decrease and increase in consumption, it might be possible to have both of these at the same time leading to a single evolutionary stable value of ϕ.

Figure 10 shows the equilibrium values of the populations for certain parameters. When consumption rate is zero, the predator population is healthy. With a high enough consumption rate the predator hunts the prey to extinction. Before this happens, the sick prey are hunted to extinction and for a certain interval of ϕ both species are healthy. Hunting the sick preys to extinction does not mean that only the prey that is sick is being hunted, but that the pathogen is not viable in that parameter range. The amount of prey decreases with increasing ϕ and the amount of prey is a factor in the basic reproduction number (19). Even though the basic reproduction numbers for the isolation case don't represent the situation exactly, they offer an approxi-

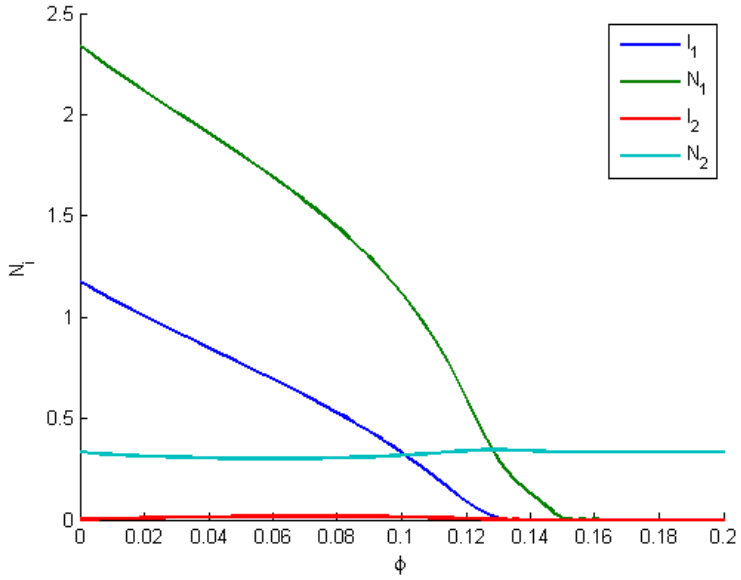

Figure 10: Parameter values used here are $\alpha_1 = 0.02$, $\alpha_2 = 0.3$, $\beta_1 = 0.209$, $\beta_2 = 0.2$, $\mu_1 = 0.2$, $\mu_2 = 0.1$, $\kappa = 0.1$, $\rho_1 = 0.25$, $\rho_2 = 0.25$, $k_1 = 0.0744$, $k_2 = 2.7489$, $e = 0.1$, $q = 0.3$, $p = 0.05$, $r = 1$ and $c = 0.95$. The equilibrium values are for the sick and the healthy predator, and prey, with different values of ϕ.

mation. We now see that we have $R_0^1 = \frac{\beta_1+\kappa N_1}{\mu_1+\alpha_1} \approx \frac{0.209+0.1\times 0.3}{0.2+0.02} = 1.086$, where the pathogen disappears from the prey.

Figure 11 shows the pairwise invadability plot of the situation. We can see that there is a singular strategy at value $\phi_s = 0.09$. This time the strategy is not attracting. With any trait value evolution drives the consumption rate away from ϕ_s. If the consumption rate is originally lower than this it is driven to zero and species become isolated. If the consumption rate is originally higher than ϕ_s, it is driven higher and the prey is hunted to extinction. The repelling singular strategy is also an ESS. This is sometimes called "The garden of Eden" after Nowak and Sigmund.[26] It cannot be reached in any way once it is lost.

The disease in these cases helps controlling the consumption rate in a sense. It makes it possible to make hunting the prey harmful to the predator so that the predator stops the hunting for good. It doesn't, however, bring a stable positive value for the consumption rate. The possible outcomes only include constantly increasing consumption rate or constantly decreasing consumption

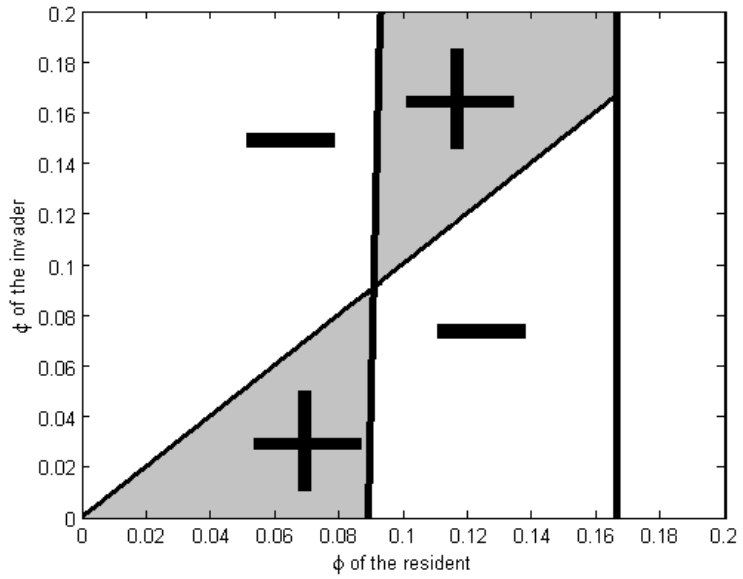

Figure 11: The pairwise invadability plot for the system with a pathogen related to figure 10. Parameter values used here are $\alpha_1 = 0.02$, $\alpha_2 = 0.3$, $\beta_1 = 0.209$, $\beta_2 = 0.2$, $\mu_1 = 0.2$, $\mu_2 = 0.1$, $\kappa = 0.1$, $\rho_1 = 0.25$, $\rho_2 = 0.25$, $k_1 = 0.0744$, $k_2 = 2.7489$, $e = 0.1$, $q = 0.3$, $p = 0.05$, $r = 1$ and $c = 0.95$. The tilting of the line is exaggerated. In reality the slope is very close to vertical.

rate. The latter, of course, leads to the case with no predation.

10 Trade-off between consumption rate and birth rate

More interesting situations arise when ϕ is connected to other qualities of the predator. These qualities could be the birth-rate or the death-rate. Increasing the consumption rate could bring more dangerous situations to the predator or be exhausting enough to increase death rate. It would be more natural that increased consumption of the prey would decrease the predators capability to take advantage of alternative food sources. Thus increasing the consumption rate would be countered with a decreasing birth-rate function. Finding a proper connection with these rates is a difficult modeling task.

Trade-offs in adaptive dynamics have been discussed in depth by Rueffler at. al.[29] When dealing with trade-off functions, performing a critical function

analysis is sometimes of use. It is a method introduced by de Mazancourt and Dieckmann.[8] The method can help uncover evolutionary scenarios that would otherwise be overlooked.[15]

One way to build a trade-off between consumption rate and birth rate of the predator is to think of them as exclusive activities. Whatever time is used to hunt the prey can not be used to consume the alternative food source. This leads us to equations

$$\dot{N}_1 = v_1(N_1)N_1 - \mu_1 N_1 - \alpha_1 I_1 - \phi\Phi(S_1 + qI_1)(S_2 + pI_2) \tag{79a}$$
$$\dot{N}_2 = (1-\phi)v_2(N_2)N_2 - \mu_2 N_2 - \alpha_2 I_2 + e\phi\Phi(S_1 + qI_1)(S_2 + pI_2) \tag{79b}$$
$$\dot{I}_1 = \beta_1 \frac{S_1 I_1}{N_1} - \mu_1 I_1 - \alpha_1 I_1 - \phi\Phi q I_1(S_2 + pI_2) + \kappa S_1(I_1 + rI_2) \tag{79c}$$
$$\dot{I}_2 = \beta_2 \frac{S_2 I_2}{N_2} - \mu_2 I_2 - \alpha_2 I_2 + cq\phi\Phi I_1 S_2. \tag{79d}$$

Here Φ is a constant describing the effect of the consumption at its maximum. $\phi \in [0, 1]$ is now the proportion of time the predator uses to hunt the prey. The rest of the time is used to feed on the alternative food source. Without the pathogen this system reduces to

$$\dot{N}_1 = v_1(N_1)N_1 - \mu_1 N_1 - \phi\Phi N_1 N_2 \tag{80a}$$
$$\dot{N}_2 = (1-\phi)v_2(N_2)N_2 - \mu_2 N_2 + e\phi\Phi N_1 N_2 \tag{80b}$$

Systems (79) and (80) are fundamentally different from the previous from an evolutionary point of view.

Now for the system (79) there are many possible outcomes when considering the evolution of the predator and its trait ϕ. Two possibilities are the end-points $\phi = 0$ and $\phi = 1$. Another would be to have any number of singular strategies inside the interval. In fact, it is possible to have any of these three different cases to exist for different parameter values.

10.1 Next-generation matrix

The next-generation matrix for this model is only a little different from the previous. The transition matrix T stays the same and the rate of predation

is changed in the transmission matrix Σ. We get

$$K = -T\Sigma^{-1} = \begin{pmatrix} \frac{\kappa N_1+\beta_1}{q\phi\Phi N_2+\alpha_1+\mu_1} & \frac{r\kappa N_1}{\alpha_2+\mu_2} \\ \frac{cq\phi\Phi N_2}{q\phi\Phi N_2+\alpha_1+\mu_1} & \frac{\beta_2}{\alpha_2+\mu_2} \end{pmatrix} \tag{81}$$

The basic reproduction numbers are different from previous, but with ecological isolation they are identical. We see that if the strength of the consumption ($\phi\Phi$) is equal to the strength of the consumption without the trade-off, the next-generation matrix is equal to (18).

10.2 Critical consumption rate

Critical consumption rate is somewhat different in this system. Both the prey and the predator can be driven to extinction with certain parameters. Previously the prey could only be driven to extinction with a high enough consumption rate and the predator with low enough consumption rate. The following shows that in this case the prey can be driven to extinction if the predator uses less of its time hunting the prey and the predator can go extinct if it spends too much time hunting.

The critical consumption rate can be calculated from (80) by dividing both equations by N_1 or N_2, setting them equal to zero and choosing N_1 or N_2 equal to zero. For the predator we have

$$\phi_{c,predator} = \frac{v_2(0) - \mu_2}{v_2(0) - e\Phi v_1^{-1}(\mu_1)}. \tag{82}$$

We now have two possibilities. If $v_2(0) - \mu_2 > 0$ the predator survives using the alternative food source only. In this case $\phi_{c,predator}$ exists and is between zero and one if $e\Phi v_1^{-1}(\mu_1) < \mu_2$.

Figure 12 shows the equilibrium values for both species with different values of ϕ. It can be seen from the figure and calculated from (82) that $\phi_{c,predator} = 0.6818$.

When $v_2(0) - \mu_2 < 0$ the predator cannot survive using the alternative food source only. In this case the only possibility for it is to hunt the prey enough to survive. In this case we must have $e\Phi v_1^{-1}(\mu_1) > \mu_2$ for the $\phi_{c,predator}$ and the predator population to exist for $\phi > \phi_{c,predator}$.

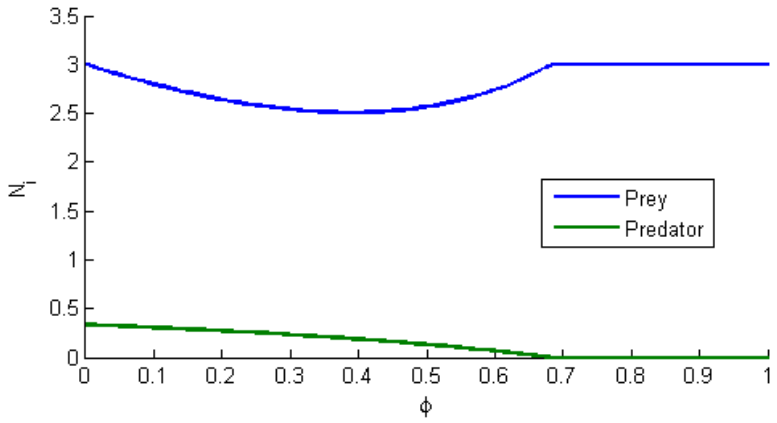

Figure 12: System with trade-off between birth rate and consumption rate without the pathogen. Parameter values used here are $\mu_1 = 0.2$, $\mu_2 = 0.1$, $\rho_1 = 0.25$, $\rho_2 = 0.25$, $k_1 = 0.0744$, $k_2 = 2.7489$, $e = 0.1$ and $\Phi = 0.1$. $\phi_{c,predator}$ can be seen to be around 0.68.

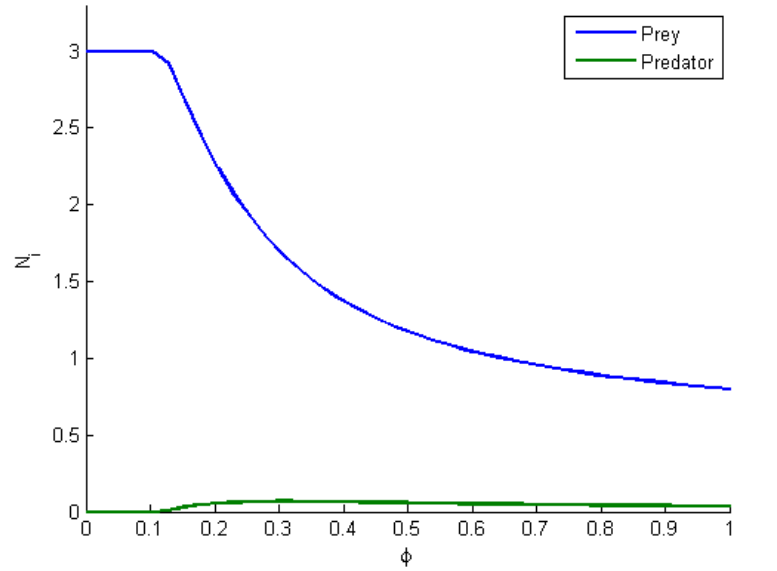

Figure 13: A system with trade-off between birth rate and consumption rate without the pathogen. The parameter values used here are $\mu_1 = 0.2$, $\mu_2 = 0.4$, $\rho_1 = 0.25$, $\rho_2 = 0.25$, $k_1 = 0.0744$, $k_2 = 2.7489$, $e = 0.5$ and $\Phi = 1$. $\phi_{c,predator}$ can be seen to be around 0.12.

Figure 13 shows such a situation. The predator population exists only for $\phi > \phi_{c,predator} = 0.12$ since the alternative food source is not enough to keep the predator alive.

Prey can go extinct in this system, too. Sadly, solving $\phi_{c,prey}$ for this is impossible analytically. $\phi_{c,prey}$is implicitly determined by the equation

$$\phi_{c,prey} = \frac{v_1(0) - \mu_1}{\Phi v_2^{-1}(\frac{\mu_2}{1-\phi_{c,prey}})}. \tag{83}$$

Now equation (83) can have an even number of solutions in interval $[0, 1]$ depending on parameters and birth rate functions. Usually this means zero or two solutions. The equation can not have only one solution because with $\phi = 0$ we have a positive equilibrium value for $\overline{N}_1 = v_1^{-1}(\mu_1)$. Similarly on the other end of the interval we have $\overline{N}_1 = \frac{\mu_2}{e\Phi}$, which is positive. The solutions to (83) occur when the equilibrium value reaches zero and goes beneath it. Because both end points are positive, the zero must be crossed an even number of times. The exception to this rule is iif the root is a double root. In this case the zero is not crossed and the prey would go extinct in only this one point. Because birth-rate functions are well behaving and non-increasing we usually have two solutions at most.

Figure 14 shows a situation with three different ϕ_c values. The prey goes extinct with $0.23 < \phi < 0.35$ and the predator with $\phi > 0.83$.

Without the trade-off it was clear that both populations could not go extinct at the same consumption rate. Now it is not that trivial because of the multiple critical values, but from the equation (80a) for the prey we can see that in order for that to happen, we should have $v_1(0) - \mu_1 = 0$ because at the critical consumption rate there are no predators consuming the prey and thus all the deaths must come from natural causes. This, of course, does not hold and thus critical consumption rates are always different for the prey and the predator.

10.3 Evolution without pathogen

Evolution without the pathogen can be solved with the tools of adaptive dynamics. Fitness of an invader in a population in an equilibrium is

$$s_\phi(\phi_{mut}) = (1 - \phi_{mut})v_2(\overline{N}_2) + \phi_{mut}e\Phi\overline{N}_1 - \mu_2. \tag{84}$$

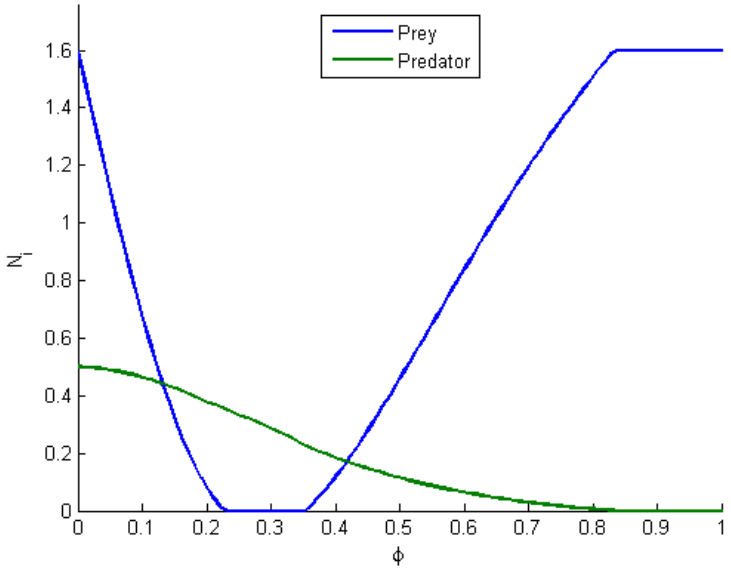

Figure 14: A system with trade-off between birth rate and consumption rate without the pathogen. The parameter values used here are $\mu_1 = 0.2$, $\mu_2 = 0.1$, $\rho_1 = 0.25$, $\rho_2 = 0.5$, $V_1 = 1$, $V_2 = 0.2$, $c_1 = 0.5$, $c_2 = 0.2$, $e = 0.005$ and $\Phi = 10$. For the extinction of the predator $\phi_{c,predator} = 0.83$ and for the prey $\phi_{c,prey} = 0.23$ and $\phi_{c,prey} = 0.35$.

We can present this with the help of change of variables. Let $x = \phi_{mut} - \phi$. Then fitness reduces to

$$s_\phi(\phi_{mut}) = (1-\phi-x)v_2(\overline{N}_2)+(\phi+x)e\Phi\overline{N}_1-\mu_2 = x[e\Phi\overline{N}_1-v_2(\overline{N}_2)]. \quad (85)$$

We can define the deciding factor, which is the selection gradient

$$A := e\Phi\overline{N}_1 - v_2(\overline{N}_2). \quad (86)$$

We can now represent this in another way. Solving $v_2(\overline{N}_2)$ from the equilibrium equation for the predator we get $v_2(\overline{N}_2) = \frac{\mu_2 - e\phi\Phi\overline{N}_1}{1-\phi}$. Inserting this to (86) and multiplying both sides with $(1-\phi)$ we get

$$(1-\phi)A = e\Phi\overline{N}_1 - \mu_2. \quad (87)$$

Now, this is maximized when N_1 achieves its maximum. N_1 is maximized when $\phi = 0$ and then $N_1 = v_1^{-1}(\mu_1)$. Now we have $A = e\Phi v_1^{-1}(\mu_1) - \mu_2$.

Earlier we saw that the critical consumption rate exists for the predator and is between 0 and 1 if we have $v_2(0) > \mu_2 > e\Phi v_1^{-1}(\mu_1)$. In this case the maximum of A is negative, which means that A is always negative. We can then make the conclusion that if the critical consumption rate exists between zero and one and the predator survives with the alternative food source, the evolution drives the consumption rate to zero.

Figure 14 shows an example of this situation. The predator cannot survive with prey only and the evolutionary drive is to $\phi = 0$. However, if the starting value is above $\phi = 0.35$, the predator will drive the prey extinct as the trait value lowers. After that, the trait value would keep decreasing, because there would not be any prey. With values lower than $\phi = 0.23$, this would not happen.

The other case for the existence of the critical consumption rate of the predator was when we had $e\Phi v_1^{-1}(\mu_1) > \mu_2 > v_2(0)$.

From the equilibrium equations we can solve

$$e\phi\Phi\overline{N}_1 = \mu_2 - (1-\phi)v_2(\overline{N}_2) \tag{88}$$

Multiplying (87) by ϕ we get

$$\phi(1-\phi)A = e\phi\Phi\overline{N}_1 - \phi\mu_2. \tag{89}$$

Now subsituting from (88) gives

$$\phi(1-\phi)A = e\phi\Phi\overline{N}_1 - \phi\mu_2 = (1-\phi)\mu_2 - (1-\phi)v_2(\overline{N}_2). \tag{90}$$

Finally we can show this as

$$\phi A = \mu_2 - v_2(\overline{N}_2) \tag{91}$$

This is just another way to represent the selection gradient A with no additional assumptions yet. We want to show that with the assumptions $e\Phi v_1^{-1}(\mu_1) > \mu_2 > v_2(0)$ we have always $A > 0$. ϕA is minimized when v_2 is maximized, which happens when $\overline{N}_2$ is the smallest it can be. This happens with $\overline{N}_2 = 0$ and we would have $\phi A = \mu_2 - v_2(0)$. Now we see

from our assumptions that this is positive since $\mu_2 > v_2(0)$. Therefore A is positive at every point with these assumptions. In this case ϕ is driven to one.

If ϕ_c does not exist in the interval of $[0, 1]$, we can have more interesting results. With critical consumption rate values, we had only evolution to the extreme. A drive to the extreme is still possible in the absence of critical consumption rates, but we can have a singular strategy in the middle of the interval, too.

10.3.1 The singular strategy

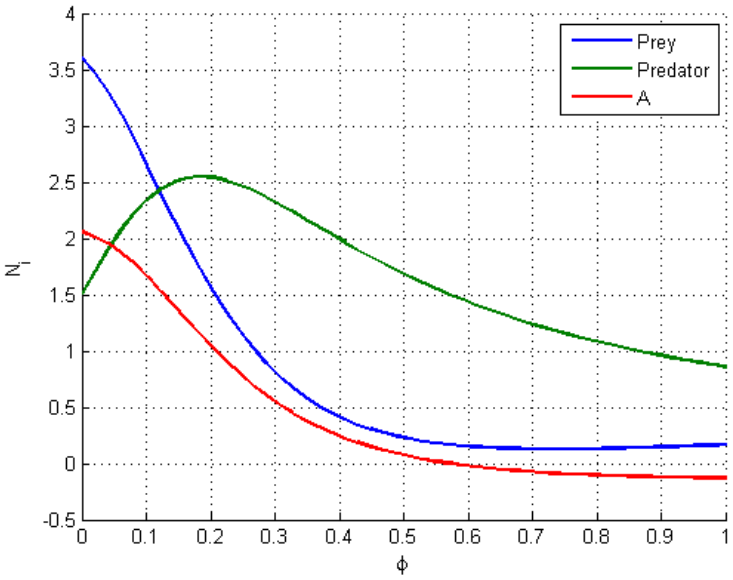

Figure 15: A system with trade-off between birth rate and consumption rate without the pathogen. The parameter values used here are $\mu_1 = 0.2$, $\mu_2 = 0.1$, $V_1 = 2$, $V_2 = 0.4$, $c_1 = 0.5$, $c_2 = 0.2$, $e = 0.3$ and $\Phi = 2$. The singular strategy in this case is $\phi = 0.57$.

We can solve the value ϕ of a singular strategy by solving equations $\dot{N}_1 = 0$, $\dot{N}_2 = 0$ and $A = 0$ when $N_1, N_2 > 0$. If $N_1 = 0$, we would have $A < 0$ and the predator should concentrate all its efforts to the alternative food source.

By solving the equations we get

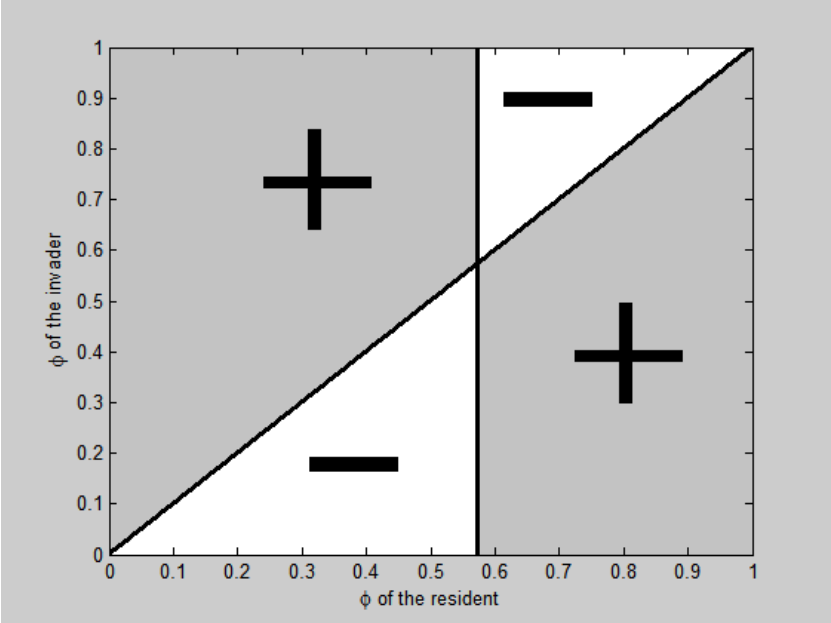

Figure 16: The pairwise invadability plot associated with figure 15. For ϕ smaller than the singular value of 0.57, any invader with a higher ϕ can invade and for values higher than 0.57, any invader with a lower value can invade. A trait value of 0.57 is a borderline case of an evolutionarily stable strategy, since any other strategy has fitness equal to zero against it.

$$N_1 = \frac{\mu_2}{e\Phi} \tag{92a}$$

$$N_2 = v_2^{-1}(\mu_2) \tag{92b}$$

$$\phi = \frac{v_1(\frac{\mu_2}{e\Phi}) - \mu_1}{\Phi v_2^{-1}(\mu_2)} \tag{92c}$$

From (92c) we can easily see when a singular strategy exists with $\phi \in [0, 1]$. We must have

$$\Phi v_2^{-1}(\mu_2) > v_1(\frac{\mu_2}{e\Phi}) - \mu_1 > 0. \tag{93}$$

Earlier we had the equation (82) for $\phi_{c,predator}$ of the predator. We showed that if a critical consumption rate for the predator $\phi_{c,predator}$ exists, no singular strategy exists. Equation (82) then provides us with necessary conditions for the existence of a singular strategy. These are

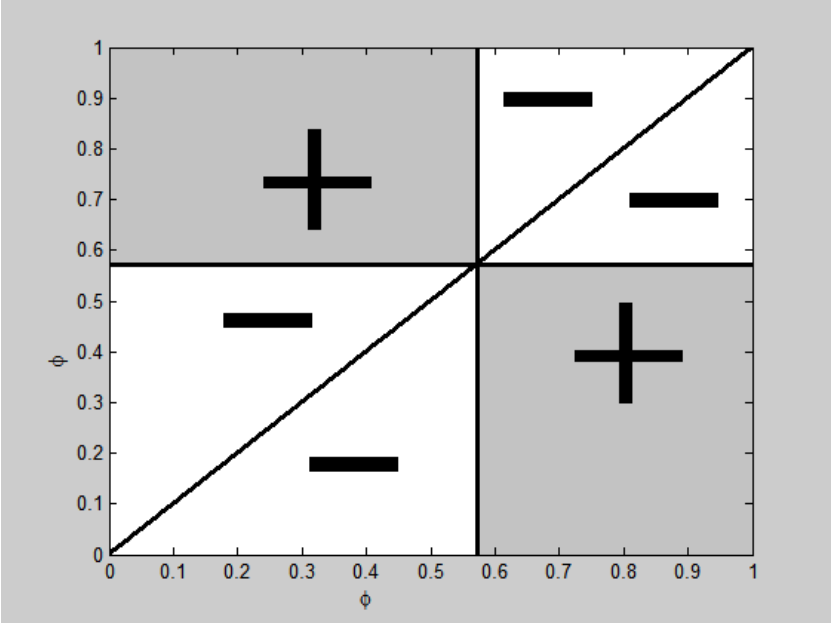

Figure 17: The mutual invadability plot associated with figures 15 and 16. The system has areas of dimorphism where two different predator populations can coexist with different trait values. These areas are marked with plus signs, whereas areas of no coexistance are marked with minus signs.

$$v_2(0) > \mu_2 \tag{94a}$$
$$e\Phi v_1^{-1}(\mu_1) > \mu_2 \tag{94b}$$

We can alternatively derive (94a) by setting the first term of (93) to greater than zero. Same way (94b) is derived by setting the second term of (93) to greater than zero. This leaves us the first inequality of (93) unused. Adding this to conditions (94) gives us sufficient conditions (the whole (93)) for the existence of a singular value.

Figure 15 shows the equilibrium densities for both species as functions of ϕ. With this information the values for A can be calculated and the root determined. It can be seen that the singular value is $\phi = 0.57$. With the help of the sign of A we can draw a pairwise indadability plot. This is shown in figure 16. It can be seen that with small mutation steps the trait ϕ is driven to the singular value. Figure 17 shows the areas of coexistence for two strategies. One example of such strategies could be $\phi_1 = 0.5, \phi_2 = 0.6$ where stable population sizes would be for prey $N_1 = 0.167$ and for predators

$N_{2,1} = 0.4167$ and $N_{2,2} = 1.0833$.

As before, the singular strategy is a borderline ESS without the pathogen because the invasion fitness is a linear function of ϕ_{mut}. The convergence stability of the singular strategy is then defined by (71). In this case the singular strategy is convergence stable.

10.4 Evolution with pathogen

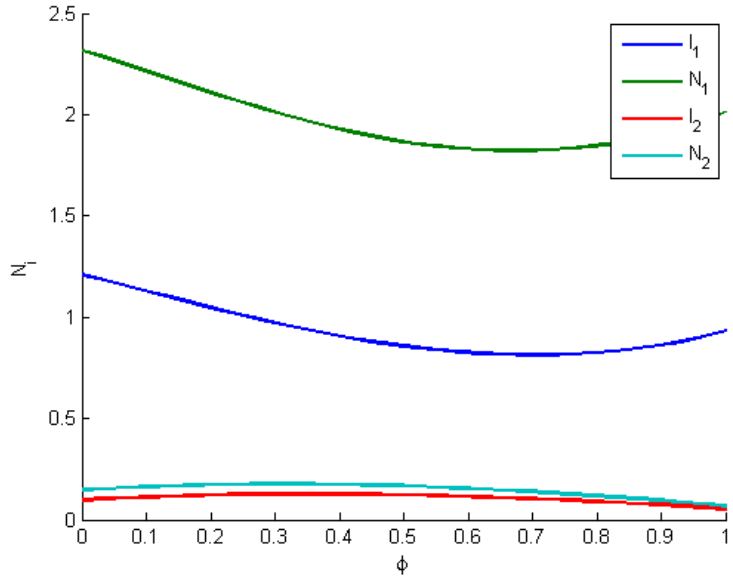

Figure 18: The system with trade-off between birth rate and consumption rate with pathogen. The equilibrium values for both species as a function of ϕ. Parameters used are $\mu_1 = 0.2$, $\mu_2 = 0.1$, $\alpha_1 = 0.02$, $\alpha_2 = 0.1$, $\beta_1 = 0.209$, $\beta_2 = 0.6$, $\rho_1 = 0.25$, $\rho_2 = 0.25$, $k_1 = 0.0744$, $k_2 = 2.7489$, $\Phi = 0.3$, $\kappa = 0.1$, $e = q = r = 1$, $c = 0.9$ and $p = 0.1$. The singular strategy in this case is $\phi = 0.57$

The evolution without pathogen was fairly simple. Dimorphism was possible, but achieving it through evolution not. Pathogen offers us with more possibilities as was shown in the situation without a tradeoff. In situations without the pathogen, the evolutionary view is always similar.

The invasion fitness must be again calculated from the determinant of the

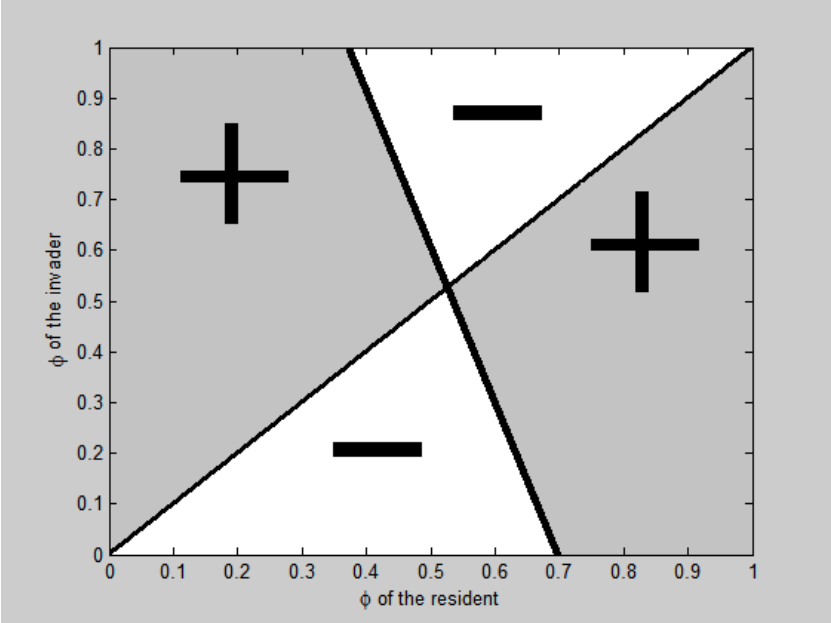

Figure 19: The pairwise invadability plot associated with figure 18. In this case the singular strategy is a real ESS. If $\phi = 0.53$ no other strategy can invade the population.

linearized system. The matrix is now

$$A = \begin{pmatrix} a_m & b_m \\ c_m & d_m \end{pmatrix}. \tag{95}$$

where

- $a_m = (1-\phi_{mut})v_2(\overline{N}_2) + e\phi_{mut}\Phi(\overline{S}_1 + q\overline{I}_1) - \mu_2 - cq\phi_{mut}\Phi\overline{I}_1 - \frac{\beta_2\overline{I}_2}{\overline{N}_2}$
- $b_m = (1-\phi_{mut})v_2(\overline{N}_2) + ep\phi_{mut}\Phi(\overline{S}_1 + q\overline{I}_1)$
- $c_m = cq\phi_{mut}\Phi\overline{I}_1 + \frac{\beta_2\overline{I}_2}{\overline{N}_2}$
- $d_m = -\alpha_2 - \mu_2$

Figure 18 shows the equilibrium values for both species as a function of ϕ with chosen parameters. The pairwise invadability plot of this situation is shown in figure 19. We can see that the singular trait value is an ESS. Evolution drives the value to $\phi = 0.53$ and after that it stays there.

Studying dimorphic populations in this case is not necessary, because they cannot become to existence.

11 Trade-off between consumption rate and death rate

Hunting the prey is sometimes a dangerous sport. It would be reasonable to think that hunting causes some extra deaths to predators. We can model this dependecy of death rate from time used to hunt the prey with a function $f(\phi)N_2$. Total amount of non-disease related deaths for the predator would in this case be $(\mu_2+f(\phi))N_2$. We assume that $f(0) = 0$. If we had $f(0) = C > 0$ we could include this in the natural death rate μ_2.

This gives us the following system

$$\dot{N}_1 = v_1(N_1)N_1 - \mu_1 N_1 - \alpha_1 I_1 - \phi(S_1 + qI_1)(S_2 + pI_2) \tag{96a}$$
$$\dot{N}_2 = v_2(N_2)N_2 - (\mu_2 + f(\phi))N_2 - \alpha_2 I_2 + e\phi(S_1 + qI_1)(S_2 + pI_2) \tag{96b}$$
$$\dot{I}_1 = \beta_1 \frac{S_1 I_1}{N_1} - (\mu_1 + \alpha_1)I_1 - \phi q I_1(S_2 + pI_2) + \kappa S_1(I_1 + rI_2) \tag{96c}$$
$$\dot{I}_2 = \beta_2 \frac{S_2 I_2}{N_2} - (\mu_2 + f(\phi) + \alpha_2)I_2 + cq\phi I_1 S_2. \tag{96d}$$

In the absence of the pathogen the system reduces to

$$\dot{N}_1 = v_1(N_1)N_1 - \mu_1 N_1 - \phi N_1 N_2 \tag{97a}$$
$$\dot{N}_2 = v_2(N_2)N_2 - (\mu_2 + f(\phi))N_2 + e\phi N_1 N_2 \tag{97b}$$

A simple choice is $f(\phi) = k\phi$. This means that hunting has a constant death rate cost to the predator. Other choices for the function are possible too. One could argue that more the predator hunts, the more skilled it becomes in hunting and thus the increase in additional deaths should lower with ϕ. This would mean that we have $f''(\phi) < 0$. On the other hand hunting can be exhausting and more hunting would make the predator tired causing the increase in the additional deaths to be higher with higher values of ϕ. This would mean that $f''(\phi) > 0$. Both approaches could be true and the sign of $f''(\phi)$ could at first be negative when the predator learns to hunt better and with higher values positive when fatigue plays bigger role.

11.1 Next-generation matrix

The next-generation matrix for this model is only a little different from the previous. The transition matrix T stays the same and extra death rate is added to the transmission matrix Σ. We get

$$K = -T\Sigma^{-1} = \begin{pmatrix} \frac{\kappa N_1+\beta_1}{q\phi N_2+\alpha_1+\mu_1} & \frac{r\kappa N_1}{\alpha_2+\mu_2+f(\phi)} \\ \frac{cq\phi N_2}{q\phi N_2+\alpha_1+\mu_1} & \frac{\beta_2}{\alpha_2+\mu_2+f(\phi)} \end{pmatrix} \tag{98}$$

The basic reproduction numbers are different from previous but with ecological isolation they are identical. We see that if there is no additional death rate, the next-generation matrix is equal to (16)

11.2 Critical consumption rate

Like before we can solve the critical consumption rate for the extinction of the prey and the predator when the pathogen is not present. For the prey we get

$$\phi_{c,prey} = \frac{v_1(0) - \mu_1}{v_2^{-1}(\mu_2 + f(\phi_{c,prey}))}. \tag{99}$$

The number of solutions depends on the characteristics of $f(\phi)$. No analytical solution is available unless $f(\phi)$ is a constant in which case it should be included in μ_2 and the trade-off doesn't really exist.

For predator we have

$$\phi_{c,predator} = \frac{f(\phi_{c,predator}) - v_2(0) + \mu_2}{ev_1^{-1}(\mu_1)}. \tag{100}$$

With $f(\phi) = k\phi$ this solves to

$$\phi_{c,predator} = \frac{v_2(0) + \mu_2}{k - ev_1^{-1}(\mu_1)}. \tag{101}$$

Without the trade-off it was clear that the both populations could not go extinct at the same consumption rate. Again from the equation for the prey we can see that in order for that to happen, we should have $v_1(0) - \mu_1 = 0$.

This of course does not hold and thus critical consumption rates are always different for the prey and the predator.

11.3 Evolution without pathogen

Without the pathogen the initial growth rate of a mutant is determined by

$$\frac{dM}{dt} = (v_2(\overline{N}_2) - \mu_2 - f(\phi_{mut}) + e\phi_{mut}\overline{N}_1)M. \tag{102}$$

Fitness is described by the factor multiplying M. With change of variables $x = \phi_{mut} - \phi$ the fitness reduces to

$$s_\phi(\phi_{mut}) = x(e\overline{N}_1 - \frac{f(\phi + x) - f(\phi)}{x}) := xA \tag{103}$$

Taking the limit $x \to 0$ we see that the last term of A goes to $f'(\phi)$. We then get

$$A = e\overline{N}_1 - f'(\phi). \tag{104}$$

We must remember when taking the limit that adaptive dynamics has discrete and finite steps in trait values. In this sense the limit is too precise and we must remember where it comes from.

11.3.1 Singular strategy

Without the pathogen singular strategies are solvable to some extent. The singular strategy can be solved similarly to the previous cases. From (97a) we can solve ϕ in the equilibrium

$$\phi = \frac{v_1(\overline{N}_1) - \mu_1}{\overline{N}_2}. \tag{105}$$

From equations (97b) and (104) we get values for $\overline{N}_1$ and $\overline{N}_2$. The equation becomes

$$\phi = \frac{v_1(\frac{f'(\phi)}{e}) - \mu_1}{v_2^{-1}(f(\phi) - \phi f'(\phi) + \mu_2)}. \tag{106}$$

With $f(\phi) = k\phi$ we have $f(\phi) = \phi f'(\phi)$. This simplifies the equation giving us an analytical solution

$$\phi = \frac{v_1(\frac{k}{e}) - \mu_1}{v_2^{-1}(\mu_2)}. \tag{107}$$

Now, this positive whenever $v_1(\frac{k}{e}) > \mu_1$ and $v_2(0) > \mu_2$.

We can again use the determinant of the linearized system to study the properties of the singular strategy. Given that the linearized system has A equal to (75) subtracted by $f(\phi_{mut})I$, where I is the identity matrix, we can say whether the singular strategy is an ESS, or not. The formula for the general case is complex, but in the absence of the pathogen by differentiating the fitness proxy we get from (104)

$$\frac{\partial^2 s_\phi(\phi_{mut})}{\partial \phi_{mut}^2} = -f''(\phi_{mut}). \tag{108}$$

This could be positive or negative. However with the choice of $f(\phi) = k\phi$ we have $f'(\phi) = k$ and $f''(\phi) = 0$ and therefore

$$\frac{\partial^2 s_\phi(\phi_{mut})}{\partial \phi_{mut}^2} = 0. \tag{109}$$

This means that with a linear trade-off in the death rate we always have a borderline ESS. This is seen in figure 21.

Choosing, for example, $f(\phi) = k\phi^z$, where z is not equal to one, we can get singular strategies that are ESS or not. Equation (108) is a simple condition for the singular strategy being an ESS. We can see that the singular strategy is an ESS if and only if the trade-off function in death rate is convex at the current value of ϕ.

Without the pathogen the course of evolution is decided by the factor A. When $f(\phi) \equiv 0$ also $f'(\phi) = 0$. This would mean that hunting causes no extra death rate, simplifying the situation to that of the earlier where ϕ is always increasing.

Assuming $f(\phi) = k\phi$ the invasion fitness is

$$A = e\overline{N}_1 - k \tag{110}$$

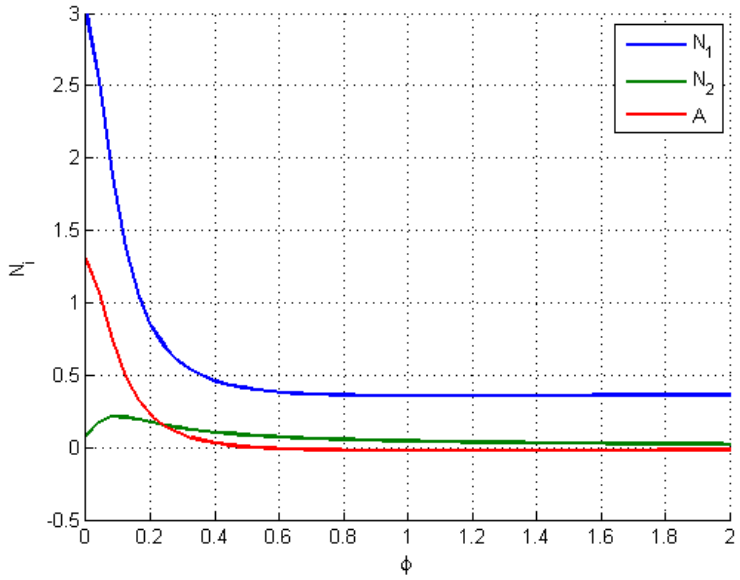

Figure 20: The system with trade-off between birth rate and death rate without the pathogen. Equilibrium values for the predator and the prey and the invasion gradient. Parameter values used are $\mu_1 = 0.2$, $\mu_2 = 0.2$, $\rho_1 = 0.25$, $\rho_2 = 0.25$, $k_1 = 0.0744$, $k_2 = 2.7489$, $e = 0.5$ and $k = 0.2$.

Figure 20 shows an example of evolution of ϕ in the absence of the pathogen. The invasion gradient is first positive and changes to negative at the singular strategy $\phi = 0.53$. The singular strategy is convergence stable like before. The pairwise invadability plot is shown in figure 21. The convergence stability can be seen from the fact that with values lower than $\phi = 0.53$ the plus region is above the $y = x$ line and with values higher than $\phi = 0.53$ the plus region is below the line. Evolution is towards the singular strategy $\phi = 0.53$, which is a borderline ESS. The effect of the trade-off is very similar to the trade-off in the consumption rate and the birth rate in the absence of the pathogen.

11.4 Evolution with pathogen

As usual, evolution with pathogen offers more diverse situations. Introduction of the pathogen in this model makes it possible to have singular strategies that are not convergence stable. The invasion fitness must again be calculated from the determinant of the linearized system. The matrix is now

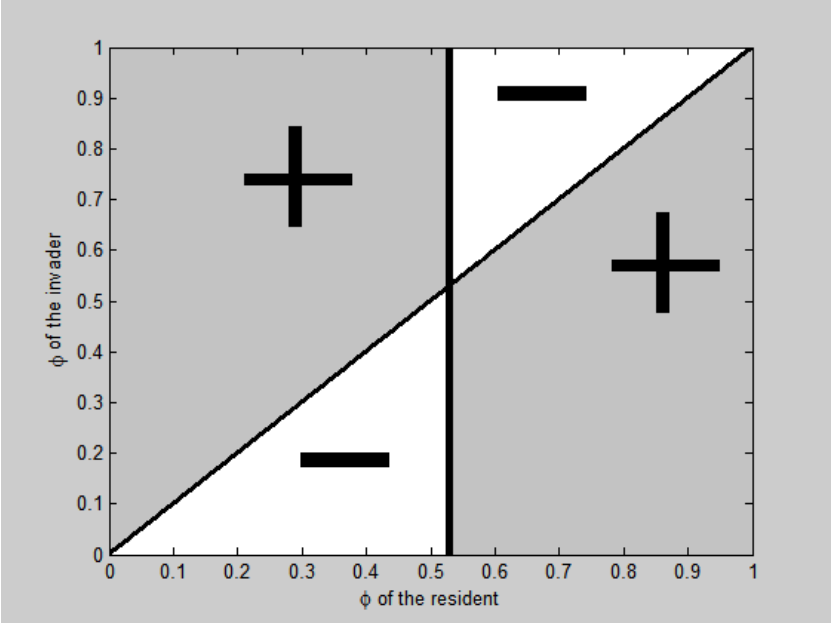

Figure 21: The pairwise invadability plot associated with figure 20. The parameters used are $\mu_1 = 0.2$, $\mu_2 = 0.2$, $\rho_1 = 0.25$, $\rho_2 = 0.25$, $k_1 = 0.0744$, $k_2 = 2.7489$, $e = 0.5$ and $k = 0.2$.

simply equal to (75) subtracted by $f(\phi_{mut})I$ where I is the identity matrix.

$$A = \begin{pmatrix} a_m & b_m \\ c_m & d_m \end{pmatrix}. \tag{111}$$

where

- $a_m = v_2(\overline{N}_2) + e\phi_{mut}(\overline{S}_1 + q\overline{I}_1) - \mu_2 - cq\phi_{mut}\overline{I}_1 - \frac{\beta_2\overline{I}_2}{\overline{N}_2} - f(\phi)$
- $b_m = v_2(\overline{N}_2) + ep\phi_{mut}(\overline{S}_1 + q\overline{I}_1)$
- $c_m = cq\phi_{mut}\overline{I}_1 + \frac{\beta_2\overline{I}_2}{\overline{N}_2}$
- $d_m = -\alpha_2 - \mu_2 - f(\phi)$

Figure 22 shows the equilibrium values for the predator and the prey with parameters much like in figure 10. Adjusting parameters a little and adding the added death rate with parameter $k = 0.11$ the pairwise invadability plot gets a very different shape. This is shown in figure 23.

We now have two singular strategies. One is convergence stable and one is not. From figure 22 we can see that if the consumption rate is greater than

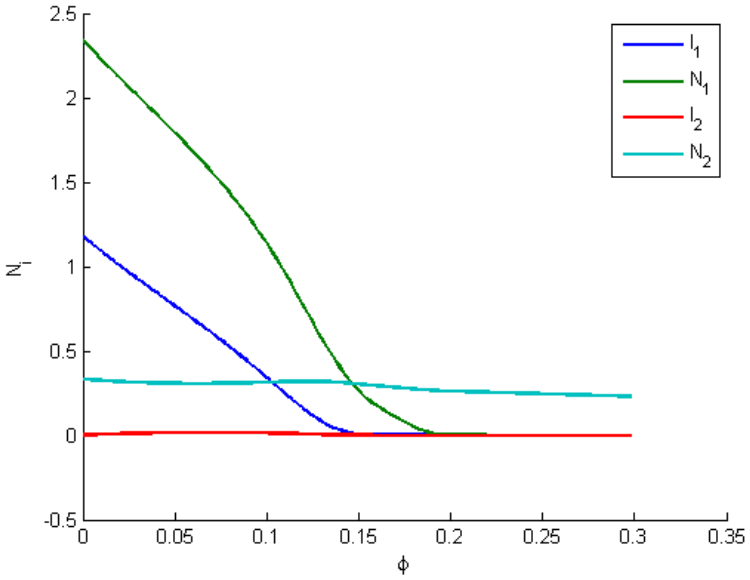

Figure 22: The equilibrium values for the predator and the prey with parameters $\mu_1 = 0.2$, $\mu_2 = 0.1$, $\rho_1 = 0.25$, $\rho_2 = 0.25$, $k_1 = 0.0744$, $k_2 = 2.7489$, $\alpha_1 = 0.02$, $\alpha_2 = 0.3$, $\beta_1 = 0.209$, $\beta_2 = 0.2$, $e = 0.21$, $k = 0.11$, $c = 0.95$, $\kappa = 0.1$, $p = 0.05$, $q = 0.3$ and $r = 1$.

0.15 the sick prey is hunted to extinction and at 0.19 the healthy prey, too. Fortunately for the prey this will not happen, since the evolutionary drive decreases the consumption. $\phi = 0.12$ is a singular value for the consumption rate. This is also an ESS and convergence stable. $\phi = 0.07$ is not convergence stable and population evolves away from it. $\phi = 0.07$ is not convergence stable, but it is an ESS. The third interesting value is $\phi = 0$, which is convergence stable from above. We can conclude that if the original value for the consumption is below 0.07, the final value is $\phi = 0$ with no predation, and if the original value for consumption is above 0.07, the final value is $\phi = 0.12$.

All in all, figure 22 shows the combined effect of the pathogen driving the consumption down, because the predator is better off not getting sick from the prey and the effect of the trade-off in the death rate. The pairwise invadability plot is a combination of figures 11 and 21. The additional death rate moves the first singular strategy down and introduces the second singular strategy. The presence of the pathogen is what makes the strategies ESS.

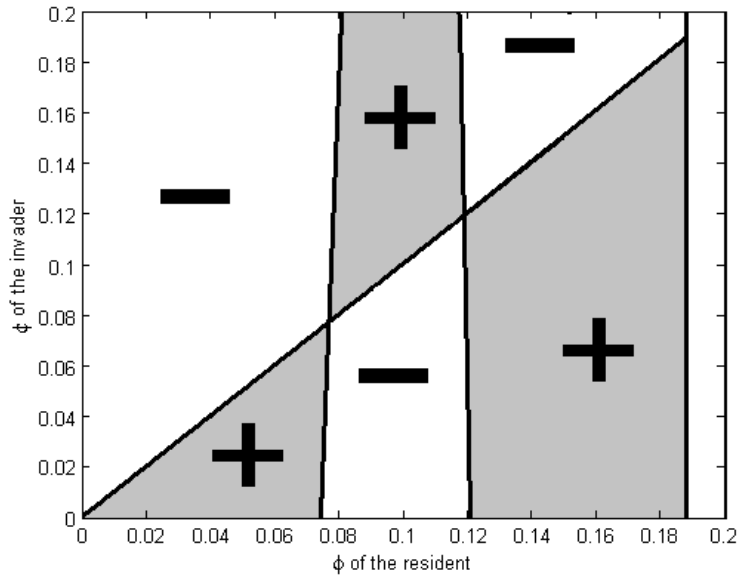

Figure 23: The pairwise invadability plot associated with figure 22. The parameters used are $\mu_1 = 0.2$, $\mu_2 = 0.1$, $\rho_1 = 0.25$, $\rho_2 = 0.25$, $k_1 = 0.0744$, $k_2 = 2.7489$, $\alpha_1 = 0.02$, $\alpha_2 = 0.3$, $\beta_1 = 0.209$, $\beta_2 = 0.2$, $e = 0.21$, $k = 0.11$, $c = 0.95$, $\kappa = 0.1$, $p = 0.05$, $q = 0.3$ and $r = 1$.

12 Trade-off between consumption rate, birth rate and death rate

The previous two trade-offs can be combined to one. This gives us the following system

$$\dot{N}_1 = v_1(N_1)N_1 - \mu_1 N_1 - \alpha_1 I_1 - \phi\Phi(S_1 + qI_1)(S_2 + pI_2) \tag{112a}$$

$$\dot{N}_2 = (1-\phi)v_2(N_2)N_2 - (\mu_2 + f(\phi))N_2 - \alpha_2 I_2 + e\phi\Phi(S_1 + qI_1)(S_2 + pI_2) \tag{112b}$$

$$\dot{I}_1 = \beta_1 \frac{S_1 I_1}{N_1} - (\mu_1 + \alpha_1)I_1 - \phi\Phi q I_1(S_2 + pI_2) + \kappa S_1(I_1 + rI_2) \tag{112c}$$

$$\dot{I}_2 = \beta_2 \frac{S_2 I_2}{N_2} - (\mu_2 + f(\phi) + \alpha_2)I_2 + cq\phi\Phi I_1 S_2. \tag{112d}$$

Here Φ is again a constant describing the effect of the consumption at its maximum and $\phi \in [0, 1]$ is the proportion of time predator uses to hunt the

prey. Without the pathogen this system reduces to

$$\dot{N_1} = v_1(N_1)N_1 - \mu_1 N_1 - \phi\Phi N_1 N_2 \tag{113a}$$
$$\dot{N_2} = (1-\phi)v_2(N_2)N_2 - (\mu_2 + f(\phi))N_2 + e\phi\Phi N_1 N_2 \tag{113b}$$

12.1 Next-generation matrix

The next-generation matrix for this model is only a little different from the previous. The transition matrix T stays the same and the transmission matrix Σ is a combination of what is seen in the case of single trade-offs. We get

$$K = -T\Sigma^{-1} = \begin{pmatrix} \frac{\kappa N_1 + \beta_1}{q\phi\Phi N_2 + \alpha_1 + \mu_1} & \frac{r\kappa N_1}{\alpha_2 + \mu_2 + f(\phi)} \\ \frac{cq\phi\Phi N_2}{q\phi\Phi N_2 + \alpha_1 + \mu_1} & \frac{\beta_2}{\alpha_2 + \mu_2 + f(\phi)} \end{pmatrix} \tag{114}$$

The basic reproduction numbers are different from previous but with ecological isolation they are identical. We see that if the strength of the consumption ($\phi \times \Phi$) is equal to the strength of the consumption without the trade-off, the next-generation matrix is equal to (98) and if there is no additional death rate, it is equal to (81).

12.2 Critical consumption rate

We can solve critical consumption rates for the prey and the predator at least numerically.

For the prey we have

$$\phi_c = \frac{v_1(0) - \mu_1}{\Phi v_2^{-1}(\frac{\mu_2 + f(\phi_c)}{1-\phi_c})}. \tag{115}$$

We can see that with $f(\phi) = 0$ this reduces to (83) as it should.

For the predator we have

$$\phi_c = \frac{v_2(0) - \mu_2}{v_2(0) + \frac{f(\phi)}{\phi} - e\Phi v_1^{-1}(\mu_1)}. \tag{116}$$

With $f(\phi) = k\phi$ we have an analytical solution for this

$$\phi_c = \frac{v_2(0) - \mu_2}{v_2(0) + k - e\Phi v_1^{-1}(\mu_1)}. \tag{117}$$

With $f(\phi) = 0$ the solution equals (82).

12.3 Evolution without pathogen

Without the pathogen the initial growth rate of a mutant is determined by

$$\frac{dM}{dt} = ((1 - \phi_{mut})v_2(\overline{N}_2) - \mu_2 - f(\phi_{mut}) + e\phi_{mut}\Phi\overline{N}_1)M. \tag{118}$$

Fitness is described by the factor multiplying M. With change of variables $x = \phi_{mut} - \phi$ the fitness reduces to

$$s_\phi(\phi_{mut}) = x(e\Phi\overline{N}_1 - v_2(\overline{N}_2) - \frac{f(\phi + x) - f(\phi)}{x}) := xA \tag{119}$$

Taking the limit $x \to 0$ we see that the last term of A goes to $f'(\phi)$. We then get

$$A = e\Phi\overline{N}_1 - v_2(\overline{N}_2) - f'(\phi). \tag{120}$$

Figure 24 shows the equilibrium values for the prey and the predator with chosen parameters. We have now chosen the additional death rate function to be of form $f(\phi) = k\phi^{4/5}$. This means that the addition in death rate becomes smaller as the predator hunts more. Both the prey and the predator have positive equilibria for every possible value of ϕ.

Figure 25 shows the pairwise invadability plot of the situation. One singular strategy exists approximately at $\phi = 0.5$. The singular strategy is convergence stable and thus the trait value tends to it. In this case the convergence stable strategy, however, is not an ESS. This means that the trait value acts as a branching point.

Figure 26 shows what happens at the singular strategy. First, evolution drives the trait value close to the branching point. Then a mutation occurs such that there exists two different traits from different sides of the branching

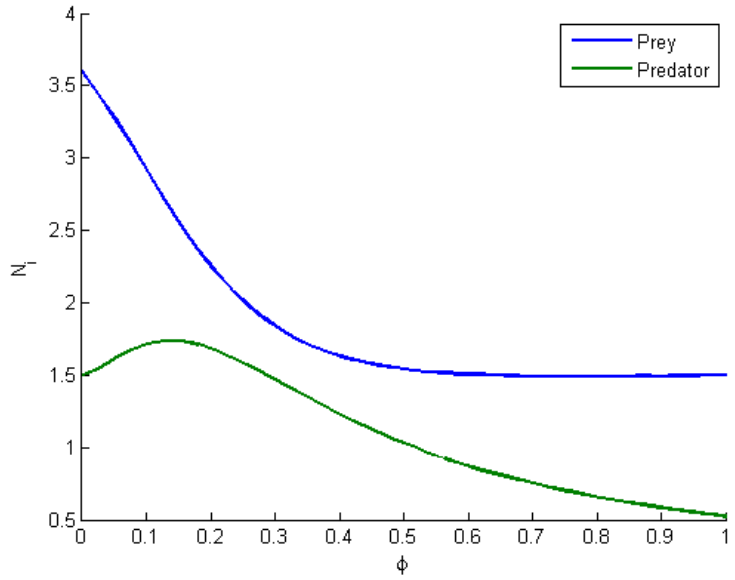

Figure 24: The equilibrium values for the predator and the prey with trade-off between the consumption rate, birth rate, and death rate. The parameters used are $\mu_1 = 0.2$, $\mu_2 = 0.1$, $V_1 = 2$, $V_2 = 0.4$, $c_1 = 0.5$, $c_2 = 0.2$, $e = 0.21$, $k = 0.8$ and $f(\phi) = k\phi^{4/5}$.

point. In this case neither of the strategies goes extinct creating a dimorphic population. Afterwards mutations occur in the dimorphic population changing the two resident trait values. The fitness of a mutant can be determined similarly to the situation with a monomorphic population. The evolution in the dimorphic population is simple. The strategies evolve away from each other and finally the result is one strategy with $\phi = 0$ and other with $\phi = 1$. This is an case of speciation. One species concentrates on feeding on the prey while the other uses the alternative food source.

The specialization of predators has been studied before. In one model a weakly concave trade-off was required for the possibility of brancing to exist.[37] In our case the trade-off in death rate was concave when the branching happened too.

To present the situation, a simulation was made with the same parameters as in figure 24. The simulation starts with only one type of predator. Equations (113) are used to simulate the dynamics of the predator-prey system and every once in a while a mutation close to one of the residents is introduced to the system. Figure 27 shows how the existing trait values change in time.

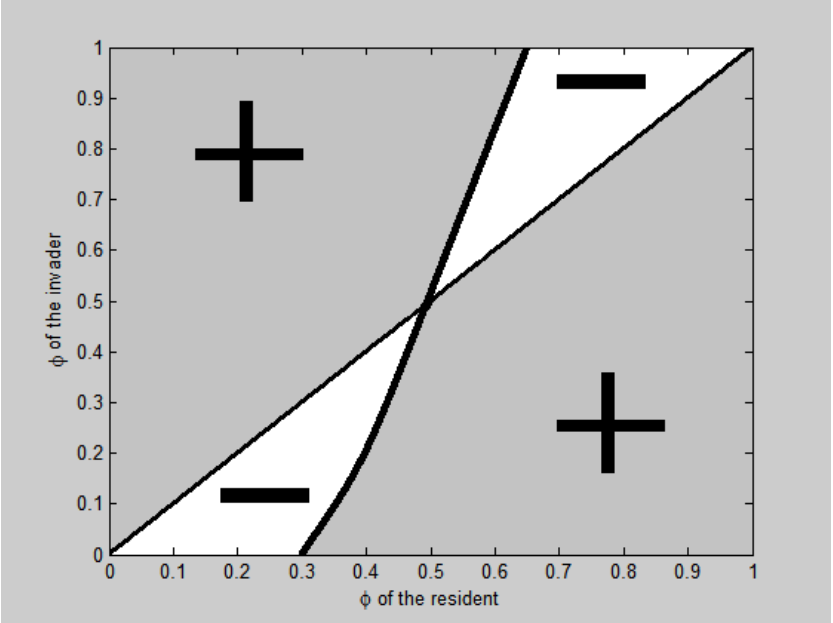

Figure 25: The pairwise invadability plot associated with figure 24. The parameters used are $\mu_1 = 0.2$, $\mu_2 = 0.1$, $V_1 = 2$, $V_2 = 0.4$, $c_1 = 0.5$, $c_2 = 0.2$, $e = 0.21$, $k = 0.8$ and $f(\phi) = k\phi^{4/5}$.

The trait value is $\phi = 0.1$ in the beginning. It evolves to $\phi = 0.5$, where branching occurs. In the end we have two traits, $\phi = 0$ and $\phi = 1$.

12.3.1 Singular strategy

Singular strategies can be solved by setting (113a), (113b) and (120) equal to zero. This system of equations cannot be solved analytically.

12.4 Evolution with pathogen

As usual, evolution with pathogen offers more diverse situations. Introduction of the pathogen in this model makes it possible to have singular strategies that are not convergence stable. The invasion fitness must be again calculated from the determinant of the linearized system. The matrix is now simply (95) subtracted by$f(\phi_{mut})I$ where I is the identity matrix.

$$A = \begin{pmatrix} a_m & b_m \\ c_m & d_m \end{pmatrix}. \tag{121}$$

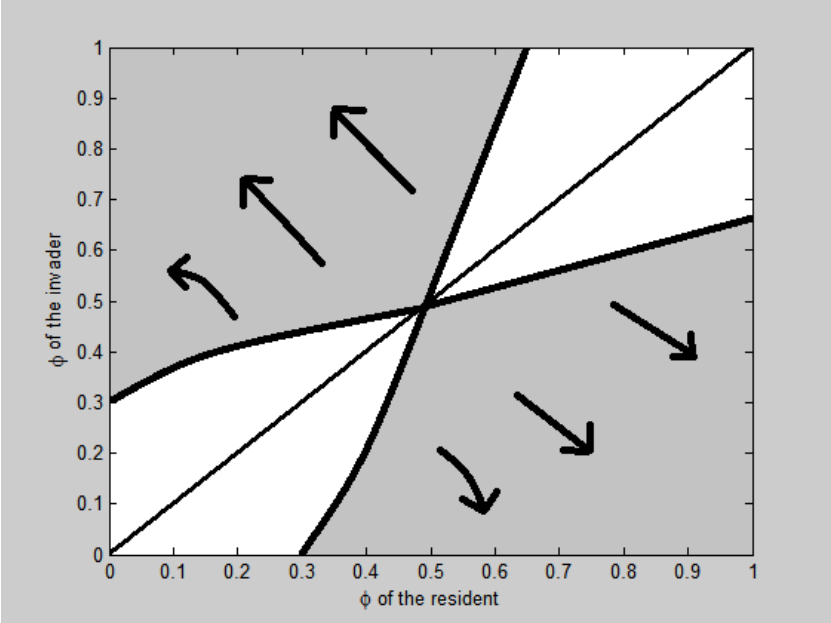

Figure 26: The mutual invadability plot associated with figures 24 and 25. The parameters used are $\mu_1 = 0.2$, $\mu_2 = 0.1$, $V_1 = 2$, $V_2 = 0.4$, $c_1 = 0.5$, $c_2 = 0.2$, $e = 0.21$, $k = 0.8$ and $f(\phi) = k\phi^{4/5}$. The vector field shows the direction of evolution.

where

- $a_m = (1-\phi_{mut})v_2(\overline{N}_2)+e\phi_{mut}\Phi(\overline{S}_1+q\overline{I}_1)-\mu_2-cq\phi_{mut}\Phi\overline{I}_1-\frac{\beta_2\overline{I}_2}{\overline{N}_2}-f(\phi)$
- $b_m = (1-\phi_{mut})v_2(\overline{N}_2)+ep\phi_{mut}\Phi(\overline{S}_1+q\overline{I}_1)$
- $c_m = cq\phi_{mut}\Phi\overline{I}_1+\frac{\beta_2\overline{I}_2}{\overline{N}_2}$
- $d_m = -\alpha_2-\mu_2-f(\phi)$

The addition of the pathogen has been shown previously. Without any trade-offs it created in some cases a singular strategy, which was an ESS and not convergence stable. With the trade-off in consumption rate and death rate it had the same effect. With trade-off in consumption rate and birth rate the effect was similar. The singular strategy became an ESS. This tendency to make singular strategies ESS can be observed when we have both trade-offs, too. We must keep in mind that this tendecy was observed in borderline ESS case only and should not be expected to generalize to situation with no ESS without further proof. In all of these cases we have had a harmful pathogen, which is usually the case.

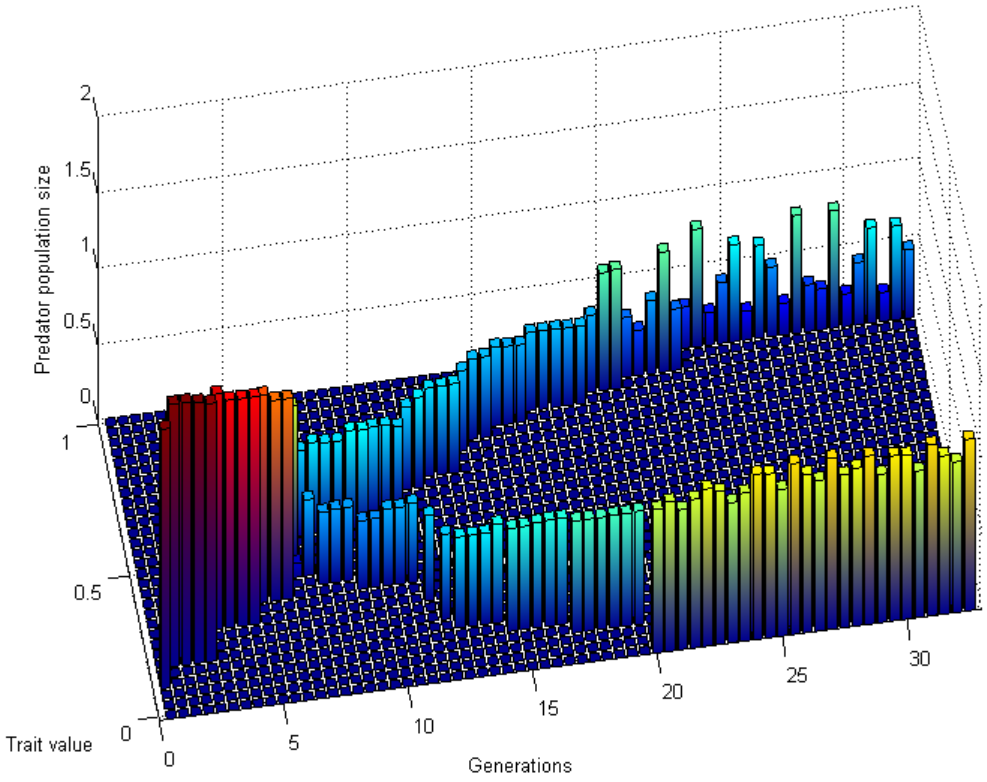

Figure 27: A simulation associated with figures 24, 25 and 26. The trait value first approaches $\phi = 0.5$ and after branching diverges to $\phi = 0$ and $\phi = 1$.

13 Birth rate functions and resource dynamics

Instead of birth rate functions the dynamics of the alternative food source can be dealt with resource-consumer dynamics. Generally consumer-resource dynamics can be represented as

$$\frac{dR}{dt} = v(R) - f(R, C), \tag{122}$$

where R is the resource and C the consumer. $v(R)$ describes how the resource is born in the absence of the consumer and $f(R, C)$ how it is consumed. A simple choice for the functions could be $v(R) = A$ and $f(R, C) = BRC$,

where A and B are constants. Generally the growth of the consumer could be a function $g(R, C)$, different from $f(R, C)$, but choosing $f(R, C) \propto g(R, C)$ is a natural choice.

Using this for our predator-prey system without trade-offs we get the following system

$$\dot{N}_1 = g_1(R_1, N_1) - \mu_1 N_1 - \alpha_1 I_1 - \phi(S_1 + qI_1)(S_2 + pI_2) \tag{123a}$$
$$\dot{N}_2 = g_2(R_2, N_2) - \mu_2 N_2 - \alpha_2 I_2 + e\phi(S_1 + qI_1)(S_2 + pI_2) \tag{123b}$$
$$\dot{I}_1 = \beta_1 \frac{S_1 I_1}{N_1} - \mu_1 I_1 - \alpha_1 I_1 - \phi q I_1(S_2 + pI_2) + \kappa S_1(I_1 + rI_2) \tag{123c}$$
$$\dot{I}_2 = \beta_2 \frac{S_2 I_2}{N_2} - \mu_2 I_2 - \alpha_2 I_2 + cq\phi I_1 S_2 \tag{123d}$$
$$\dot{R}_1 = v_1(R_1) - f_1(R, N_1) \tag{123e}$$
$$\dot{R}_2 = v_2(R_2) - f_2(R, N_2) \tag{123f}$$

It is assumed that healthy and sick predator and prey consume the resource at a similar rate.

This system is in a way a predator-prey model with two preys. In the eyes of the predator the resource acts like another prey. Models with multiple types of prey have been studied by Abrams [1], Fryxell [13] and Křivan [21] [23] [22].

13.1 Separation of time scales

We can analyze this system with the help of fast and slow dynamics. This is called separating the timescales. We assume that the resource R has considerably faster dynamics compared to the predator or the prey. This way we can assume that the resource always reaches its equilibrium associated with N_1 and N_2 and we can analyze the system in parts.

In biochemistry this method is called the quasi-steady-state approximation (QSSA). It is the standard practice in reducing the number of differential euqations in the system.[12] Perhaps the most widely known example is Briggs' and Haldanes work on Michaelis-Menten kinetics.[5]

First we calculate the equilibria for R_1 and R_2 from equation $v_i(\overline{R}_i) = f(\overline{R}_i, N_i)$. This equilibrium is now a function of N_i and can be inserted

back to equations (123a)-(123d). Resource then becomes invisible to the system and is only shown in the form of a birth-rate function.

If we choose the resource dynamics to be of the form of the logistic equation and the consuming to be linear we get

$$\frac{dR_i}{dt} = v_i(R_i) - f_i(R_i, N_i) = rR_i(1 - \frac{R_i}{K}) - \Gamma R_i N_i, \tag{124}$$

where K is the carrying capacity of the logistic equation, r is the initial growth rate and Γ describes the power of the consuming. We can scale the resource so that we get rid of Γ if we want to. The equilibrium for the resource solves now to

$$\overline{R}_i = K(1 - \frac{\Gamma}{r}N_i). \tag{125}$$

With a large enough N_i this could be negative. On the fast time scale this would lead to a trivial equilibrium of $R_i = 0$ for the resource giving the following results with a sort of a truncation. If the amount of the resource became zero, it would stay there forever. If we choose $g_i(R_i, N_i) = f_i(R_i, N_i) = \Gamma R_i N_i$ growth of the consumer due to resource is

$$\frac{dN_i}{dt} = \Gamma \overline{R}_i N_i = \Gamma K(1 - \frac{\Gamma}{r}N_i)N_i. \tag{126}$$

This is equal to having a birth-rate function $v_i(N_i) = \Gamma K - \frac{\Gamma^2 K}{r}N_i$. This is the linearly decreasing birth-rate function.

With the choice $v_i(R_i) = B_i - c_i R_i$ and $f_i(R_i, N_i) = g_i(R_i, N_i) = A_i R_i N_i$ for the resource we get $\overline{R}_i = \frac{B_i}{c_i + A_i N_i}$. For the consumer we get

$$\frac{dN_i}{dt} = A_i \overline{R}_i N_i = \frac{A_i B_i}{c_i + A_i N_i} N_i. \tag{127}$$

If $c_i = 0$, we get the constant births model with B_i births per time unit. With a positive c_i the birth rate would approach a constant value with large values of N_i anyway but near zero $c_i = 0$ would produce more realistic results with the birth rate going to zero when $N_i = 0$.

Exponentially decreasing birth-rate function can be formulated this way, too. We get it by choosing $v_i(R_i) = B_i$, $f_i(R_i, N_i) = e^{k_i N_i}$ and $g_i(R_i, N_i) =$

$A_i R_i N_i$. In this context this would be a dubious choice because the behaviour of f and g are fundamentally different. Such a difference is very hard to justify biologically. This doesn't mean that exponentially decreasing birth rate function is not biologically acceptable. It could arise from another kind of formulation.

Using the approximation above is a drastic matter. A more careful approach can be made with the help of singular approximation theory. The shortcomings of the approximation are shown in the following model. Lets say the following model describes the system fully.

$$\dot{N}_1 = a_1 R_1 N_1 - \mu_1 N_1 - \alpha_1 I_1 - \phi(S_1 + qI_1)(S_2 + pI_2) \tag{128a}$$
$$\dot{N}_2 = a_2 R_2 N_2 - \mu_2 N_2 - \alpha_2 I_2 + e\phi(S_1 + qI_1)(S_2 + pI_2) \tag{128b}$$
$$\dot{I}_1 = \beta_1 \frac{S_1 I_1}{N_1} - \mu_1 I_1 - \alpha_1 I_1 - \phi q I_1 (S_2 + pI_2) + \kappa S_1 (I_1 + rI_2) \tag{128c}$$
$$\dot{I}_2 = \beta_2 \frac{S_2 I_2}{N_2} - \mu_2 I_2 - \alpha_2 I_2 + cq\phi I_1 S_2 \tag{128d}$$
$$\dot{R}_1 = v_1 R_1 - b_1 N_1 R_1 \tag{128e}$$
$$\dot{R}_2 = v_2 R_2 - b_1 N_2 R_2 \tag{128f}$$

This is a real model with proper equilibrium. If we study this by separating time scales, what we get for the resource is exponential growth with rate $r = v_i - b_i N_i$ with no equilibrium unless $b_i = v_i N_i$. In this case the approximation would be completely wrong. In reality the system can have complex dynamics evolving away from an unstable equilibrium.

In light of this we can say that it is possible that the birth rate function we used has its basis in reality. Using a birth-rate function is much simpler for analysis, and keeping the whole resource-dynamics in place when it is of no interest would be useless.

14 Conclusions

This thesis studied a predator-prey model with a shared disease. In addition to the prey, the predator has an alternative food source, which it consumes. Both species are vulnerable to a pathogen that spreads not only from prey to prey or from predator to predator. Predators can get infected when consuming infected prey and prey can get infected from environmental transmission.

Properties of the model and its behaviour was studied. This includes the basic reproduction number, the next-generation matrix and the stability of equilibria. Factors affecting the properties of the equilibria are identified. Consumption rate, the rate at which the predator consumes the prey, is studied and critical consumption rate, where the prey or the predator population ceases to exist, is identified.

Evolution of the consumpion rate was studied within the adaptive dynamics framework. Without the presence of the pathogen the consumption rate increases indefinitely, but introducing the pathogen to the system can change the course of evolution and make a decrease in the consumption rate possible.

The evolution of the consumption rate was explored further with help of trade-offs. Taking into account that increase in consumption rate might be costly to the predator provides alternative outcomes. Two trade-offs that are studied are trade-off between consumption rate and the consumption of the alternative food source, and trade-off between consumption and death rate. These trade-offs can exist concurrently or independently.

Having both of the trade-offs in place we can identify situations where we have a branching point. In such a situation the consumption rate evolves first to this brancing point, where two different branches arise and disruptive evolution takes place. The end result is two different types of predator, one using only the alternative food source and one using only the prey as its source of food.

A lot is still left to explore. The model is easily modified to include multiple preys and predators. Different preys could spread different diseases to the predator. The consumption part of the model was simple and thus allowed for many analytical solutions. A more realistic model could represent the conversion factor e as a decreasing function of ϕ. The decrease would be natural if we accept that the predator uses the nutrients more efficiently when starving. The trade-off between consumption rate and birth rate was linear and based on very intuitive reasoning. In some situations a nonlinear connection between these two could be useful similarly to the case of the death

rate. Focusing on hunting might make the predator a better hunter creating a nonlinearity, which alone could be enough for branching and speciation.

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