Theoretical Ecology: Research projects

At this time point in our course you have learned enough to make your first steps as a researcher in Theoretical Ecology. You have been trained to read and formulate mathematical models, and to analyze these graphically, mathematically, and numerically. You now have a few weeks to work on a somewhat larger project by yourself. The main goal of this project is that you apply your newly acquired skills to do a piece of research of your own. Most of the projects that we propose here start with a recent paper, but we also welcome proposals to work on a project of your own (please contact us to discuss this).

The projects described below typically start with studying the paper that we have suggested. Read the paper, repeat the results yourself by coding their model into `grind.R`. Find other papers, and define extensions of the research yourself. It is your own responsibility to study exciting new extensions of the work, and to make this a truly interesting project.

Please work in small groups of 3 students; we should aim for no more than 12 groups. Every project will be assigned a supervisor whom you can contact for help. A good time for this is after the seminar discussions and after the guest lectures. We have reserved computer-rooms but we would recommend to work on your own laptop. Make a concise "lab-journal" in which you **shortly** describe your progress every day that you worked on the project. This journal has to be handed in together with your written report.

Friday 10 November there will be a symposium at which every group presents their work using an electronic slide show. The oral presentation is in English, is short (10 minutes and 5 minutes discussion), and should be exciting for the audience. You have to make clear to the student audience what work your did, why it was interesting, and what results you have obtained. Too technical details should be avoided (as these will be explained in the written report). Subdivide your presentation into natural parts such that all members of your small research team get to speak! Your presentation should be enthusiastic and strongly focus on your main line of research.

The written report has to be delivered on Friday the 10th of November (electronic submissions of PDF-files by email to `r.j.deboer@uu.nl` are accepted until Sunday the 12th November). The report should have a summary, and start with an Introduction explaining the project, its context, and having a short review of the relevant literature. In the Methods section you can define and explain the mathematical model. In the Results section you can mix the results of the original paper with your own extensions of the research, and you can provide your extensions of the mathematical model. In the Discussion you describe possible problems/shortcomings, other extensions, and you provide further context of your work. Use the instructions on writing reports that you received in earlier courses, and **carefully read the short tutorial on writing scientific reports that this course provides**.

We will give written feedback on your oral and written presentation later by email. Please make an appointment to discuss that feedback with us. With the project we hope to increase your experience with the techniques you have encountered so far, and to show that you have arrived at a stage where you can critically continue the work of recent papers in this field.

1 Co-existence by chaos

The paradox of the plankton is the observation that plankton communities can be very diverse and are nevertheless limited by only a handful of resources. This is a paradox because the principle of competitive exclusion dictates that n resources can maximally sustain n consumers at equilibrium. We have seen in the course that 2 consumer can co-exist on a single resource on a 3-dimensional limit cycle.

Huisman & Weissing (1999) take this much further and show that many consumers can persist on a handful of resources when the system behavior is periodic or chaotic. Repeat their results with `grind.R` and discuss the parameter choices they make to get the desired behavior. Notice that they use minimum functions to implement Liebig's law of a single limiting resource, and try whether their results are affected if you change this into a smooth function (they have another paper on this). In another paper Beninca *et al.* (2008) also describe on a chaotic time series. Relate that to your work on this project.

2 Density dependent predation

In a recent review Terborgh (2015) summarized the importance of predation and disease in the maintenance of diversity of ecosystems. His article reviews several theories of species diversity, and his main take home message is that "keystone" predators killing the most abundant species play an essential role in ecosystem diversity. He calls this a "top-down" forcing of the food web (as opposed to the "bottom-up" theories on competition).

Read the paper and define for yourself what he means by density dependent predation. Study simple ODE models with density dependent predation in `grind.R` to explore the effects of this type of predation on competitive exclusion. For instance, how many prey species can co-exist by top-down control of one predator? How many predators can co-exist on a few resources, and could there be a feedback where an increase of prey diversity allows for an increase in predator diversity, which in turn increases the prey diversity?

3 CRISPR mediated immunity

The past few years we have learned that bacteria and Archeae protect themselves from infection by phages by a form of adaptive immunity. Bacteria acquire short (26-72 base pairs) DNA sequences from a phage that is infecting them and store these in their own genome in the form of Clustered Regularly Interspaced Short Palindromic Repeats (CRISPR). This protects them against infection by all phages expressing exactly the same sequence (protospacer). Phages adapt to that by mutations in these protospaces or in the sequence motifs around them (protospacer adjacent motif, or PAM). The molecular biology of this exciting system is rapidly being revealed, but the evolutionary population biology of co-evolving bacteria picking up protospacers and phages escaping from CRISPRs has hardly been studied. Levin *et al.* (2013) and Jiang *et al.* (2013) describe a number of unexpected observations in experimental studies of *in vitro* bacterial CRISPR evolution. They base their expectations on simple mathematical models. Read their papers and reproduce their results. Also read the review by Koonin & Wolf (2015) and try to generalize your results as much as possible.

One the website we provide two versions of this model in `grind.R`. One implements the delay-differential equations of the original model, an the other re-formulates that model into ODEs by

adding an intermediate variable. Both models can be fitted to the data in Figure 2 of the Levin *et al.* (2013) paper (these data are available on the website).

Estimate the parameters of the model using the `fit()` function. Do you require a time delay for obtaining a good description of the data? Implement their extended model and/or develop you own extensions that would provide alternative interpretations of the same surprising observations. Do you agree with their conclusion that CRISPR mediated immunity is not as powerful as was suggested by the molecular biology studies? You can study an evolutionary cascade of bacteria and evolving phages by allowing for stochastic mutations and/or stochastic accrual of CRISPR mediated immunity. Read the `grind.R` tutorial to see how to implement mutations in `grind.R`.

4 Group formation and predator-prey dynamics

Fryxell *et al.* (2007) have investigated the effect of the functional response on the stability of predator-prey interactions. First they measure the number of groups of lions and their eight prey species in the Serengeti national park as a function of the total population density in the park. Increases in the population density typically translate into more groups (prides) with a similar number of individuals. The effect of group size on ecosystem stability is studied by testing various functional responses in predator prey models. They obtain increased stability because group living decreases the *per capita* food intake.

Read the paper and the accompanying article by Coulson in the same issue of Nature, and redo the analysis to investigate if you agree with their results. Is this lower food intake an ESS?

5 Apparent competition and equal predation

The paper of Bonsall & Hassell (1997) illustrates the concept of "Apparent competition" where two prey species that are not competing with each other, do exclude each other when they share a predator that is eating them both. Write a 3-dimensional model for their experiments and study whether and how you can obtain apparent competition. You can use the `cube.R` extension of `grind.R` to plot 3-dimensional nullclines and trajectories.

One can also obtain the opposite result: a shared predator can prevent competitive exclusion between competing species. For instance the high diversity of grazed meadows is partly explained by herbivores keeping the population sizes small. Study whether and how you can get coexistence by predation. To make the problem interesting confine yourself to "equal predation", i.e., let the predator be blind to what it is eating. Help yourself by defining an r-selected and a K-selected prey. It could be good to define a resource for which you make a QSSA. One you have the coexistence make a bifurcation diagram corresponding to increasing the productivity of the food chain.

6 Competitive exclusion and parasitism

It is recently becoming clear that many species are suffering from a heavy burden with pathogens (Dobson *et al.*, 2008). If pathogens truly control population densities, this may increase ecosystem diversity by reducing competitive exclusion. This is often referred to as the Janzen-Connell hypothesis, e.g., in Sedio & Ostling (2013) and Bagchi *et al.* (2014). In the course we considered several populations of bird species with a birth rate declining linearly with the population size, and with a death rate

that is independent of the population density. We let the individuals be susceptible to an infection with a parasite that increases the death rate somewhat, but hardly affects the birth rate. We assumed that transmission of parasites occurs upon contacts between infected and susceptible individuals of the same species, and obeys mass action kinetics. Further there was no vertical transmission, i.e., the parasite is not transmitted to eggs. Thus, we let $N_j = S_j + I_j$ be the total number of birds, S_j be the susceptible non-infected birds, and I_j be the infected birds of the j^{th} species:

$$\frac{\mathrm{d}S_j}{\mathrm{d}t} = bN_j(1 - N_j/k) - d_j S_j - \beta S_j I_j \quad \text{and} \quad \frac{\mathrm{d}I_j}{\mathrm{d}t} = \beta S_j I_j - (d_j + \delta) I_j \ ,$$

where δ reflects the deleterious effect of the infection.

First analyze a 2-dimensional system, i.e., let $j = 1$ and consider one species. Second, study how many new species you can add to this one-species ecosystem assuming that (1) all bird species occupy the same niche, and (2) every new species has a faster death rate, i.e., a lower fitness, than the previous one ($d_{j+1} > d_j$). Make a simple function describing how d_j depends on j. Note that you can define vectors of equations in `grind.R` (see the tutorial).

The Janzen-Connell hypothesis typically states that pathogens are expected to evolve towards infecting the most abundant species. This is called negative density dependence (Bagchi *et al.*, 2014). Can you modify this model to study such effects of pathogen evolution? Other studies suggest that host and pathogen diversity in a community may also affect the infection rates (Johnson *et al.*, 2013). How would that affect these results?

7 Ontogenetic development for dummies

Persson & De Roos (2013) and De Roos & Persson (2013) summarize their extensive work on the effects of having juveniles and adults with different energetic requirements. These surprising effects include increases of the population size when the death rate increases, implicit Allee effects, and several more. The use both ODEs and PDEs for the modeling of the age dependent growth of the biomass of adults and juveniles, and these models are fairly complicated.

The aim of this project is to see whether their interesting effects can also be found in more simple (phenomenological) models, e.g.,

$$R = K - c_1 J - c_2 A \ , \frac{\mathrm{d}J}{\mathrm{d}t} = \frac{eAR}{h_2 + R} - \frac{mJR}{h_1 + R} - \mu d_1 J \quad \text{and} \quad \frac{\mathrm{d}A}{\mathrm{d}t} = \frac{mJR}{h_1 + R} - \mu d_2 A$$

where R is the available amount of resource, K the total, and c_1 and c_2 determine how much stored in juveniles, J, and adults, A. The rates at which juveniles mature, and the rate at which adults produce juveniles, depend on the availability of the resource. With the two h_i parameters one can change the symmetry of this dependence on the resource ($h_1 = h_2$ would be a conventional symmetric system). With the parameter μ one can increase the death rate of both juveniles and adults simultaneously.

Read their paper and try to repeat as much of their results with this toy model. You may also enjoy watching these lectures: `https://staff.fnwi.uva.nl/a.m.deroos/Research/Webinars/`.

8 Long term effects of vaccination

Holdo *et al.* (2009) investigate the limiting factors determining the wildebeest population size in the Serengeti ecosystem in East Africa (see also the primer by Getz (2009)). Possible factors are the tree

cover, which is related to rainfall and frequent fires, disease outbreaks, and competing herbivores like elephants. They study this by analyzing long time series (1960-2003) by fitting statistical models. At the start of this period the wildebeest were vaccinated to rinderpest, and as a consequence the wildebeest population increased. Rinderpest was eradicated in 2012 and is the second pathogen that went extinct due to our vaccination efforts.

See if you can describe the outcome of all these interactions with simple ODE models. You can introduce environmental variation, like fires, by allowing for noise on some of the parameters. (see the `grind.R` tutorial).

9 Critical Transitions (Marten Scheffer)

Recently the idea that we might be able to observe "early warning" signals in time series data of systems that are about to collapse is receiving a lot of attention (Scheffer *et al.*, 2009; Veraart *et al.*, 2012; Scheffer *et al.*, 2012).

a. Read the Scheffer *et al.* (2009) paper, and study the boxes on the underlying theory.

b. Write a `grind.R` model for equation in the legend of Figure 1: `x '= x*(1-x/k) - c*x^2/(h^2+x^2) + i;`. Note that they set $k = 10$. Plot the steady state of x as a function of the consumption parameter c using the `continue()` function.

c. Add noise to the system by adding and removing individuals using the option `after` in your call to `run()` (see the `grind.R` tutorial), and check for different values of c, and the standard deviation, how the system responds to the noise.

d. Study the behavior of the model by very slowing changing c, while performing a simulation (with or without noise). Add `c '= epsilon;` to model and make epsilon a very slow parameter, and perform a long `run()`. It is wise to prevent negative values of x by again using the option `after` (see the `grind.R` tutorial).

e. Note that you plot autocorrelations by saving the data delivered by model simulations, e.g., `data <- run(1000,...,table=TRUE)`, and then plot the value of x as a function of a previous value of x, e.g., `plot(data$x[1:999],data$x[2:1000],pch=".")`. Look at the autocorrelation for different values of c. Finally note that you can do correlations with the R-function `cor()`.

f. Another form of stochasticity is to allow for noise on a parameter, e.g., on k or h. Test how the system responds to noise on one of its parameters.

g. What do you think of the paper? Do you think you would be able to predict a catastrophic bifurcation, and what would be the best approach to detect this?

Read other papers on these critical transitions and test whether you can model the other situations with `grind.R`.

In the seminar we will discuss the Boerlijst *et al.* (2013) paper. Incorporate their arguments in your projects. There is also a series of papers by Boettiger and Hastings doubting the predictability of critical transitions (Boettiger & Hastings, 2012, 2013; Boettiger *et al.*, 2016).

10 Algae-Zooplankton oscillations and their functional response

We discussed in the course that when a normal saturated response is used for modeling how zooplankton feed on algae, one obtains too large amplitude oscillations, even if all parameters of the model are set to reasonable values. Several papers have addressed this and have suggested several solutions (Scheffer & De Boer, 1995; McCauley *et al.*, 1999).

In the course we have discussed a functional response with an explicit curvature parameter that can be

used to scale between a type-I and a type-II functional response, and we have argued that the presence of the Hopf bifurcation, and hence the amplitude of the limit cycles, may depend on the curvature of the functional response. Thus it is interesting to study data collected on the consumption rate of zooplankton, and test which values of the curvature parameter describes these data best. The book by Arditi & Ginzburg (2012) reviews several data sets that have been fitted to functional responses. Subsequently, one can study how these "realistic" functional responses affect the stability and the amplitude of the limit cycles of algae-zooplankton models.

11 Tilman's competition model

In the "Modeling Population Dynamics" book we sketched Tilman diagrams Tilman (1980, 1982). These diagrams are fully explained in his famous book "Resource competition and community structure" (Tilman, 1982), which is available in the UU library, and a recent review is provided by Tilman's chapter in the book of McLean & May (2007).

In a more recent paper Tilman *et al.* (1997) generalize this model to have many species, and suggest that these can all be maintained along a gradient of just two resources. Additionally, he studied the co-existence of many species by formulating models with temperature gradients. Make yourself more familiar with Tilman diagrams for the consumption of two essential resources (using Hill functions and minimum functions), and study the extensions in the more recent papers Tilman *et al.* (1997); McLean & May (2007) Several papers have confronted Tilman's ideas with experiments (Adler *et al.*, 2011; Reich *et al.*, 2012; Farrior *et al.*, 2013; Hautier *et al.*, 2015).

12 Tilman's metapopulation model

In the course we have studied a 2-dimensional version of the metapopulation model developed by Nee & May (1992) and Tilman *et al.* (1994). They describe very surprising results. First, the best competitor is driven to extinction when precisely its steady state density of occupied patches is destructed. Second, we have seen that habitat destruction can increase the total diversity of the system.

One assumption of the model is that the probability that an empty patch is colonized increases linearly with the number of migrants (e.g., seeds). One could argue that its is more realistic to describe this as a Poisson process, i.e., to write that the probability that a patch is **not** colonized is proportional to e^{-S}, where S is the average number of seeds per patch. The colonization rate for the top species, species p_1 in the book, then becomes $(1-e^{-c_1 p_1})$, instead of $c_1 p_1$. Note that this is a simple saturation function that you may simplify into a Hill-function. Study whether this new colonization rate would strongly affect the results.

During the course one of the students suggested that habitat destruction can also be implemented by increasing the extinction rate because pristine habitats are not only removed, but also become smaller by human activity. How does the effect of decreasing the size of the patches compare to reducing the number of patches?

Think of other improvements of the model and test whether these would change the results. Also, study a 3-dimensional model.

13 Influenza infections

Every year many people become vaccinated with a current influenza vaccine. It is a major challenge to design such a vaccine because influenza is evolving, and differs from year to year. It has been described that if a major fraction of the population is immune to the currently dominant strain, a new strain may evolve during the season for which most of the population has no immunity. This is called "strain replacement". People may even be infected with both strains during a season because the crossreactive immunity for both strains is short-lived. See Furuse & Oshitani (2016) for a recent paper on this topic.

Make an SIR model for a season of influenza, with two strains, while assuming that the population size is not changing during the season, and study how the size of the total epidemic depends on the fraction of people that are vaccinated before the season starts. A cool paper to discuss during you presentation is Smith *et al.* (2004), who depict the evolution of influenza in a 2-dimensional antigenic map.

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