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Eight Research Projects

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Stochastic impediments to biological diversification

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Goal

To devise a theoretical framework for understanding the impacts of demographic and environmental variation on evolutionary branching.

Background and motivation

Speciation is evolution at the grandest scale. Speciation is the mechanism responsible for generating the astounding biodiversity that is the most salient feature of life on Earth, and one of our most threatened and irreplaceable resources. Speciation is also one of the greatest mysteries of evolution, occurring primarily on scales of space and time beyond our experimental reach, and through a delicate interplay of events from the molecular shifting of nucleotides to the geologic shifting of mountains and rivers. For organisms with sexual reproduction, the process of speciation must account for the genetic isolation of incipient species, and speciation models must therefore embrace genetic dynamics (Coyne & Orr 2004). Accounting for this genetic dimension of speciation has been achieved largely at the expense of simplifying, or ignoring altogether, speciation's ecological dimension. The latter results from the complex features and interactions that distinguish species from one another and that enable a rich diversity of species to persist.

The importance of ecological interactions in speciation has been acknowledged for some time (the concept of reinforcement goes back to Dobzhansky and Wallace), yet few studies have had more influence in highlighting the ecological basis for speciation then that by Dieckmann and Doebeli (1999), which brought the nascent theory of mutation-limited long-term phenotypic evolution known as 'adaptive dynamics' to the attention of empirical and theoretical evolutionary ecologists. While genetic dynamics are addressed in this study, at its heart lies an ecological model for the evolutionary branching of a phenotypic trait describing competitive interactions: in the course of adaptation, this trait approaches an evolutionary branching point, i.e., an attractor of the evolutionary dynamics of a single morph that, once reached through directional selection, naturally leads to the emergence of a diverging dimorphism through frequency-dependent disruptive selection. In this project, we will focus on the dynamics around such evolutionary branching points. While ignoring genetic details for a start, we will extend previous work by focusing on the impacts of stochastic processes on the evolutionary dynamics leading to biological diversification.

Almost all quantitative theories of evolution have relied on stochastic models to address the impacts of chance events – such as mutations, births, deaths, and environmental changes – that affect the evolutionary process (Fisher 1937; Lande 1979; Coyne & Orr 2004). Adaptive dynamics theory typically considers two sources of stochasticity: random mutations and the loss of advantageous mutants to chance extinction while their numbers are small. What these models typically do not consider is demographic stochasticity of resident populations (resulting from intrinsic fluctua-

tions through stochastic birth and death events in finite populations of discrete individuals) and environmental stochasticity (resulting from external fluctuations in the environmental conditions a population experiences).

Several recent studies have tried to overcome these limitations. Claessen et al. (2007, 2008) explore simulations of evolutionary branching in small populations and found that demographic stochasticity can delay or halt diversification. Johansson and Ripa (2006) explored the impact that correlated environmental stochasticity has on diversification. They assume that as populations diverge, the environmental influences upon them become less similar. They demonstrate that this correlation can facilitate coexistence early in the process of branching, and as it weakens, the chance that a branch is lost increases. While these studies make an excellent start, a more general theory and mathematical framework is needed to untangle and quantify the multitude of effects stochasticity has on evolutionary branching.

Research questions

How do demographic and environmental stochasticity influence the process of evolutionary branching? What is the waiting-time distribution for evolutionary branching to persist under these stochastic processes?

Methods and work plan

We consider a general model of evolutionary branching in a one-dimensional trait and examine it through a series of approximations. We assume individuals are born at rate b and die at rate d. These rates may depend on the number N of resident individuals, on their traits x, and on a fluctuating environment E. Upon birth, an individual faithfully inherits the trait value of its parent with probability $1-\mu$. With probability μ, it experiences a mutation: its trait value is then drawn from a normal distribution around that of its parent. This results in a stochastic processes with two components: a demographic jump process on the positive integers N describing the population's size, and a mutational jump process on the continuous trait x.

While easy to implement numerically, this model is not directly amenable to analytical study. In an effort to make the analysis tractable, we will therefore consider several biological limits before relaxing the associated simplifying assumptions. First, we can choose functions for b and d ensuring that the total population is large at demographic equilibrium, so extinction of the entire population and fixation of deleterious mutations can be ignored. Second, we can assume that mutations are rare (mutation-limited evolution), so the resident population is close to its equilibrium when new mutations arise. Third, we can assume that variations in the environment E are negligible.

Based on these assumptions, we can divide the process of evolutionary branching into four stages and calculate the resultant distribution of waiting times until persistent evolutionary branching:

- Starting from a monomorphic resident population in the vicinity of the branching point, we first calculate the waiting time for a mutation to occur that, together with the resident trait, falls within the region P_2 of dimorphic trait space that allows for the mutual invasibility of each morph by the other and therefore contains all protected dimorphisms. When the resident population is situated exactly at the

branching point, all mutations fall into P_2, but when the resident population has not yet converged to the branching point, mutational steps have to exceed a minimum size to enter P_2.

- Even though a mutant entering P_2 experiences a positive growth rate, it must establish itself by reaching a population size that makes chance extinction unlikely. From the first stage, we know the probability density of mutants entering P_2. For each such mutant, we can determine the probability that it escapes initial chance extinction.
- Having survived initial chance extinction, the mutant finds itself in a dimorphic population. As the resident trait and the mutant trait are very similar, and since both lie near the branching point, the frequency-dependent selection promoting their coexistence is very weak, so that the random drift of their relative frequency readily leads to the loss of mutant or resident before further mutations allow the dimorphism to widen.
- In a fourth stage, the now established populations undergo a random walk of their trait values in P_2. Should this random walk hit the boundary of P_2, one morph goes extinct and the entire process must start again. By calculating the probability that the dimorphic random walk escapes the boundary, we can thus quantify the fourth and final stage of evolutionary branching.

Combining results for each stage, we can determine the distribution of waiting times until persistent evolutionary branching.

Relevance and link to EEP's research plan

This work is directly relevant to EEP's project on *Adaptive Dynamics Theory*, by extending the analysis of evolutionary branching to include the effects of demographic and environmental stochasticity.

Expected output and publications

The results of this research will be submitted as a jointly authored paper to a peer-reviewed international scientific journal in the area of ecology and evolution.

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Public-goods games under time pressure

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Goal

To investigate the role of time and time pressure in the contributions to a general public good.

Background and motivation

A group's public goods require costly investments by individual group members while benefiting all group members irrespective of their investments. This leads to a so-called social dilemma: as non-contributors cannot be excluded from the benefits of the public good, there is a strong incentive for free riding (Hardin 1968). Without mechanisms ensuring the cooperation of players, this usually results in the failure of the public good (Ledyard 1995).

Much research in the last decade has focused on public-goods games and on mechanisms that maintain cooperation. These approaches include the possibility of punishing free riders (e.g., Fehr & Gächter 2000; Sigmund et al. 2001), voluntary participation (e.g., Semmann et al. 2003; Hauert et al. 2002), and the combination of the public-goods game with pairwise interactions (e.g., Milinski et al. 2002; Panchanathan & Boyd 2004).

However, important examples of public goods contain the strategic element of time, which has largely been neglected by experimental studies and theoretical analyses to date. Examples are given by effort levels in joint projects or by the "probably greatest public-goods game" (Milinski et al. 2006), the earth's climate. In both games, the strategy "wait and see" obviously plays a key role. Additionally, both examples are characterized by time pressure: joint projects usually have a deadline, and actions against climate change are more effective the earlier they are implemented. If the effectiveness of contributions to the public good decreases over time, the social dilemma is intensified, as the "wait and see" strategy may lead to late (and hence expensive) contributions to the public good. Without explicitly incorporating a temporal dimension, these important features are not adequately captured.

There is another interesting feature of the public good given by earth's climate: Unlike in typical public-goods games, the benefits of investments in stabilizing the earth's climate are not equally shared, since the consequences of global warming on single countries differ. From the point of view of a country for which consequences are moderate, such a setting might provide an additional incentive to adopt a "wait and see" strategy.

Surprisingly, despite the importance of these and other examples of public-goods games, no theoretical model and only a handful of experimental studies (Milinski et al. 2006, Milinski et al. 2008) as yet explicitly address the dimension of time in these games. The experiments of Milinski et al. (2008) try to imitate investments into the prevention of climate change. They find that, if investments have to exceed a certain

threshold to be effective, the social dilemma can only be relieved if a failure to invest enough results in grave losses for individuals.

Some theoretical studies have dealt with the strategic element of time in a different setting: for example, Eskola (2009) discusses the optimal timing of reproduction, and Uehara et al. (2007) deal with the optimal display duration in animal contests. Eriksson et al. (2004) examine the war of attrition with an implicit time cost. We plan to transfer ideas developed in those other contexts to social dilemmas over public goods.

Research questions

The aim of this research project is to obtain a better understanding of optimal schedules for timing investments into a public good under time pressure. More specifically, we will try to answer the following questions:

- What are the conditions that prevent a public good under time pressure from failing? Will there be a stable mixture of different strategies (e.g., a coexistence of high contributors with low contributors), or will the adaptation of investment schedules lead to uniform contributions?
- What is the eventual shape of the investment schedule? Will there be some last-minute effects, i.e., will there be a jump in the level of contributions when players perceive the impending failure of a public good?
- What is the impact of group size on the cooperation level of individuals, especially if the benefit of a public good does not depend linearly on contributions?
- As an extension, one may consider the effects of heterogeneity in the benefits different players derive from a public good. Does this lead to an increase of contributions by those players who gain a relatively high benefit from the public good? At equilibrium, will all players obtain the same net benefit from the public good?

Methods and work plan

As these questions have so far not been treated explicitly, we will firstly use agent-based simulations to get an overview of possible settings for which a public good can be maintained under time pressure. As a starting point, we will use the following model. We consider a population of N players who are engaged in a public-goods game. Each player $i = 1,\ldots,N$ decides independently of the others about the time t and level c of its contributions, based on a function-valued strategy $c_i(t)$, with $c_i(t) \geq 0$ for all t. Additionally, we assume that the capacities of players to contribute to the common resource at a given time are bounded, $c_i(t) \leq \overline{c}$ for all t.

The public-goods game is played in a randomly formed group of n players. We denote the total instantaneous contributions at time t by $C(t) = \sum_{i=1}^{n} c_i(t)$. Then the-benefit derived from the public good at time T has the following general form, $B(T) = F\left(\int_0^T e(t)C(t)dt\right)$. In this expression, the function F describes the benefit derived from the public good in dependence on the total effective contribution made toward the public good until time T. The function $e(t) \geq 0$ describes the efficiency of contributions made at time t. In the simplest case $e(t) = 1$, it does not matter at which time the contributions are made. By specifying functions $e(t)$ with $de/dt \leq 0$, one can incorporate the possible assumption that the efficiency of contributions toward the

public good time decreases with time: the later contributions are made, the more input is needed to reach a certain level of total effective input. This assumption can be illustrated by considering the example of investing into the stabilization of the earth's climate. In this case, $e(t)$ can be regarded as a discounting factor for contributions made at time t. Generally, $e(t)$ might take any functional form, but for this project I will use two standard forms: $e_1(t) = 1$ (no discounting of future contributions) and $e_2(t) = \exp(-\alpha t)$ (discounting with a constant rate α). Also the function $F(x)$, which relates total effective inputs to benefits derived from the public good, can take any functional form that respects that benefits are non-negative ($F(x) \geq 0$ for all x) and that benefits do not decrease with the total effective input ($dF/dx \geq 0$ for all x). In addition, realistic examples often require respecting the following two conditions:

- There is a maximum benefit $B_{\max}$, so that even arbitrarily large total effective contributions toward the public good result in a limited benefit: $F(x) \leq B_{\max}$ for all x with $\lim_{x\to\infty} F(x) = B_{\max}$, describing a diminishing return.
- A minimum level of total effective contribution is required for the public good to yield a benefit larger than $B_{\max}/2$.

For example, a function that fulfills both of these assumptions is $F(x) = B_{\max} e^{\alpha(x-\beta)}/(\gamma + e^{\alpha(x-\beta)})$ with positive parameters α and γ. Then $F(x) > B_{\max}/2$ requires $x > \beta + \alpha^{-1}\ln(2\gamma)$.

We furthermore assume that the duration t^* of each public-goods game is a random variable drawn from the unit interval $[0,1]$. We consider several options for the distribution of t^*:

- The game duration is fixed to $t^* = 1$ (i.e., t^* follows a Dirac delta distribution with a peak at 1). This corresponds, for example, to joint projects with equally long deadlines.
- The game duration is drawn from a uniform, exponential, or Weibull distribution. This can be illustrated, for example, by investments into the defense of a community continually threatened by external attacks.

If the benefit of the public good is shared equally among all players, then the total payoff of each player is given by

$$P_i(t^*) = \frac{B(t^*)}{n} - \int_0^{t^*} c_i(t)dt\,.$$

In order to include strategic interactions among players, we additionally assume that the contributions of a player at time t may depend on one or more of the following three quantities:

- The recent effective total contributions of all players, $\int_0^t e(\tau)C(\tau)d\tau$. This is a measure of the input that may still be needed to reach a certain level of benefit,
- The player's own effective total contributions, $\int_0^t c_i(\tau)d\tau$. This is relevant since each player's resources are limited and because players want to prevent being exploited and thus need to monitor their own investments.
- The total instantaneous investments of all players made at any given time t, $C(t)$.

In carrying out this research, we will first focus on separately exploring each of these three listed dependences. In order to maintain cooperation in the public-goods game, some mechanism is needed that favor cooperators over free-riders. The easiest

mechanism we will consider is a snowdrift game with parameters such that a single cooperator excels in a population of free-riders. The type of game can vary with group size n, so that changing n can turn a snowdrift game into a prisoner's dilemma. In such cases, an additional mechanism is needed for the evolution of cooperation to prevent the public good from failing.

A different approach would follow the assumptions of Eriksson et al. (2004). They consider a public-goods game that is played until a certain benefit $B_{\min}$ is reached. This implies that cooperators on average play longer games than free-riders, and hence may receive higher total payoffs. One way to include such an idea into our setting is to assume that, with the game's maximal duration being fixed to 1, players first may contribute toward the public good until the time t^* when $B_{\min}$ is reached, after which each player obtains a benefit of $\sigma(1-t^*)$.

We embed the public-goods game described above into an evolutionary process, so that investment schedules evolve according to their relative success in the game. This can be modeled by a stochastic process, which may be similar to the one investigated by Nakamaru & Dieckmann (2009). Each individual may die according to the probabilistic rate $d_i = 1/N$. Its place is then filled by the offspring of player i with probability $P_i / \sum_{j=1}^{N} P_j$. The offspring's strategy changes from that of its parent i with probability m, from $c_i(t)$ to $c_i(t) + s_i(t)$, where the total change is small, $\int_0^1 |s_i(t)|\, dt < \varepsilon$. Finally, we assume that there is a small probability that player i switches to full free-riding, $c_i(t) = 0$ for all t.

After the identification of interesting settings through agent-based simulations, we will try to obtain analytic results using the adaptive dynamics theory of function-valued traits (Dieckmann et al. 2006; Parvinen et al. 2006).

Relevance and link to EEP's research plan

This project applies techniques that have largely been developed by researchers associated with EEP's research project on *Adaptive Dynamics Theory* (e.g., Dieckmann et al. 2006; Parvinen et al. 2006). Additionally, the theoretical analysis of public-goods games is a core theme of EEP's research project on the *Evolution of Cooperation*, which has made many interesting contributions to this field (e.g., Hauert et al. 2002; Nakamaru & Dieckmann 2009).

Expected output and publications

The research is intended for publication as a coauthored article in an international scientific journal and will form an essential part of my PhD thesis.

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Virulence evolution in fragmented populations

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Goal

To study how virulence evolution is altered by the fragmentation of an infectious disease's host population.

Background and motivation

Epidemiological models focus on infection dynamics without considering evolution of the infectious agent. However, since the generation times of infectious agents are often much shorter than the generation times of their hosts, infectious agents often evolve on timescales considerably shorter than a host generation. This means that the evolution of an infectious agent can be important in studying disease dynamics, giving rise to the field of evolutionary epidemiology (Ewald & de Leo 2002; Restif 2009).

Virulence is typically measured by the extent to which the infectious agent reduces the fitness of its host (Alizon *et al*. 2009; Galvani 2003). Virulence evolution is often governed by a trade-off between an infectious agent's needs for achieving a high rate of transmission between hosts while keeping them alive to prolong such transmission. Classical models for the evolution of virulence therefore assume a trade-off between transmission rate and virulence, with elevated virulence implying higher transmission rate (Galvani 2003). Since directly transmitted diseases are dependent on the movement of their hosts for transmission, it is assumed that the transmission rate decreases for high virulence, reflecting the immobilizing effect the infectious agent has on the host (Ewald & de Leo 2002).

Models for the evolution of virulence also often assume that the host population is well-mixed, so that all pairs of hosts experience equal encounter rates (Read & Keeling 2003). However, many natural host populations are spatially structured. An important form of spatial structure is described by metapopulations: populations of hosts that are internally well-mixed inhabit different patches that are loosely connected to each other, enabling some exchange of hosts between the populations. Typical examples are groups of villages for human diseases or groups of farms for livestock diseases. To describe the epidemiological dynamics of directly transmitted diseases in metapopulations, it is crucial to consider the movement of hosts between patches. This is especially important if the size of populations is so small that the disease cannot persist in a patch in the absence of such movement. In that case, the recurrent spread of the infectious agent into patches with susceptible individuals is critical for the disease to persist in the metapopulation (Hanski 1999).

In this project, we will model a metapopulation in which an infectious disease spreads within and between populations. We consider a directly transmitted infectious disease, which only spreads through the movement of its hosts. Modeling the movement of hosts between patches allows us to investigate the possible immobilizing effect of the infectious agent on the host: hosts infected by agents with higher virulence may stay longer in a patch before moving to another. Similarly, higher virulence may

reduce the number of patches that hosts pass through during a movement step, and increase the mortality they suffer during movement.

In models of disease evolution, virulence often affects the disease's transmission rate, the host's recovery rate, or the host's mortality rate (Alizon *et al*. 2009; Galvani 2003). We will extend these earlier studies, by investigating the staying duration, movement distance, and movement mortality of hosts as functions of virulence. We expect that, depending on these dependences, costs and benefits of virulence balance at different levels of virulence. This will allow us to predict how a disease's impact on host mobility alters its severity.

Research questions

We will study how virulence evolution depends on the impact of virulence on the demography, epidemiology, and movement of hosts in a metapopulation. Specific questions that will be addressed are as follows:

- How is virulence evolution affected by the impact of virulence on the average duration a host stays in a patch, on the average number of patches it traverses during movement, and on the average probability of dying it experiences during such movement?
- How are these effects altered by the impacts of virulence on the death rate and recovery rate of hosts, and on the transmission rate among hosts?
- How does a metapopulation's connectivity structure influence virulence evolution?

Methods and work plan

The model we will examine is stochastic and continuous in time and describes the spread and virulence evolution of an infectious disease in a metapopulation of hosts. Hosts can move between the metapopulation's patches, and these movements are modeled explicitly. In each patch, an $\mathrm{SI}_v\mathrm{S}$-type dynamics occurs, with each host being either susceptible (S) or infected (I) by a strain with virulence v. Infected hosts are infectious. While a host is infected by one strain of the infectious agent, it cannot be infected by a different strain (i.e., super-infection and co-infection are precluded). The disease is transmitted directly, which makes the movement of hosts among metapopulation patches a key factor for its spread and persistence.

The populations in each patch are well-mixed and change through the following events: birth, death, infection, recovery, and movement to and from another patch. We assume a maximum population size of K for each patch; beyond this maximum size, there is no birth or immigration. When hosts emigrate from a patch, they enter the disperser pool. Since mortality during movement is an important factor regulating the evolution of virulence (Ewald & de Leo 2002), we consider how the disease's virulence alters the survival probability of hosts in the disperser pool. Individuals trying to leave the disperser pool can only enter patches in which the total number of hosts is smaller than K.

The equations describing this model extend the traditional differential equations for the number or density of susceptible and infected hosts in a single population. In our model, the state of the metapopulation is described by the fractions $p_{s,i}$ of popula-

tions with precisely s susceptible and i infected hosts. Following Metz & Gyllenberg (2001), the equations for the dynamics of $p_{s,i}$ are combined with equations for the dispersal of susceptible and infected hosts. We denote the birth rate by b, the death rate by μ, the disease-induced mortality by α, the transmission rate by β, the recovery rate by γ, the emigration rate by m_{out}, and the immigration rate by m_{in}. The latter two rates may differ between susceptible and infected hosts. The metapopulation dynamics are then given by

$$\frac{d}{dt}p_{0,0} = -p_{0,0}[m_{\text{in,S}}D_{\text{S}} + m_{\text{in,I}}D_{\text{I}}] + p_{0,1}[\mu + \alpha + m_{\text{out,S}}] + p_{1,0}[\mu + m_{\text{out,S}}],$$

$$\begin{aligned}\frac{d}{dt}p_{s,i} = &-p_{s,i}[b(s+i) + \mu(s+i) + \alpha i + \gamma i + \beta\frac{si}{s+i} + m_{\text{in,S}}D_{\text{S}} + m_{\text{in,I}}D_{\text{I}} + m_{\text{out,S}}s + m_{\text{out,I}}i]\\ &+ p_{s+1,i}[\mu(s+1) + m_{\text{out,S}}(s+1)] + p_{s,i+1}[\mu(i+1) + \alpha(i+1) + m_{\text{out,I}}(i+1)]\\ &+ p_{s+1,i-1}\beta\frac{(s+1)(i-1)}{s+i} + p_{s-1,i+1}\gamma(i+1) + p_{s-1,i}[m_{\text{in,S}}D_{\text{S}} + b(s-1+i)] + p_{s,i-1}m_{\text{in,I}}D_{\text{I}}.\end{aligned}$$

The fraction of susceptible and infected hosts in the disperser pool, D_{S} and D_{I}, respectively, change according to

$$\frac{d}{dt}D_{\text{S}} = m_{\text{out,S}}\sum_{s,i=1}^{s+i\le K} p_{s,i}s - m_{\text{in,S}}D_{\text{S}}\sum_{s,i=1}^{s+i<K} p_{s,i} - \mu_{\text{D,S}}D_{\text{S}},$$

$$\frac{d}{dt}D_{\text{I}} = m_{\text{out,I}}\sum_{s,i=1}^{s+i\le K} p_{s,i}i - m_{\text{in,I}}D_{\text{I}}\sum_{s,i=1}^{s+i<K} p_{s,i} - \mu_{\text{D,I}}D_{\text{I}},$$

where $\mu_{\text{D,S}}$ and $\mu_{\text{D,I}}$ denote a susceptible or infected host's death rate during movement. Since the maximum population size in a patch is K, the fraction of patches with more than K hosts equals zero ($p_{s,i} = 0$ for $s+i > K$). Obviously, negative population sizes are also not feasible ($p_{s,i} = 0$ for $s,i < 0$).

Variations in virulence are introduced by allowing the infectious agent to mutate within an infected host. Such a mutation will be expressed when an infected host infects a susceptible host. Simple functions will be used to describe the effects of virulence. For example, for the transmission rate we will use the function $f(v) = \sigma v/(1+\sigma v)$, while for the duration of stay we will use the function $f(v) = m/(1+\sigma v)$, where σ specifies the function's sensitivity to variations in virulence and m specifies the function's maximum.

In order to get some feeling for this model, it will first be studied semi-analytically in the case that (i) the number of patches is large, (ii) the mutation probability is small, and (iii) all patches are equivalent and equally connected. The invasion fitness defined by Metz & Gyllenberg (2001) will be used to derive the fitness of virulence mutants. This invasion fitness, the equivalent of the basic reproduction number R_0 for single populations, measures the expected number of mutants leaving the disperser pool for each mutant entering it (Parvinen et al. 2008). First, the equilibrium of the metapopulation model will be determined numerically for a monomorphic resident population infected by a disease with virulence v. Then, the invasion fitness of a mu-

tant infectious agent with virulence v' will be calculated. Based on this invasion fitness, the course and outcome of virulence evolution can be predicted.

After these semi-analytical studies, the model will be programmed in C++ in order to study the effects of relaxing the simplifying assumptions (i) to (iii). In particular variations in connectivity structure are considered important for virulence evolution (Alizon *et al.* 2009; Read & Keeling 2003). The Gillespie algorithm (Gillespie 1976) will be used to determine the schedule of events, resulting in exponentially distributed waiting times between events and in events being chosen according to their relative probabilistic rates. We will examine situations in which host movement is restricted to nearest neighbors on a square grid with periodic boundaries, before allowing movement over longer distances and/or on other connectivity structures.

If time permits, we will include the possibility of recovery with permanent immunity. This requires the consideration of recovered-and-immune hosts and studying the epidemiological and evolutionary aspects of the resultant SI_vR -dynamics.

Relevance and link to EEP's research plan

Contributing to EEP's research project on *Adaptive Dynamics Theory*, this project combines evolutionary biology with epidemiology and ecological realism. It is fundamental in the sense that it broadens our knowledge of virulence evolution in metapopulations.

Expected output and publications

This research is intended for co-authored publication in an international scientific journal and will be part of my PhD thesis.

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Evolution of vegetation structure

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Goal

To explore how evolutionary diversification through endogenous selection on tree architecture depends on ecological conditions.

Background and motivation

Trees and plants are of key importance for upholding the world's biodiversity, as they provide habitats and serve as a source of food for a great number of different animals. An exceptional case of plant biodiversity is reported by Wright (2002), who found that a tropical rain forest in Ecuador supported 1104 tree species in an area as small as 0.25 square kilometers. Understanding the principles that underpin the emergence of such remarkable diversity is of tremendous importance if we are to preserve our world's ecosystems for future generations.

Trees prominently differ in the architecture of their stems and crowns. In a landmark study, Iwasa et al. (1985) derive evolutionarily stable tree heights and crown shapes by analyzing competition for light. A key finding of that study is that trees invest large amounts of energy into tall trunks, something that can only be understood in the context of their competition with other trees. When growing in solitude or among kin, trees save the effort of producing tall trunks and enjoy higher fitness. Similarly, crown shape depends on how other trees shape the light environment. A further interesting result reported by Iwasa et al. (1985) is that a polymorphic evolutionary equilibrium is reached when a canopy is thin enough, consisting of trees with equal fitness but different heights (Falster and Westoby 2003).

Niklas (1992) describes a detailed tree-architecture model in an evolutionary context. The trees in his model have branches, in contrast to the simple geometric objects and functions usually examined in ecological tree models. Niklas explores the evolution of tree architecture under optimizing selection, maximizing light interception, mechanical stability, and reproductive capacity. While this yields insights into important factors determining tree architecture, the definition of fitness in this model is statically prescribed rather than dynamically derived from an underlying ecological model. The focus on optimizing selection neglects the endogenous environment that is created by the trees themselves, and thereby prohibits the emergence and coexistence of different tree types through frequency-dependent interactions.

While the studies by Iwasa et al. (1985) and Niklas (1992) were major leaps forward, neither account for the process of ontogenetic growth from seedlings to large trees. As tree architectures that are competitively superior in early phases of vegetative growth need not do as well during later phases, the ontogenetic growth process potentially has important evolutionary implications. A first inroad into understanding these implications has been provided by Yokozawa et al. (1996), who studied the coexistence of conic-canopy plants (conifers) and spheroidal-canopy plants (hard-

woods). However, no study to date has considered the evolution of tree architecture in an ecologically realistic model of growing plants.

Research questions

The aim of this research project is to understand the evolution of tree architecture in an ecologically realistic vegetation model that explicitly incorporates ontogenetic growth. I propose two main research questions:

- What is the optimal architecture for a tree growing in solitude, or in a group of genetically identical trees?
- What is the stable monomorphic or polymorphic evolutionarily stable strategy in a given environment?

Explicit consideration of external environmental factors such as wind, fire, grazing, and light will be essential in answering these questions. My focus will initially be on wind and light, since these factors most clearly affect trees differentially depending on their architecture.

Methods and work plan

Metapopulation dynamics

I will investigate a spatially implicit metapopulation model of competing trees. The ecological dynamics will describe a structured population and the evolutionary dynamics will be explored using adaptive dynamics techniques and the canonical equation (Dieckmann & Law 1996; Geritz et al. 1998).

As a first approximation, trees will be assumed to have simple geometric shapes consisting of a spheroidal crown, whose top is attached to the tip of a conical trunk. Each tree will be characterized by three adaptive phenotypic traits: the trunk's apex angle, the relation of crown width to crown height, and the proportion of energy available for growth invested into the crown as opposed to the trunk.

The model comprises two coupled components, with the first describing the age distribution of stands and the second describing the size distribution within stands. Stands only interact through random seed dispersal. Seeds enter a common seed pool and are then redistributed randomly among stands. There is no horizontal structure within stands, meaning that trees are interacting through a "mean field". No edge effects exist in or between patches. Stands are destroyed and regenerated with new saplings according to a probabilistic rate depending on patch age.

A stand's size structure is governed by a diffusion equation describing the vegetation density $n(m,a)$ at age a and plant mass m,

$$\frac{\partial n(m,a)}{\partial a} + \frac{\partial}{\partial m}\left[g(m,a)n(m,a)\right] = -d(m,a)n(m,a),$$

where $g(m,a)$ is the mean growth rate and $d(m,a)$ is the mortality rate. A numerical discontinuous Galerkin method implemented in Matlab will be used for solving this equation.

The physical properties of trees will be modeled with simple relations that try to capture the most important features. Using the three adaptive traits mentioned above, we will try to describe trees as realistically as possible. This project will therefore build on ongoing work by Daniel Falster from Macquarie University in Sydney, Aus-

tralia, former participant in IIASA's YSSP, to make full use of insights derived from his earlier work. By starting from the model he designed, a great deal of effort can be saved.

Light environment

Once the distribution $n(m,a)$ is known, we can calculate the average biomass density at any height. We obtain the light intensity at canopy depth d through the Beer-Lambert law $I(d) = I(0)\exp(-kF(d))$, where $F(d)$ is a patch's accumulated leaf area above depth d, and k describes the extent of light attenuation through leafs. The light intensity above the canopy at depth 0 is $I(0)$ (Prentice et al. 1990). The actual energy intake derived from light interception depends on a tree's photosynthetic rate, which grows linearly with light intensity when the latter is small and is saturated at high light intensities. The sun will be modeled as having an annually varying angle α above the horizon, with light intensity diminishing at small angles. By averaging light interception over the distribution of angles corresponding to a climate zone, the effective annual light interception can be estimated. As a start, just keeping the sun fixed at a given angle should yield interesting results.

Self-shading will be added to the mean-field shading of a stand to study the effects of crown architecture. A two-dimensional ellipse is easier to integrate than a three-dimensional spheroid, and therefore preferable for describing crown shape. We need to determine the distance travelled by light inside the spheroid, and this can be found by basic linear algebra. The leaf distribution within a crown is assumed to be uniform, so that the accumulated leaf area is proportional to this distance. Knowing the light intensity at many points in the ellipse, we can approximate light intensity within the whole ellipse. The distance from an interior point to the edge of the ellipse trivially scales with the size of the ellipse, so that distances can be computed in advance independent of crown sizes.

Only crowns will be shading, not trunks. The light angle is important for how crowns are shaped, as it alters self-shading. In northern boreal and sub-boreal forests conifers are successful, whereas on the African savannah acacias are successful. The maximizing of light interception helps explain these patterns.

Energy allocation

Energy gained from photosynthesis will be allocated to different physiological functions. First, a portion of energy intake will be used for maintenance. The allocation to maintenance depends on the mass of leaves. What is left will be used for reproduction and growth. The relative proportion of growth energy directed to the crown as opposed to the trunk is described by the adaptive trait r which is one. In the model by Daniel Falster, the mass of sapwood, bark, heartwood, and fine roots are all related to the mass of leaves through fixed equations depending on traits r.

A complicating factor is that the energy allocation of trees may vary throughout their lifetime, so more realistically r should dependent on tree biomass. The pipe model (Shinozaki et al. 1964 a,b) states that the cross-section of a tree's stem at any height above ground is proportional to the accumulated weight of its leaves situated above that height. This will guide us in devising, as an extension, a more realistic time-dependent model of energy allocation.

Disturbances

Breakage of branches and removal of leaves will occur when trees are affected by wind and grazing. Since we do not explicitly model branches and leaves, we will account for breakage through its secondary effects. There are three essential ways to do this: increased maintenance cost, reduced leaf area density, and increased mortality risk. Grazing implies the investment of energy into producing leaves that will be of no use. Assuming that grazing primarily affects low leaves, it can be described by reducing light intensity at low heights.

Wind affects trees through the breakage of branches and defoliation, as well as through the breakage of entire trees and the removal of stands. Ignoring the effects of turbulence, the wind strength can be thought of as decreasing exponentially with a stand's biomass at a certain height. Knowing the horizontal wind strength at every vertical point, we can approximate the defoliation rate and the breakage rate of branches. Reasoning as in the case of grazing, wind can be simulated as a light filter. This filter will depend on a stand's biomasses at different heights, so that light intensity will decrease more on those horizontal levels on which biomass is low. For the mortality rate resulting from entire trees breaking in a storm, the distance from the ground to their centers of mass is important. A tree with a height deviating greatly from those in its stand will experience a higher breakage risk than other trees, elevating its instantaneous mortality rate.

Catastrophic events like storms and fires will occasionally wipe out entire stands, thereby decreasing the mean disturbance interval in the metapopulation.

Possible extensions

After the successful completion of the proposed tasks, many extensions are possible. For example, instead of using a spheroid, a crown can be shaped like a cylinder, a cone, or according to a profile defined by a function-valued trait. Likewise, a trunk can be modeled as a cylinder or be shaped according to another function-valued trait. However, trunk shapes are less interesting than crown shapes, because the former largely depend on the mass distribution within crowns.

Relevance and link to EEP's research plan

This research project relates directly to EEP's research project on Evolving Biodiversity. It is aimed to yield new fundamental insights in the theory of plant evolution and ecology, and is expected to strengthen EEP's participation in the international collaboration on Evolutionary Ecology Vegetation Models (EEVMs). The anticipated results may furthermore be of interest to researchers working with dynamic global vegetation models.

Expected output and publications

I expect the results of this project to be part of my PhD dissertation; they will also be published as a co-authored research article in an international scientific journal.

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Fisheries-induced evolution of neutral and selected genetic markers

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Goal

To develop a multi-trait individual-based eco-genetic model for studying the roles of neutral and adaptive fisheries-induced evolution in harvested fish populations.

Background and motivation

Various studies – based on field observations, experiments, and theoretical models – have highlighted that, on top of its demographic impact on harvested populations, fishing might induce individual life-history traits to evolve (e.g., Jørgensen et al. 2007; Hutchings and Fraser 2008; Kuparinen and Merilä 2007). Because of the very high levels of fishing mortality and its size-selectivity, individuals with slower growth rate, earlier maturation at smaller size, and higher reproductive investment are predicted by life-history theory to be selectively favoured. In line with such predictions, experiments have shown that unexpectedly rapid harvest-induced evolution of life-history traits is possible (e.g., Conover and Munch 2002; Reznick et al. 1996; Reznick et al. 2005). Furthermore, changes in life-history traits, particularly in the age and size at maturation, have been reported in a number of harvested fish stocks. These changes in maturation can have two origins. First, fisheries-induced evolution due to selective forces imposed by fishing may have occurred. Alternatively, such changes may result from phenotypic plasticity alone, if, for example, a fisheries-induced reduction in stock biomass relaxes intraspecific competition, and thereby leads to faster individual growth and hence to earlier maturation.

In an effort to disentangle phenotypically plastic changes in maturation in response to fishing from genetic changes, probabilistic maturation reaction norms (PMRNs) have been introduced to characterize maturation independently of growth and survival (e.g., Barot et al. 2004; Heino et al. 2002; Olsen et al. 2004; Grift et al. 2007). Size-selective fishing mortality can cause PMRNs to shift toward smaller ages and sizes at maturation. Such a shift may be evolutionary, because PMRNs account for phenotypic plasticity resulting from changes in average juvenile growth rates, which are causing a major part of environmental variation in maturation (Ernande et al. 2004). However, even when estimating PMRNs, evidence for fisheries-induced maturation evolution is only available at the phenotypic level, whereas one would ideally want to know whether genes underlying maturation tendency have evolved at the molecular level. Unfortunately, no genes specifically coding for maturation schedules have as yet been identified. Their identification is the goal of ongoing work that, in the longer run, might provide the necessary molecular tools for assessing genetic evolution underlying phenotypic maturation changes.

In parallel to these phenotypic studies of adaptive evolution, others have investigated the influence of fishing pressures on the neutral genetic diversity of specific stocks, using DNA extracted from archived collections of otoliths or scales. These

studies, based on neutral genetic markers (mostly microsatellite loci and/or mitochondrial DNA), have shown a loss of neutral genetic diversity in several harvested populations, including orange roughy (Smith et al. 1991), red drum (Turner et al. 2002), New Zealand snapper (Hauser et al. 2002), and North Sea cod (Hutchinson et al. 2003). While neutral genetic markers are insensitive to selection, studying their variation has many applications, including genetic mapping of a population's geographic structure, stock identification, and phylogeny estimation. Neutral genetic markers are influenced by genetic drift, which is the change in the genetic composition of a population by purely random processes unrelated to selection pressures; these processes are especially important when for populations of small size. Genetic drift also influences genes coding for life-history traits influenced by fisheries-induced evolution. To disentangle these two causes, changes in the genetic composition of adaptive genetic markers have to be assessed against the baseline of changes observed in neutral genetic markers.

In summary, fishing mortality may increase genetic drift and generate selection pressures, both affecting the composition of coding genes in exploited fish stocks, whereas only the former affects the composition of neutral genetic markers. In turn, changes in the neutral genetic markers leave traits unchanged, whereas changes in coding genes modify phenotypes.

Research questions

The aim of this project is to study the effects of commercial fishing on the evolution of PMRNs, somatic growth rates, and reproductive investments, by developing an individual-based eco-genetic model with an explicit description of genetic transmission during sexual reproduction. In this model, individuals will carry neutral genetic markers and genes coding for the considered life-history traits. This will allow studying the interplay between neutral and adaptive evolution in the context of fishing.

Specifically, the following research questions will be addressed:

- What are the effects of different kinds of fishing pressures on neutral genetic diversity? Changes in neutral diversity are caused by increases in genetic drift due to decreases in population size. The extent to which different fishing practices, characterized by their selectivity and intensity, may have different consequences in terms of neutral genetic diversity is currently unknown.
- What are the consequences of changes in neutral genetic diversity for the ecological properties of a stock? Traditional approaches in population genetics interpret changes in neutral genetic diversity as being related to stock characteristics such as population size, reproductive potential and success, spawning stock biomass, or level of recruitment. In particular, decreases in neutral genetic diversity are interpreted as indicators of detrimental changes in these characteristics. However, so far no formal link between neutral genetic diversity and the ecological properties of stocks has been established in a realistic model.
- What are the relative strengths of fisheries-induced genetic drift and adaptive fisheries-induced evolution on the evolution of the composition of coding genes in exploited fish stocks? Changes in this composition may result from either mechanism, which complicates the analysis of empirical genetic data. Additionally, po-

tential decreases in genetic diversity are often presented as potentially hampering the adaptive genetic responses of life-history traits. Ecologically and genetically detailed and sufficiently realistic models are needed to predict the strengths of the two mechanisms of genetic change, as well as to develop and test new statistical methods to deal with empirical data.

- What are the links between changes in the composition of neutral genetic markers and coding genes? Because of the ease and cost-effectiveness of carrying out molecular analyses on neutral markers, it would be highly desirable to develop operational tools for inferring some relevant changes in the composition of coding genes in exploited stocks from observed changes in neutral markers. If this were feasible, neutral molecular markers could be used to establish early-warning signals for detecting significant and relevant fisheries-induced evolutionary changes in the composition of coding genes of exploited stocks.

Methods and work plan

To study the effect of size-selective harvesting on the genetic composition of a stock, we will devise a generic individual-based eco-genetic model with allelic inheritance. Below, the model is described in two parts, focusing in turn on the ecological setting and genetic architecture.

Ecological setting

The ecology of fish stocks will be described based on the following ingredients.

To model somatic growth, we will use a generic biphasic growth model based on energy allocation principles developed by Quince et al. (2008), generalizing a closely related model previously proposed by Lester et al. (2004). Unlike Lester et al.'s model, Quince et al.'s model allows the rate of mass acquisition to scale with any power of somatic mass (instead of being fixed at a power of 2/3 as in Lester et al.'s model) and the gonado-somatic index (GSI) to vary with an individual's age. These extensions are important, since the scaling exponent of mass acquisition in fish often exceeds 2/3, and since fish often show an increase in GSI with age. Mass acquisition will be density-dependent and will decrease nonlinearly with population biomass. This means that both somatic growth and fecundity will be affected by density dependence.

The transition from the juvenile to the adult part of the life cycle will be determined by a PMRN. For the sake of simplicity, we will assume a linear PMRN with constant width, described by its intercept, slope, and width.

Fecundity will equal gonad weight divided by the weight of an egg, with gonad weight being derived from the growth model. The number of new individuals recruiting to the population at age 1 will be determined from a Beverton-Holt stock-recruitment function.

Natural mortality will consist of two components: a size-independent mortality due to, for example, diseases and parasites; and a size-dependent mortality nonlinearly decreasing with body size (according to a power function or exponential function), with the decrease primarily being due to the diminished vulnerability of larger fish to predators.

In addition to natural mortality, individuals will be subject to size-dependent fishing mortality. The size-selectivity of fishing will take different shapes, which will allow considering the different selection pressures resulting from different gears: a sigmoid curve will be used to model trawl selectivity, and a normal curve will be used for gillnet selectivity.

We will consider the evolution of three life-history processes: somatic growth, maturation, and reproductive investment, described by a total of five adaptive traits – three traits related to growth and reproductive investment, and two traits related to maturation. The traits describing growth and reproductive investment will be the maximum annual increase in size as determined by energy acquisition, the proportion of acquired energy that is devoted to somatic growth in the first adult year, and the relative annual increase in reproductive investment during adulthood. The parameters related to maturation will be the PMRN intercept and the PMRN slope.

Genetic architecture

We will focus on diploid organisms. The inheritance of adaptive traits will be described either through quantitative characters or through the transmission of alleles according to Mendelian laws. The inheritance of neutral markers will always be described by allelic transmission. Under quantitative inheritance, offspring trait values will vary around mid-parental trait values according to a normal segregation-recombination-mutation kernel. Under allelic inheritance, haploid gametes will be formed during meiosis by independently drawing one of the two homologous alleles at each locus, representing the effect of genetic recombination. Reproduction will occur randomly between pairs of mature individuals, and the fusion of two random gametes will create new offspring.

Under allelic inheritance, each of the five adaptive traits will be controlled by several loci, each of them being polymorphic (i.e., exhibiting more than one allele). The effect of these loci will be additive (neglecting dominance and epistasis), but we will consider two types of distribution of genetic effects across loci: either a uniform distribution of small effects of equal magnitude, thus following the classical assumption of quantitative genetics, or an exponentially decreasing distribution of effects, thus matching empirical results for quantitative trait loci (QTL), where a few major loci have strong effects while many other minor loci have weaker effects. The number of loci and of alleles per locus will be set as model parameters, and we will determine how many loci are needed to achieve stable results. To obtain values of the adaptive genetic traits, allelic values will be summed over all corresponding loci.

Neutral loci will be treated analogously, without affecting the expression of adaptive traits. The number of neutral loci, of alleles per locus, and of their mutation rates will be set in accordance with empirical results.

Work plan

As a first step, we will calibrate the allelic inheritance of adaptive genetic traits by using a uniform distribution of small phenotypic effects across loci, to obtain results similar to those for the quantitative inheritance of adaptive genetic traits.

As a second step, we will study neutral and adaptive evolution under varying fishing selectivity and mortality. We will (i) assess changes in neutral diversity and investigate whether we can find consistent patterns between neutral diversity and the eco-

logical properties of fish stocks, (ii) evaluate the relative contribution of neutral and adaptive fisheries-induced evolutionary changes of life-history traits, and (iii) investigate whether we can identify correlations between changes in a stock's composition of neutral genetic markers and coding genes.

Time permitting, as a third step, we will carry out an analogous investigation using a genetic architecture with exponentially decreasing effects of loci and compare results with the previous one.

Relevance and link to EEP's research plan

This project aims to assess the influence of size-selective fishing on the genetic composition and the ecological properties of commercially exploited stock. It is therefore directly linked to EEP's focus on *Evolutionary Fisheries Management*. In addition, the development of an allelic model with rich ecological detail is a novel contribution to existing applications of eco-genetic models using quantitative inheritance.

Expected output and publications

The results of this work will be included in my PhD thesis and are intended for publication as a co-authored research article in an international scientific journal.

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Food-web evolution in multivariate niche spaces

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Goal

To develop a model of food-web evolution based on multivariate trophic traits and to compare the structural properties of emerged food webs with those of real food webs.

Background and motivation

Recently, the evolutionary emergence of food-web structures has become a focus of mounting attention. One of the first studies addressing this issue is Drossel et al. (2001) who considered species that are characterized by a finite number of features determining their roles as predators and prey. Ito and Ikegami (2006) and Ito et al. (2009) considered one-dimensional niche spaces and applied a reaction-diffusion model to explore food-web evolution. Rossberg et al. (2006, 2008) investigated multivariate foraging and vulnerability traits, and assumed that predation of one species upon another depends on the distance between the former's foraging traits and the latter's vulnerability traits. Brännström et al. (preprint) studied food-web evolution by focusing on adaptations in body size, assuming that larger species can predate on smaller ones. Once in an evolutionarily steady state, the structural properties of model food webs – including the distributions of trophic links and of species abundances – can be compared with empirical data.

Several traits determine the foraging efficiency and vulnerability of a species. These include habitat preferences, body size, foraging behavior, and defensive mechanisms. Therefore, it is natural to consider species defined by their position in a multivariate niche space. Brännström et al (*preprint*) focused on body size as the only adaptive trait: all interactions between species were assumed to depend on that trait. Ito's model examined evolution of a single foraging trait and a single vulnerability trait. Drossel et al. (2001) and Rossberg et al. (2006) considered multiple traits, but assumed that trait values were binary, taking no values other than 0 or 1. These studies assume that population dynamics are governed by deterministic differential equations, which ignores potentially important effects of demographic stochasticity. To date, only Rikvold and Sevim (2007) and Powell and Boland (2009) appear to have studied the effects of demographic stochasticity in evolutionary food-web models, but neither considered the gradual evolution of continuous traits.

Furthermore, Rossberg et al. (2006, 2008) assumed that the evolving community reached a steady state before a further speciation occurs, which cannot represent the simultaneous evolution of species. In addition, some ecologically important phenomena, such as succession and invasion of alien species, do not occur in a community residing in a steady state. Also Yoshida (2008) assumed deterministic population dynamics and the evolution of body size as the only adaptive trait, and then explored the relationships between the duration of food-web evolution and the number of extinctions that are caused by the introduction of alien species.

To further understand the dynamics of emerging structural properties of evolving food webs requires a model that includes multivariate continuous traits, demographic stochasticity, and non-equilibrium dynamics. The aim of this project is to implement such a model from an individual-based perspective.

Research questions

I will try to advance understanding of how ecological, evolutionary, and environmental factors alter the dynamics and outcomes of food-web evolution. To this end, I will create an individual-based model that assumes asynchronous birth and death events based on interactions between individuals in continuous time, where each individual is characterized by two vectors of continuous foraging and vulnerability traits.

I will focus on the following questions:

- What are the effects of niche-space dimensionality, demographic stochasticity, trophic interactions, interference competition, functional responses, and of the cost of extreme traits values on the structure and diversity of emerged food webs?
- Which choices of the aforementioned factors result in food webs whose structural properties agree with empirical data on real food webs?

In the longer term, I will consider the following extension:

- How does the introduction of patch structure affect food-web evolution in metacommunities?

Methods and work plan

Model description

Following previous studies of food-web evolution, I assume that the trophic traits consist of foraging traits and vulnerability traits, each taken from a continuous multivariate niche space. Each individual is assumed to be haploid with nearly-faithful asexual reproduction. All individuals are thus considered to reproduce clonally and to produce mutated offspring with a small probability m. I assume that a mutation alters a trait value by $t' = t + \xi_m$, where t', t, and ξ_m are, respectively, the value of offspring's trait, that of its parent, and a random deviate following a normal distribution with mean 0 and variance σ_m^2. No intrinsic difference between trophic classes – such as plants, animals, carnivores, and herbivores – is assumed (Rossberg 2008; Yoshida 2008). Instead, following Ito and Ikegami (2006), I will assume an external resource with a fixed vulnerability trait v_0. Initially, the population consists only of individuals adapted to forage on this resource.

The evolving community is updated asynchronously. For each update, the birth and death rates of all individuals are calculated. We denote the ith individual's birth event by e_{ib} and its death event by e_{id}. With $\mathrm{N}(x,\sigma) = \exp(-\frac{1}{2}x^2/\sigma^2)$, the rates of these events are given by

$$r_{ib} = ac_F \sum_j C_1(v_i) F_{i\to j} \text{ and}$$

$$r_{id} = c_F \sum_j C_1(v_j) F_{j\to i} + c_I \sum_j \mathrm{N}(f_i - f_j, \sigma_I) + c_d C_2(v_i),$$

respectively, where f_k and v_k are the foraging and vulnerability traits of the kth individual, a represents assimilation efficiency, and d is the intrinsic death rate. The sec-

ond sum in the expression for the death rate above represents the effects of interference competition between predators. The difference $r_{ib} - r_{id}$ measures the per capita growth rate of individual i. $F_{i\to j}$ denotes the functional response, i.e., the predation intensity of species i on species j. I will investigate several forms of this functional response:

(i) $F_{i\to j} = \mathrm{N}(f_i - v_j, \sigma_F)$
(linear functional response),

(ii) $F_{i\to j} = \alpha\,\mathrm{N}(f_i - v_j, \sigma_F)/[\beta + \alpha\sum_k \mathrm{N}(f_i - v_k, \sigma_F)]$
(Holling type-II functional response),

(iii) $F_{i\to j} = \mathrm{N}(f_i - v_j, \sigma_F)/[1 + \alpha\sum_k \mathrm{N}(f_i - v_k, \sigma_F) + \beta\sum_k \mathrm{N}(f_i - f_k, \sigma_C)]$
(Beddington functional response), and

(iv) $F_{i\to j} = \mathrm{N}(f_i - v_j, \sigma_F)/[\beta + \alpha\sum_k \mathrm{N}(f_k - f_i, \sigma_C)\,\mathrm{N}(f_k - v_j, \sigma_F)]$
(ratio-dependent functional response; Drossel et al. 2001).

The parameters σ_F and σ_C, respectively, represent the trophic niche width of each individual and the width of the effect of similarity in foraging traits on reduced predation intensity through interference competition.

We assume that extreme values of vulnerability traits are costly, so that trait evolution toward infinite values will not occur. After a suitable coordinate transformation, the functional form of this cost can be approximated to be radially symmetric with a minimum at the origin. The reduction in assimilation rate, and thus in birth rate, resulting from this cost is measured by $C_1(v_i)$. With $\mathrm{C}(v) = c_c v^2$, we will consider three functional forms: (i) $C_1(v_i) = 1$ (no cost), (ii) $C_1(v_i) = 1/[1 + \mathrm{C}(v_i)]$, and (iii) $C_1(v_i) = \exp[-C(v_i)]$. The increase in death rate resulting from this cost is measured by $C_2(v_i)$. We will consider two functional forms: (i) $C_2(v_i) = 0$ (no cost) and (ii) $C_2(v_i) = \mathrm{C}(v_i)$.

After calculating the birth and death rates for all n individuals in the evolving community, one event $e^* \in \{e_{1b}, \ldots, e_{nb}, e_{1d}, \ldots, e_{nd}\}$ is randomly chosen with probability $\Pr[e_{ix}] = r_{ix}/\sum_{jy} r_{jy}$. If a birth event occurs, one individual is added to the population, potentially with a mutation of its trait values relative to those of its parent. Otherwise, a death event occurs, and the target individual is removed.

To enhance computational efficiency, I will group individuals with the same trait values , and increment such a clone's abundance by 1 upon a birth event without mutation occurring in the clone, and decrement it by 1 upon a death event occurring in the clone. When a mutation occurs, a new clone with an initial abundance of 1 is added to the evolving community. Extinction of a clone occurs when its abundance falls to 0. Also, to increase computational efficiency when calculating interactions between clones, I will partition trophic niche space into cells, so that clones interact non-negligibly only with clones in neighboring cells.

For comparing the evolving food webs with empirical data, I will distinguish species based on cluster analysis, such as the *k*-means method or QT clustering (Heyer et al. 1999). On this basis, I will compare the species-abundance curve, species link distribution, and the maximum trophic level of evolving food webs with previous studies and empirical data, such as that of a Caribbean marine ecosystem (Bascompte et al.

2004). Recently, Kondoh (2008) showed that the distribution of intraguild predation modules in empirical food webs is not random. If time permits, I will try to check the distribution of these modules in the food webs generated by my model.

Work plan

To achieve my goals, I intend to complete the following steps:

- Implement the individual-based model of food-web evolution.
- Using R, convert the model's output into clustered food-web data.
- Compare the emerged food-web structure with that of previous studies. The model should produce results that are qualitatively similar to previous studies.
- By changing parameters and functional forms involved in the model, find settings that produce high species diversity.
- Compare model results with empirical food-web data, such as that from a Caribbean marine ecosystem (Bascompte et al 2004). This comparison is important because a high diversity of modeled food webs does not necessarily mean that they are similar to real food webs. Based on this comparison, model settings will be adjusted.

In future, I intend to extend this model to include migration between multiple patches.

Relevance and link to EEP's research plan

Clarifying the evolutionary dynamics of biodiversity is a main theme of the research project on *Evolving Biodiversity* of the Evolution and Ecology Program at IIASA. Since species in ecosystems strongly interact through prey-predator relationships, to understand the structure of ecosystems we need to understand how trophic networks are created and maintained by evolution. This project aims to develop a basic and extensible model for comparison with other food-web models and empirical data.

Expected output and publications

This work will be included as a part of my Ph.D. thesis, and I intend to publish it as a co-authored article in an international scientific journal.

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Modeling mussel cultivation at Gouqi Island

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Goal

To quantify an economically optimal strategy for mussel cultivation and evaluate its ecological impacts on other parts of the ecosystem.

Background and motivation

The coastal zone of Gouqi Island in the East China Sea is one of the important mussel-cultivation areas in China. The mussel-cultivation catamarans are located close to the island, and a third of the about 2300 local fishermen derive their livelihood from mussel cultivation. It is therefore important to ensure sustainable and stable cultivation. Furthermore, an assessment of the maximum economic yield of cultivated mussels should also take into account the capacity of the ecosystem to support mariculture activities.

Two species of mussels are farmed in this area: an introduced species *Mytilus edilus* is harvest after one year, whereas the less common native mussel species *Mytilus coruscus* grows to larger size while typically being cultivated for two years before harvesting. Fishermen can choose how many juvenile mussels of each species to plant at sea. The commercial value of the annual yield depends on the number and weight of harvested mussels. Their growth is limited by environmental conditions and food concentration. The latter includes particulate organic matter and phytoplankton. Modeling the impact of mussel cultivation on these factors allows predicting mussel growth in dependence on other environmental factors (Cédric et al. 2003). On this basis, optimal stocking densities for mussel cultivation can be determined (Mazouni et al. 1998).

Mussels are mainly phytoplankton grazers (Rouillon et al. 2005). According to an experiment conducted by Chang and Wu (2007), for water depths of 20-30 m and cultivation densities of 3.0-4.5 kg m^{-2}, *M. coruscus* larva can graze 7.75×10^7 phytoplankton cells per liter and day. The weight at age of *M. coruscus* is about 1.5 times as large as that of *M. edulis*. The quantity of food that mussels graze is mainly dependent on their size, and amounts to roughly 5% of their weight per day (Li et al. 1999). Larger mussels will earn more money for the fishermen than smaller ones: sometimes *M. coruscus* can be sold at a price that is four times higher than that of *M. edilus*.

While mussels need enough phytoplankton intake to grow, other species in the ecosystem also feed on this food source: these include zooplankton, some crustaceans, and most juvenile fishes. Other species, in turn, live on zooplankton: these include benthos, some plankton-eating fish, and some polyphagia fish that are predated upon by other fish. Hence, if mussel cultivation reduces phytoplankton density too much, this can cause modifications in food-web structure and functioning. The impact of mussel cultivation on the ecosystem therefore depends not only on the stocking density of cultivated mussels, but also on the ecological interactions within the ecosystem. To assess how the ecosystem is affected by mussel cultivation, it is therefore im-

portant to understand and quantify these ecological interactions. If species at higher trophic levels of the ecosystem – such as fishes, crabs, or cephalopods – lose their ecological positions in the ecosystem, this causes damages not only to the ecosystem, but also to the economic interests of fishermen who live on fishery catches from this ecosystem.

Research questions

In this project, I will try to find the economically optimal strategy for mussel cultivation around Gouqi Island. In addition, as a guide for fishermen and policy makers, I will try to predict the environmental and ecological impacts of mussel cultivation around Gouqi Island. I will focus on the following research questions:

- What is the optimal strategy for mussel cultivation?
- What are the impacts of mussel cultivation on other species in the ecosystem?
- What models and methods should be used to quantify the ecological and production carrying capacity for mussel cultivation?

Methods and work plan

I will address the above questions using two complementary approaches. First, a size-structured model of commercial mussel cultivation will be used to explore the profitability of different mussel-cultivation strategies. This model will help to evaluate which of the two mussel species should be chosen for cultivation and to predict the resultant economic benefits. Second, a model based on the 'Ecopath with Ecosim' (EwE) framework will be used to determine the ecological impacts of mussel cultivation.

Size-structured model of mussel cultivation

Assuming that fishermen plant all spat at roughly the same time of the year, we can represent the planted mussels as a cohort and describe changes in the size s_i and abundance n_i of mussel species i by two ordinary differential equations,

$$\frac{ds_i}{dt} = g_i(s_i, z, T) \text{ and } \frac{dn_i}{dt} = -d_i(s_i, z, T),$$

with $s_i(0) = s_{i,0}$ and $n_i(0) = n_{i,0}$, where $g_i = g_i(s_i, z, T)$ and $d_i = d_i(s_i, z, T)$ are the growth rate and mortality rate, respectively, of a mussel of species i with size s_i when the water temperature is T and the density of phytoplankton is z. The mussels interact through filter-feeding on phytoplankton. I will model the dynamics of the phytoplankton density under the assumption of an inflow from the kelp beds in the coastal area and a constant outflow of water from the area under consideration. Using this model, we will first identify the economically optimal amount of spat $n_{1,0}$ and $n_{2,0}$ of the two mussel species to plant each year. Second, we will consider different harvesting strategies. In particular, we will explore whether partial harvesting can be profitable.

A major challenge will be to choose suitable functions for describing the mortality rate and the growth rate. We will attempt this based on a simple energy-budget model, which will be parameterized from available data on mortality and growth trajectories under different water temperatures and phytoplankton concentrations. If the data

proves insufficient to quantify both dependences, we will either leave out the former dependence or attempt to include it based on general knowledge about the scaling of metabolic rates with an organism's body size and ambient temperature.

Ecopath model of ecosystem impacts

'Ecopath with Ecosim' (EwE) is a software tool for representing the trophic structure of an ecosystem and for providing basic information for the management of fishery resources involving multiple trophic levels (Pauly et al. 2000; Jiang et al. 2005). It can be used to model the trophic relationships among functional groups, to assess flows of biomass among several trophic levels, and to identify other salient ecosystem properties. Using the EwE framework, I will integrate recent survey data and information from published literature (Zuozhi et al. 2008; Hong et al. 2008) to build a food-web model describing the trophic structure and energy flows of the Gouqi Island ecosystem.

We divide the aquatic organisms of the Gouqi Island ecosystem into 17 functional groups. Each such functional group can represent a single species or a group of species. Several species can be grouped if they occur in a common physical habitat and if they have similar food preferences and life-history characteristics. A particular species can be represented separately based on its ecological and economic importance in the ecosystem.

Considering the features of, and interactions among, all functional groups, a balanced model of trophic flows can be constructed (Christensen et al. 2005),

$$B_i(P/B)_i EE_i - \sum_j B_j(Q/B)_j DC_{ij} - Y_i - E_i = 0,$$

where B_i is the biomass of species i, $(P/B)_i$ is the production rate of species i, EE_i is the ecotrophic efficiency of species i, $(Q/B)_i$ is the consumption rate of species i, DC_{ij} is the proportion of species i in the average diet of species j, Y_i is the annual catch of species i, and E_i is the net export of species i through the boundaries of the modeled ecosystem. We intend to integrate into this EwE model as much as possible empirical knowledge on biomasses, production rates, consumption rates, diet compositions, and, if applicable, catch. The data required for calibrating the model originates from survey data, stomach-content analyses, and published literature. The calibration will allow the EwE software to compute the ecotrophic efficiencies (Blanchard et al. 2002). A successful calibration will ensure that all values of ecotrophic efficiency – defined as the fraction of the production of a functional group that is trophically used by the ecosystem – are smaller than 1. Once this is achieved by manual tuning, the automatic balancing routine of the EwE software will be used to complete model calibration through balancing biomass fluxes. This will be followed by a consistency check using physiological criteria, to verify that the respiration/assimilation ratio and the production/respiration ratio are smaller than 1 and that the production/consumption ratio is smaller than about 0.2 for all functional groups.

After balancing the EwE model, we can evaluate the ecological and production carrying capacities for mussel cultivation, based on considering ecosystem responses (in terms of biomass changes, transfer efficiencies between groups and trophic levels, competition, detritus recycling, etc.) to changes in the biomasses and harvest rates of the cultivated mussels. We will also try to determine under which conditions the tro-

phic resources used by the cultivated mussels are available to the extended needed for obtaining good economic benefits.

Relevance and link to EEP's research plan

This project aims to evaluate the ecological and production carrying capacities for mussel cultivation at Gouqi Island, while considering how to minimize the associated environmental degradation. This work not only allows us to develop ideas pertinent to other instances of this unique kind of aquatic ecosystem, but also provide managers with sustainable options for mariculture exploitation. It is therefore linked to EEP's research project on *Evolutionary Fisheries Management*.

Expected output and publications

This work will be a part of my PhD thesis and is intended for publication as a co-authored article in an international scientific journal.

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Evolutionary community assembly with size-structured populations

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Goal

To develop a continuously size-structured population model with two evolving traits describing body size at maturation and ecological niche, to explore conditions under which species can diversify in these traits, and to examine the resultant multi-species communities.

Background and motivation

To date, two categories of evolutionary food web models with explicit population dynamics have been investigated. The first comprises physiologically unstructured population models in which individuals are characterized by one or more evolving traits, while the other comprises continuously size-structured population models in which individuals are characterized by size at maturation as the single evolving trait. While these two modeling frameworks already reach a relatively high degree of evolutionary and ecological realism, they still have important limitations.

On the one hand, species in unstructured population models are usually represented by aggregate variables such as total abundance, density, or biomass. When these models describe predation, food intake causes an immediate increase in abundance or biomass that, again immedeiately, contributes to reproduction. In reality, these effects are delayed through gradual growth and through the need for individuals to mature before being able to reproduce.

On the other hand, in continuously size-structured population models (Andersen & Beyer 2006; Pedersen et al. 2009), species are characterized only by their size at maturation, which is assumed to be proportional to their maximally attainable body size. Here, size at maturation is assumed to be the most important determinant of a species' ecological niche. In comparison with unstructured population models, these models are more realistic by describing continuous size structure. However, a major limitation of these models is that they are unable to explain the coexistence of species with comparable sizes at maturation.

An intermediate approach is to divide populations into several discrete stages, such as juveniles and adults, and then to describe transitions between such stages. In this context, delay differential equations help taking into account the time required for transistions between stages. However, the introduction of discrete stages is a rather drastic simplification of life histories. It is therefore desirable to model gradual changes in individual physiology and life history. Here gradual changes in body size are key, since body size has implications beyond determining an individual's trophic position, because of its significant role in determining the type and strength of ecological processes occurring within a community. Additionally, most life-history processes – including food diet selection, foraging-behavior, growth, maturation,

reproduction, mortality etc. – are well described as functions of body size (Peters 1983; Ebenman & Persson 1988; Brown et al. 2004).

In continuously size-structured population models, it is then further desirable to involve more than one niche trait. In order to distinguish among species with approximately the same body size, Roy et al. (2008) introduced an evolving trait describing an individual's ecological niche in addition to one describing its asymptotic size. This extends previous work by Brännström et al. (preprint) and Loeuille & Loreau (2005), which investigates how evolution of a body-size trait can lead to diverse food webs. Roy et al. (2008) found that the evolving communities do not generally reach an evolutionarily stable state, but evolution instead proceeds in complex non-equilibrium patterns of diversification and extinction. This contrasts sharply with results found for food webs evolving in terms of a single trait, which normally reach an evolutionarily stable endpoint.

To better understand evolutionary community dynamics with diversification and extinction, I will synthesize and extend the two categories of models described above, by developing and investigating a continuously size-structured population model with two evolving traits, with the first one describing an individual's ecological niche and the second one describing its size at maturation.

Research questions

The main questions to be addressed in this project concern the evolutionary dynamics of community assembly of continuously size-structured populations with a body-size trait and a niche trait. In particular, three questions will be examined:

- What conditions enable diversification of a single-species to a multi-speices community?
- What is the eventual outcome of such diversifying evolution?
- What relationship, if any, exists between the initial and final diversification?

If time allows, I will also explore the influence of continuous size structure on the evolution of a community's maximum trophic level.

Methods and work plan

To investigate these questions, I will first develop and implement a continuously size-structured population model in which each species is characterized by its size at maturation and its niche trait.

I will assume that large individuals consume smaller ones, if their niche traits are in the vicinity of that of the predators, and that species with similar body sizes interact through interference competition. The dynamic behavior of species abundances is governed by the growth rate, the fecundity rate, as well as by the mortality rates caused by starvation, predation, and interference competition.

The growth rate, fecundity rate, and the rate of starvation mortality are determined by the energy derived from food intake. This energy will first be used for metabolism and then for reproduction. Additional mortality results from trophic interactions among species, i.e., from the predation by larger individuals on smaller individuals (including those of their own species, through cannibalism), and from interference competition. Finally, we assume that there is one primary resource (such as

zooplankton) that is subject to logistic growth and that is normally distributed along the niche-trait axis.

With these assumptions, population dynamics are governed by the following partial differential equation of transport type,

$$\frac{\partial}{\partial t}N_i(t,m)+\frac{\partial}{\partial m}(g_i(m\sum_j a_{ij}N_j(t,m)N_i(t,m))=-\mu_i(m\sum_j b_{ij}N_j(t,m))N_i(t,m),$$

where the sums are taken over all extant species. The boundary condition describes recruitment,

$$R_i(t)=g_i(m_0,\sum_j a_{ij}N_j(t,m_0))N_i(t,m_0))N_i(t,m_0),$$

where N_i is the density of species i $(i=1...N)$. The latter equation means that the total reproduction rate R_i equals to the total growth rate g_iN_i of new recruits with body size m_0. The growth rate g_i is given

g_i = food intake – metabolism – reproduction.

The mortality rate μ_i comprises the losses from starvation, predation, and interference competition.

The coefficients a_{ij} and b_{ij} are obtained as functions of the niche–trait value and of the size at maturation of species i and j.

The gradual evolutionary dynamics of these traits in each species will be described by the canonical equation of adaptive dynamics (Dieckmann and Law 1996). Extinctions occur when a species' density falls below a given low threshold value without rebounding. Diversification through evolutionary branching may take place at evolutionarily singular strategies, where the gradient of a species fitness landscape vanishes. The emergence of a new species is reflected by adding an additional partial differential equation for its population dynamics and two additional differential equations for its evolutionary dynamics.

The model will be implemented and studied in Matlab.

Relevance and link to EEP's research plan

The project seeks to establish insights into the ecological diversification of continuously size-structured populations involving a body-size trait and a niche trait. This extends ongoing research on the evolution and assembly of food webs in the EEP project on *Evolving Biodiversity* and advances our understanding of the mechanisms and processes underlying biodiversity changes in nature.

Expected output and publications

The work is expected to result in one or more coauthored publications in international scientific journals.

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