

Young Scientists Summer Program 2005

Seven Research Projects

**Adaptive Dynamics Network
International Institute for Applied Systems Analysis
Laxenburg, Austria**



Emergence of Influenza A

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Goal

To investigate the ecological and evolutionary factors contributing to the emergence of influenza A viruses in different species of hosts.

Background and motivation

Many infectious diseases emerge by increasing their host range; that is, they cause one or more outbreaks in a new host population. The probability of an emergence event is influenced by ecological and evolutionary factors. Disentangling the relative contribution of each factor has important consequences for disease management, including gauging the risk of spillovers, predicting the severity of outbreaks, and controlling virulence. Many analyses infer the conditions for emergence from an ecological framework, such as SIR models for multispecies communities (e.g. Dobson & Foufopoulos 2001), or by using evolutionary optimization models (e.g. Parker et al. 2003). Thus, they implicitly assume either no or very rapid adaptation to changing ecological conditions. Other studies have shown, however, that these kinds of assumptions can lead to substantial underestimates of disease burden (Koelle et al. 2005).

These assumptions are also unlikely to lead to sufficient descriptions of parasites that, like influenza A viruses, evolve rapidly but not instantaneously in variable host communities. In aquatic birds, their natural reservoir, influenza A viruses achieve high transmissibility with negligible fitness costs: they are considered “optimally adapted” and in evolutionary stasis (Ito & Kawaoka 1998). Their dynamics in poultry, swine, and humans are more complicated (Webster et al. 1992; Webby & Webster 2001). Though the molecular determinants of host range, transmissibility, and virulence are incompletely known (Baigent & McCauley 2003), a major determinant of a virus’s ability to infect a host cell is its receptor preference. The intestinal cells of aquatic birds have sialic acid receptors linked to galactose units in an $\alpha 2,3$ conformation, and humans have these receptors in an $\alpha 2,6$ conformation. Viruses in each population show a strong preference for their host’s receptor type. The cells of chickens and pigs have receptors in both conformations, allowing them to be infected by viruses adapted to ducks and humans (Scholtissek et al. 1998; Gambaryan et al. 2002a; Gambaryan et al. 2002b). Thus, chickens and pigs may serve as key intermediate hosts by allowing reassortment between antigenically novel subtypes from the natural reservoir and subtypes that have adapted to replication and transmission in other hosts. These reassortment events have preceded the emergence of most pandemic influenza viruses, which escape host immunity through acquisition of foreign surface proteins (Webster & Hulse 2004).

There is evidence of frequent transmission of influenza viruses between different species, especially in farms and markets in Asia (Banks et al. 2000; Lin et al. 2000; Peiris et al. 2001; Bridges et al. 2002; Liu et al. 2003a) and in commercial poultry and swine operations in Europe and North America (Castrucci et al. 1993; Claas et al.

1994; Olsen et al. 2002; Enserink 2004). These settings provide distinct interspecific transmission opportunities, which are shaped by host population dynamics, the mode of transmission, host immunity, seasonal migration, and vaccination history. The fitness of an invading strain with a particular receptor preference is thus highly contingent on local ecology. An analytic and quantitative framework to study the interplay of these ecological processes with fundamental evolutionary adaptations could be useful in understanding the long-term dynamics of influenza viruses and other zoonotic RNA viruses (Cleaveland et al. 2001; Webster & Hulse 2004).

Research questions

The primary focus of this research is to compare the expected conditions for emergence in different hosts between (1) a simple multihost SIR model of influenza's ecology; (2) an adaptive dynamics model for one subtype, constrained by the tradeoff in receptor preference; and (3) an adaptive dynamics model allowing reassortment among multiple subtypes under the same constraints in receptor preference. Secondary questions to be asked of all models include:

- How does each species contribute to the probability of outbreaks in other species? In a purely ecological model, this question amounts to investigating the ecological force of infection. In an adaptive dynamics simulation, this ecological force of infection is mediated by the extent and direction of adaptation in each host species (for models with one subtype) and the contribution of each species to reassortment events, i.e., by subtype donation or supplying cells where reassortments occur (for models with multiple subtypes).
- How does the strength of the tradeoff between preference for one receptor and probability of infecting cells with other receptors affect results?

Examining the sensitivity of the above dynamics to herd size and vaccination practices could also yield important insights to disease management. Another trait potentially worth investigating is virulence (Baigent & McCauley 2003).

Methods and work plan

I will first explore predictions for emergence by modeling influenza's ecology without evolution, using ordinary differential equations to represent dynamics on rural farms and markets in south-eastern China. The initial model will consider five host classes: wild waterfowl, domesticated free-roaming ducks, chickens, pigs, and humans. Intraspecific transmission will follow a SIS model for waterfowl, including ducks, and SIRS for other classes. For hosts with permanent immunity to a particular strain, such as humans, the R-to-S transition implicitly approximates antigenic drift as a gradual decay of immunity. The model will include parameters for rates of intraspecific transmission (β_{ii}), birth, death, recovery, decay of immunity; and static host-specific parameters such as innate susceptibility, infectiousness, and disease-induced mortality. The interspecific transmission rate β_{ij} is the number of contacts that an infected individual of species j has with susceptible individuals of species i per unit time, multiplied by the probability that contact results in infection. The probability that contact results in infection depends on receptor compatibility, host-specific susceptibility, host-specific infectiousness, and the mode of transmission. Most of these parameters will be estimated with data from farms and markets in southeastern China and multihost challenge experiments (Alexander et al. 1986; Kida et al. 1994; Alexander 2000; Gambaryan et al. 2002a; Bulaga et al. 2003; Liu et al. 2003b; Cheng

et al. 2004). This model will yield a preliminary measure of outbreak probability (measured by R_0) and forces of infection. These values are given by the dominant eigenvalue and summed rows and columns of the modified “Who Acquires Infection from Whom” transmission matrix (Dieckmann et al. 1990).

To explore the ecological and evolutionary dynamics in tandem, I will develop an individual-based simulation that allows the transmission rates β_{ii} and β_{ij} to change as a function of evolving receptor preference. Preference for a receptor type can be described as a continuous quantitative phenotypic trait with minimal genetic variance, following the hypothesis that viral preference evolves along a “spectrum” of $\alpha_{2,3}$ and $\alpha_{2,6}$ receptor types in hosts (Gambaryan et al. 2002a). Preference for cells with one receptor type necessarily involves a decreased ability to infect cells with other receptor types, thereby constraining trait space and host range. This model will use an exact stochastic approach for the SIR and evolutionary components (Gillespie 1976). The effects of ecological parameters on the frequency and size of outbreaks in different host species will be explored. The individual-based setup of the model may also require extrapolation of results to larger population sizes.

In modeling multiple subtypes, each strain will be described by one of 16 forms of hemagglutinin, one of nine forms of neuraminidase, and its receptor preference. Reassortment events occur stochastically in hosts infected with multiple subtypes. The model will track individual hosts’ immunity to particular hemagglutinin and neuraminidase antigens. Outbreaks occur following appearance of new subtypes (i.e., hemagglutinin-neuraminidase combinations) in a host population, provided R_0 exceeds unity. Particular attention will be paid to the sensitivity of the results to tradeoff strength and rates of reassortment, interspecific contact, and immune decay, which are the least understood aspects of influenza’s ecology and evolution. The model can be extended to consider cross-immunity between subtypes, e.g. H9N2 and H5N1 in poultry.

To the extent feasible and time permitting, I will also explore simplifications of these models. Retaining essential stochastic processes, such as interspecific contact, reassortment, and possibly the evolution of receptor preference, while treating population dynamics deterministically might recover key results of the individual-based, wholly stochastic approach.

Relevance and link to ADN’s research plan

This research addresses central questions in virulence management (Dieckmann et al. 2002), which is one of ADN's research foci.

Expected output and publications

This work is intended for publication as a jointly authored research article in a scientific journal. It will also be included in research presented for my preliminary examinations and dissertation.

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Fisheries-induced Evolution in Northeast Arctic Cod

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Goal

To calibrate and apply an individual-based, eco-genetic model to study fisheries-induced evolution in Northeast Arctic cod.

Background and motivation

The Northeast Arctic (NEA) cod stock is currently the world's largest stock of Atlantic cod (*Gadus morhua*), and sustains both large open-ocean trawling fisheries (mainly from Norway and Russia) as well as fishing with conventional gear on the Norwegian coast (Nakken 1998). The fisheries can change the cod's age and size at maturation in two ways: first, by reducing the stock's biomass, which leads to faster individual growth (Heino and Godø 2002) and thereby to earlier maturation through phenotypic plasticity, and secondly, by altering the selective pressures so that the stock evolves towards maturing at a smaller size and younger age through a shift in its maturation reaction norm (Heino et al. 2002a; Barot et al. 2004; Olsen et al. 2004). As a result, NEA cod's median age at maturity has decreased from >10 years in the 1940s to 6-7 years in the 1980s and 1990s (Godø 2000), probably as a result of both the aforementioned processes. In addition, temperature variability has influenced the individual growth of NEA cod in the past (Godø 2000), and is expected to be further shaped by global climate change in the future (Brander 1994; Brander 1995). These changes in maturation dynamics influence various components of the cod-fishing fleet differently: for example, open-sea trawlers capturing young and small cod will be affected differently from conventional fishing boats targeting mature cod.

Besides being economically important, NEA cod is ecologically important because it is the main fish predator of the Barents Sea (Bogstad et al. 2000). By the age of 3-4 years, NEA cod consume large amounts of post-larval capelin (*Mallotus villosus*) and follow the spawning migration of capelin to the coasts of Northern Norway and Northwest Russia in spring (Hjermann et al. 2004a; Hjermann et al. 2004b). At maturation, NEA cod migrate from the Barents Sea to spawn along the Norwegian coast in March-May (Bergstad et al. 1987). The eggs and larvae are carried by currents into the southern Barents Sea and south and west of Svalbard. The immature cod make seasonal migrations whose extent increases with age (Hjermann et al. 2004b). Length growth has been found to be influenced by the abundance of capelin (Bogstad and Mehl 1997).

To fully understand the impacts of fishing on the life-history traits of a population, it is important to distinguish between plastic and genetic responses (Rijnsdorp 1993). One method of disentangling plastic from genetic responses is to examine trends in a maturation reaction norm. A reaction norm describes the range of phenotypes produced by a genotype under different environmental conditions (e.g., Roff 1992). Previous work has shown that mortality can influence growth and maturation in several fish populations (Reznick et al. 1996; Conover and Munch 2002). As growth rates may vary plastically in response to the environment, age and size at maturation

may also vary with growth rates in a plastic way. Within a population, this range of growth rates and the subsequent range in size and age at maturation determine the observable maturation reaction norm (Stearns and Koella 1986). Hence, a reaction norm for age and size at maturation illustrates the maturation schedule of a genotype under different growth conditions. Extending the original deterministic notion of maturation reaction norms (Stearns and Koella 1986), probabilistic maturation reaction norms are defined by the probability that immature individuals at a given age and size will mature during a given time interval (Heino et al. 2002b).

Commercial fishing can be size-selective because larger members of a stock are often targeted and removed by the fishery (Law 2000). Size-selective fishing mortality can act on the ages and sizes at maturation and cause the maturation reaction norm of a population to shift away from its original position. Such a shift may be genetic because the reaction norm itself is genetically determined (Olsen et al. 2004). Alternatively, fishing may alter the somatic growth rates of a population through its impact on population density, thereby shifting the ages and sizes at maturation along the reaction norm. Such a response is plastic because the ages and sizes at maturation are shifting in direct response to changing growth rates (Heino and Godø 2002). Several recent studies have shown that shifts in maturation reaction norms can be rapid (Grift et al. 2003; Barot et al. 2004; Olsen et al. 2004). Therefore, estimating maturation reaction norms can help disentangling plastic and evolutionary changes in the age and size at maturation (Heino et al. 2002b). This is important from a management perspective, as genetic changes are bound to be more difficult to reverse (Law 2000). Genetic changes in life-history traits thus give rise to concerns (Olsen et al. 2004) – in particular, since the consequences of fishery-induced evolution can result in lower sustainable yields (Law 2000; Conover and Munch 2002) and reduced stock stability (Ottersen et al. 2005). Relaxing the fisheries-induced selection pressures may halt the rapid evolution of key life-history traits in harvested populations, and is thus an important target for the management of fisheries-induced evolutionary change.

In this project, my aim is to study the effects of commercial fishing on the evolution of maturation, individual growth rate, and reproductive investment by using an individual-based eco-genetic model (Dunlop et al. 2005b). As mentioned previously, fishing is expected to cause a downward shift in the maturation reaction norm towards younger ages and smaller sizes at maturation (Heino et al. 2002a; Olsen et al. 2004). Fishing may also cause evolution of individual growth rate. For example, experimental harvest of Atlantic silversides resulted in rapid evolution of slower growth rates, since slower-growing individuals were more likely to escape size-selective harvest before reaching maturity (Conover and Munch 2002). However, fast-growing individuals may suffer a survival cost because they forage more actively and thereby are exposed to predators and fishing gear (Heino and Godø 2002). Harvest may also influence patterns of reproductive investment. High mortality selects for greater reproductive investment (Reznick and Ghalambor 2005), but greater investment into reproduction may also incur a cost. Furthermore, fishing is expected to alter traits affecting behavior and morphology (Heino and Godø 2002), but such effects are beyond the scope of this study.

An individual-based modeling approach will be chosen for this study because it allows an intuitive merging of genetics and demography, both of which are important in the context of fishing-induced change (i.e., Chambers 1993; Jager 2001; Dunlop et al. 2005b;). An individual-based approach also allows for modeling maturation as probabilistic, which is likely more realistic given the inherent stochasticity of the

maturation process (Heino et al. 2002b).

Time permitting, an important extension of the individual-based model will be to consider the effects of climate fluctuations and climate change. This is in light of the large changes in the Barents Sea climate predicted to occur towards the middle of the 21st century.

Fishery-induced life-history changes may alter the economic conditions of the cod fisheries and lead to changes in the fleet's structure and allocation; this in turn may either contribute towards enhancing or diminishing the ongoing changes. Optimal management strategies will be affected. As far as we know, this topic has not been treated in the bio-economic literature on NEA cod. Time permitting, this project will therefore aim at incorporating the fishery's effect on the evolving traits into a bio-economic model, in order to quantify the long-term cost of overfishing. I will study how the costs and benefits of different fishing strategies may change by using an existing, semi-spatial bio-economic model in which both the state (effort and allocation) of the fishing fleet, as well as the state of the cod stock, are dynamic variables, mutually affecting each other.

Research questions

I will use an individual-based model to predict how fishing influences the evolution of growth, reproduction, and maturation of NEA cod. The considered evolving traits include the immature somatic growth rate (i.e., the fraction of energy devoted to growth prior to maturation), the reproductive investment (i.e., the gonado-somatic index), and parameters describing the maturation reaction norm of NEA cod. A previously developed individual-based model by Dunlop et al. (2005) focused on evolution of the maturation reaction norm alone. Research suggests that other traits such as growth (Conover and Munch 2002) and reproductive investment (e.g., Roff 1992) might also be affected by fisheries-induced evolution. In addition to characterizing the magnitude and rate of fisheries-induced evolution in NEA cod, we plan to evaluate how different management strategies alter the evolutionary response. We also plan to assess the impact of the modeled evolutionary changes on yield and recovery potential. Finally, by introducing environmental fluctuations into the model, we may evaluate the impact of the evolutionary changes on stock stability.

Time permitting, I will enhance the developed model by adding a bio-economic component or by interfacing it with an existing bio-economic model. Also the effects of climate change may be examined.

Methods and work plan

I will use an individual-based model to examine the evolution of five quantitative traits: intercept of the maturation reaction norm, angle of the maturation reaction norm, width of the maturation reaction norm, gonado-somatic index, and fraction of energy devoted to growth prior to maturation. This model will be expanded from the existing model by Dunlop et al. (2005) and specifically parameterized for the NEA cod. The model will be run on yearly time steps with the processes of maturation, reproduction, growth, and mortality occurring on an annual basis. Growth will be assumed as density-dependent and linear prior to maturation. Following maturation, a component of the available energy will be devoted to reproduction. Maturation status will be based on an individual's probabilistic maturation reaction norm, and reproduction will occur randomly between pairs of mature individuals. The evolving traits will be passed on to offspring in dependence on the two parental trait values using a normal recombination kernel. Mortality of newborns will be determined

through a Beverton-Holt stock-recruitment function (Hjermann et al. in prep.). In all simulations, a constant level of age and/or size-specific natural mortality will be applied. A tradeoff between juvenile growth rate and survival will also be included. Realistic fishing mortality rates will be applied and their effect on the evolution of traits will be examined. The model will also include the effects of temperature on recruitment and length growth.

The individual-based model will be parameterized from existing data on NEA cod. When parameterizing the model, I will take into account important sources of varying natural mortality in NEA cod: climate-linked mortality at the larval stage (Sundby 2000) and cannibalism (linked to the abundance of capelin) for 1-3 year old cod (Hjermann et al. 2004a; ICES 2004). I will use data on length, weight, maturation, and abundance collected from research surveys in the Barents Sea (the cod's feeding grounds) and in the Lofoten region (the cod's spawning grounds) conducted by the Institute of Marine Research (Bergen) and by the Polar Research Institute of Marine Fisheries and Oceanography (Murmansk), available from the ICES report of 2004. Several existing laboratory studies, mesocosm studies, and field studies on growth and maturation (Suthers and Sundby 1993, 1996; Svasand et al. 1996; Suthers et al. 1999; Clemmesen et al. 2003; Godø 2003; van der Meeren and Moksness 2003) will also be considered.

The first steps in examining the parameterized model will focus on viable management strategies that reduce or slow potential fisheries-induced evolution. Preliminary re-search suggests that increasing the minimum size limit or implementing protective slot limits will significantly slow down evolution of the maturation reaction norm (Dunlop et al. 2005a).

The final stage of the project will be to assess the economic impacts of fisheries-induced evolution. I will use the results from the individual-based eco-genetic model and include estimates on maturation and individual growth as parameters in an existing bio-economic model for the NEA cod in the Barents Sea, developed at the Centre of Ecological and Evolutionary Synthesis (Oslo). On this basis, I can study how management strategies might change in light of the economic analyses. The fishery is modeled from the perspective of a resource manager who is interested in comparing the effects of different fishing strategies on the overall utilization of the stock. The manager has two main controls: total amount of effort and (ii) age selectivity of harvest, that is where and how effort is expended, with respect to impacts on different age classes. The spatial distribution of effort has an age selective effect where fish of different sizes are located in different regions: Older fish migrate to spawn in the Lofoten area, while younger fish remain in the Barents Sea or along the coast of northern Norway. During the summer, younger fish are also typically located further east and north in the Barents Sea. The combination of gear type and spatial distribution of effort determines the overall extent to which fish of different age groups are targeted. The economic model has no fleet structure, and cost and income are driven by fishing effort. Given different fishing strategies, the model can estimate cost and income. The first (and simplest) option is to use results from the evolutionary model as input to the bio-economic model, to analyze how profit and optimal fishing strategy are affected by changes in the cod's life-history parameters. The second option is to link the eco-genetic model to the bio-economic model, so that for every year in the simulation the output from the eco-genetic model (e.g., length at maturation) is used as input to the economic model, whose output (e.g., fishing mortality as a function of length) is used, in turn, as input for the eco-genetic model.

Relevance and link to ADN's research plan

This project aims to model the evolutionary effects of fishing in NEA cod and thus directly links to ADN's research focus on Fisheries-Induced Adaptive Change.

Expected output and publications

This work is intended for publication as a co-authored paper in an international scientific journal, and is expected to be integrated as a chapter into my PhD thesis.

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Evolution of Dispersal Kernels

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Goal

To investigate the evolution of dispersal kernels in the spatial logistic model, driven by either inter- or intraspecific competition in continuous space and time.

Background and motivation

From an individual's perspective, dispersal may be motivated by a number of (not mutually exclusive) reasons: avoiding competition for resources (either inter- or intraspecific) (Lambin et al., 2001), minimizing kin competition (e.g. Hamilton & May, 1977; Comins, 1982; Frank, 1986; Kisdi, 2004), avoiding inbreeding (Perrin & Goudet, 2001), or coping with the temporal variability of resource availabilities (Levin et al., 1984; Travis & Dytham, 1999; Gandon & Michalakis, 2001). Generally, dispersal is favoured as long as individuals have a higher inclusive fitness when they move away from their natal habitat (Frank, 1986; Metz & Gyllenberg, 2001; Poethke & Hovestadt, 2002; Dytham, 2003). Law & Dieckmann (2000) and Law et al. (2003) showed that the "individual's-eye view" is essential in modeling and analyzing the spatial dynamics resulting from intra- and interspecific interactions. These interactions result in spatial patterns of species abundance, which develop according to the individuals' competitive abilities (Law et al., 2003). When the landscape is assumed to be homogeneous in space and time, it is only the abundance and spatial distribution of individuals that define the heterogeneous environmental conditions to which the potential for spreading adapts. This eco-evolutionary feedback results in a highly dynamic fitness landscape that exerts the selective pressures driving the evolution of dispersal traits. In the modeling approach of (Dieckmann & Law 2000), dispersal kernels are used for describing the propagation of individuals. Such kernels are defined as probability densities, with their shape determining the relative spatial distribution of an individual's offspring around its parent. Evolution in the shape of dispersal kernels in a dynamically changing environment has not been investigated to date. Any approach in this direction will have to account for the fact that, without any costs of dispersal, the evolving distances are likely to escalate evolutionarily, since this minimizes kin competition and maximizes the chance for escape from unfavorable environmental conditions -- like a highly crowded neighbourhood (Leturque & Rousset, 2002). It is therefore important to either incorporate costs of dispersal, or to reasonably limit dispersal ranges.

As we investigate the evolution of dispersal, we utilize function-valued traits, measuring the amount of dispersal over a continuum of relevant distances. Function-valued traits give a very detailed description of adaptive features in biological organisms. However, directly following the resultant stochastic dynamics of function-valued traits through individual-based simulations is a fairly complex undertaking. Therefore, the formal link between the individual-based ecology, which is fast and stochastic, and a description of the expected evolutionary process, which is slow and deterministic, has to be carefully constructed (Dieckmann et al., 2005), in order to arrive at simplified models. In this context, particular attention must be devoted to the parameterization of function-valued adaptive traits and the formulation of their

evolutionary constraints. While striving for parsimony, an exaggerated parameter-reduction can lead to spurious results because then the evolutionary dynamics can easily get trapped in local optima since low-dimensional parameterizations are prone to inhibit so-called 'extra-dimensional bypasses' (Conrad, 1990).

The modeling of small-scale interactions has already led to a better understanding of spatial population dynamics (Law et al., 2003), by enabling, e.g. species coexistence where mean-field approximations predict the extinction of less competitive species (Law & Dieckmann, 2000). Incorporation of adaptive dynamics into such models will result in an even more realistic picture of spatial population dynamics

Research questions

I will try to find out, whether there are evolutionary attractors of dispersal kernel shape, and, given such attractors are found, how these depend on the competition regime considered in the model. For the sake of simplicity, I will concentrate on systems consisting of one and (time permitting) two species with different competitive abilities.

Methods and work plan

The framework of adaptive dynamics of function-valued traits offers a suitable method for investigating the course and outcome of long-term evolution of dispersal kernels under the assumption of asexual inheritance (Dieckmann & Law, 1996; Dieckmann & Ferrière, 2004; Dieckmann et al., 2005). The population dynamics driving the eco-evolutionary process will incorporate resource competition owing to local intra- and interspecific competition. Such competitive interactions can be described by spatial logistic models in continuous space and time (Law et al., 2003). I will implement two individual-based models describing the polymorphic stochastic dynamics of populations with either scalar-valued or function-valued traits, which determine the shape of the corresponding dispersal kernels. To be able to identify evolutionary attractors in the shapes of dispersal kernels, it is helpful to gain a deterministic description of the same population dynamics. Therefore, it is necessary to reduce the polymorphic stochastic models (*PSM*) to corresponding monomorphic deterministic models (*MDM*). Altogether there are four models, which I describe in the following:

1. *Polymorphic stochastic model with scalar-valued adaptive traits (PSM^s)*
To derive an eco-evolutionary population dynamic with the full spectrum of stochasticity resulting from interacting and mutating individuals, I will implement an individual-based model based on spatial logistic equations for species interactions similar to the approach of Law et al. (2003). In this step, I use a bivariate Gaussian function as dispersal kernel,

$$m_i(x - x') = \frac{1}{M} \exp\left(-\frac{|x' - x|^2}{2(S_{m_i})^2}\right). \quad (1)$$

This function depends on a scalar-valued trait, as its shape is given by only one parameter: the standard deviation S_{m_i} , which denotes the trait value of the individual of species i in focus, and determines the kernel's width.

By considering natal dispersal, the dispersal kernel is directly linked to the probability for an individual of type i , located at x , to give birth to an offspring at x' : $B_i(x, x') = b_i m_i(x - x')$, with the intrinsic birth rate b_i .

Mutations generate variability with a probability μ_i per birth event and mutant trait values are obtained by adding a mutation effect drawn from a Gaussian distribution with mean 0 and given (small) standard deviation.

Competition affects the probability that an individual of species i at location x dies,

$$D_i(x, p) = d_i + \sum_j d'_{ij} \int w_{ij}(x' - x) [p_j(x', t) - \delta_{ij} \delta_x(x')] dx', \quad (2)$$

with the interaction kernel

$$w_{ij}(x - x') = \frac{1}{W} \exp\left(-\frac{|x' - x|^2}{2(S_{w_{ij}})^2}\right). \quad (3)$$

The kernels are normalized so that their integrals over $x' - x$ are equal to 1, with M and W being the normalization constants. d_i denotes a species' intrinsic death rate, whereas d'_{ij} defines the strength of influence of either intraspecific ($i = j$) or interspecific ($i \neq j$) competition of species j on i . $S_{w_{ij}}$ indicates the characteristic range of competitive influence of species i on j . The local density of individuals of type i at point x' is denoted by $p_i(x', t)$, and the expression $\delta_{ij} \delta_x(x')$ removes the individual of type i at x , because it does not compete with itself (Law et al., 2003).

2. *Polymorphic stochastic model with function-valued adaptive traits (PSM_f)*
The description of the birth process in this step is similar to the one above, but with the function-valued trait $m_i(a)$, which measures the amount of dispersal over a distance $a \in [0, L]$; $L > 0$, L being the upper limit for dispersal distances. To facilitate the implementation of this model, the function-valued traits will be reasonably discretized into a histogram of distance classes wherein mutations result in shifts in the dispersal amplitude of a distance class. More refined mutation models (based on explicit variance-covariance functions) may be considered later.
3. *Monomorphic deterministic model with scalar-valued adaptive traits (MDM_s)*
Assuming the ecological and the evolutionary timescale to be sufficiently separated, we regard each species within the population as monomorphic. Thus, selection has enough time to take effect before a new viable and potentially advantageous mutant replaces a resident trait: $m_i \rightarrow m'_i$ (Dieckmann et al., 2005). To devise a spatially implicit version of our model, we use $C(\xi)$, the second spatial moment, which describes how pair densities vary with the distance ξ between a pair's members (Dieckmann & Law, 2000).

In the following, index 1 denotes the resident species with trait value S_m and 2 the corresponding mutant species with trait value S'_m . The differential equations describing the population dynamics of densities N_i in the resident and mutant populations are given by

$$\begin{aligned} \frac{d}{dt} N_i &= (b_i - d_i) N_i \\ - \sum_j d'_{ij} \int w_{ij}(\xi) C_{ij}(\xi) d\xi, \quad i, j \in \{1, 2\}. \end{aligned} \quad (4)$$

The dynamics of pair of densities $C_{ij}(\xi)$ is given by

$$\frac{d}{dt} C_{ij}(\xi) = +\delta_{ij} b_i m_i(-\xi) N_i \quad (5a)$$

$$+ b_i \int m_i(\xi') C_{ij}(\xi + \xi') d\xi \quad (5b)$$

$$- d_i C_{ij}(\xi) \quad (6a)$$

$$- \sum_k d'_{ik} \int w_{ik}(\xi'') T_{ijk}(\xi, \xi'') d\xi'' \quad (6b)$$

$$- d'_{ij} w_{ij}(\xi) C_{ij}(\xi) \quad (6c)$$

$$- \sum_k d'_{ik} \int w_{ik}(\xi'') T_{ijk}(\xi, \xi'') d\xi'' \quad (6d)$$

$$- d'_{ij} w_{ij}(\xi) C_{ij}(\xi) \quad (6e)$$

$$- |m_i| C_{ij}(\xi) \quad (7a)$$

$$+ \int m_i(\xi') C_{ij}(\xi + \xi') d\xi' \quad (7b)$$

$$+ \langle i, j, \xi \rightarrow j, i, -\xi \rangle, \quad i, j \in \{1, 2\}, \quad (7c)$$

with the contribution of birth events (5), death events (6), and movement events (7) to changes in $C_{ij}(\xi)$. The term $\langle i, j, \xi \rightarrow j, i, -\xi \rangle$ accounts for the fact that all birth and death events do not only occur to an i individual, but can also happen to the j individual of the ij pair: it is shorthand for all preceding terms after changing i to j , j to i and ξ to $-\xi$. The moment closure needed for the triplet density T_{ijk} in (6b) and (6d) will be a power-2 closure (Murrell et al., 2004).

There are three time scales present in the invasion dynamics of a mutant in an established resident population: the slowest one is the development of the resident population (N_1, C_{11}), which is considered to be at equilibrium. The intermediate timescale is the progress of the mutant population size (N_2), and the fastest development occurs in the spatial distribution of mutants (C_{12}, C_{22}). Thus, each iteration of the model consists of three steps: After calculating the resident dynamic, the equations for C_{12}, C_{22} can be solved, assuming them to be at a local pseudo-equilibrium, to calculate the mutant population's per capita growth rate $\frac{1}{N_2} \frac{d}{dt} N_2$. The latter quantity is referred to as the mutant's invasion fitness $f(S'_m, S_m)$ in the given resident population. Its derivative, $g(S_m) = \partial f(S'_m, S_m) / \partial S'_m|_{S'_m=S_m}$, known as the selection gradient determines the expected rate of evolutionary change according to the canonical equation of (scalar-valued) adaptive dynamics (Dieckmann & Law, 1996),

$$\frac{d}{dt} S_m = \frac{1}{2} \mu_{S_m} \sigma_{S_m}^2 \hat{N}_m g(S_m). \quad (8)$$

Here, \hat{N}_m denotes the equilibrium population size of the resident population, and μ_{S_m} is the fraction of mutations per birth. $\sigma_{S_m}^2$ is the variance of the mutation distribution M (Dieckmann & Law, 1996),

$$\sigma_m^2(S_m) = \int \Delta S_m^2 M(S_m, \Delta S_m) d\Delta S_m. \quad (9)$$

4. Monomorphic deterministic model with function-valued adaptive traits (MDM_f)

The main difference to the step above is the switch to function-valued adaptive traits. This approach enables individuals to adapt freely their dispersal kernel in response to a ecological environment.

Based on the prerequisites provided in the step above, we solve the canonical equation, governing the expected adaptive dynamics of function-valued traits (Dieckmann et al., 2005),

$$\frac{d}{dt} m(a) = \frac{1}{2} \mu_m \hat{N}_m \int \sigma_m^2(a', a) g_m(a') da'. \quad (10)$$

Here, σ_m^2 is the variance-covariance function of the mutation distribution M at trait value m ,

$$\sigma_m^2(a, a') = \int [m'(a') - m(a)][m'(a') - m(a)] M(m, m') dm', \quad (11)$$

where the integration extends over all feasible trait values m' . The function g is the selection gradient and is obtained as the functional derivative of the invasion fitness $f(m', m)$,

$$g_m(a) = \lim_{\varepsilon \rightarrow 0} [f(m + \varepsilon \delta_a, m) - f(m, m)] / \varepsilon = \left. \frac{\partial}{\partial \varepsilon} f(m + \varepsilon \delta_a, m) \right|_{\varepsilon=0}. \quad (12)$$

The MDM models will be used to identify evolutionary attractors in the shape of dispersal kernels and the corresponding PSM models will be used for comparison. Also, by comparing the results of the scalar-valued and function-valued models, it is possible to evaluate the capacity of the simpler scalar-valued adaptive traits to approximate the kernel shapes of the more complex function-valued adaptive traits.

Relevance and link to ADN's research plan

This project extends the work of the ADN Program on dispersal evolution, the spatial logistic equation, spatial invasion fitness, and the adaptive dynamics of function-valued traits.

Expected output and publications

This work is intended for publication as a co-authored research article and will also be included in my PhD thesis.

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Modeling the Evolution of Influenza in Human Population

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Goal

To devise models of evolutionary branching and extinction under frequency-dependent selection with no separation of ecological and evolutionary timescales and to develop a model of evolution of human influenza A virus as a typical instance of such systems.

Background and motivation

Influenza is a common respiratory disease caused by an RNA virus from the family Orthomyxoviridae. Despite decades of intensive research, influenza remains one of the most important causes of mortality and morbidity in humans (Earn et al., 2002). The influenza virus is classified into three subtypes, A, B and C, with subtype A being the most significant from the epidemiological and most interesting from the evolutionary and ecological points of view. One of the peculiarities of influenza A is the fact that ecological and evolutionary timescales are about the same. Complicated interactions between the rapidly changing virus and the human immune system result at the level of the whole population in a rather unusual phylogenetic pattern (Fitch et al., 1997; Grenfell et al., 2004): even though at each point in time the virus shows substantial diversity, only one of the variants survives in the long run resulting in a phylogenetic tree with a single trunk. It is still unclear what combination of factors underpins this kind of evolution.

Many attempts have been made to describe the persistence of the virus in the human population from the purely epidemiological point of view (see, for example, Lin et al., 2003; Andreasen et al., 1997), but only a few models incorporated the evolution of the virus (Ferguson et al., 2003). Although these evolutionary models yield qualitatively correct phylogenetic trees, they are complicated and based on assumptions of debatable validity. In order to understand the mechanisms underlying the remarkable persistence and peculiar single-trunk evolution of influenza, simpler models with fewer assumptions are necessary.

The recently developed adaptive dynamics theory is a good candidate framework for constructing such a model. However, so far adaptive dynamics models have considered mostly systems that allowed separation of ecological and evolutionary timescales (Dieckmann & Law, 1996), which is not the case in the human-influenza system and many other real ecological systems. It is a challenge to relax this assumption and to start investigating rapidly evolving systems such as those formed by complex interactions between pathogens and their hosts.

Research questions

This research aims at elucidating the conditions that are necessary and sufficient for a model to produce the single-trunk phylogenetic tree characteristic to influenza. In other words, what are the essential features of the real host-pathogen system that result in the kind of evolution we observe?

Methods and work plan

As mentioned above, the evolutionary dynamics of influenza is characterized by two key features: (a) relatively large diversity at each time point and (b) survival of only one variant in the long run. In other words, evolutionary branching happens constantly but almost all of the branches die out relatively quickly. Previous work suggests that under some conditions adaptive dynamics models can give rise to evolutionary branching (Geritz et al., 1997; Dieckmann & Doebeli, 1999) and evolutionary extinction (Ferrière, 2000). These conditions however are not satisfied in the human-influenza system. Therefore the research plan is the following.

I will try to devise a model of the human-influenza system in which competition between different virus variants for susceptible hosts plays an analogous role as the resource competition (Roughgarden, 1976) in the previous adaptive dynamics models (Dieckmann & Doebeli, 1999). As the first step in utilizing this analogy we need to find a suitable description of our system: we need to understand, on the one hand, what viral traits should be considered and, on the other hand, how to describe the host immune system. The later “remembers” all viral variants that the host individual has been infected with in the past and provides protection against them and closely related variants in the future, thus locally depleting the pool of susceptibles with respect to these variants.

Once a suitable description is found and the model is constructed it is necessary to assess its quality. One of the ways to do so is through the comparison of phylogenetic trees generated by the model with the real ones using appropriate metrics on the space of trees. If the phylogenetic patterns are similar it would be interesting to understand the mechanism underlying the single trunk phylogeny in the human-influenza system.

Relevance and link to ADN’s research plan

In this project we aim at extending the applicability of adaptive dynamics theory to rapidly evolving complex host-pathogen systems, of which influenza A is an example. Thus, this research falls naturally into ADN’s research focus on *Foundations of Adaptive Dynamics*, and is also linked to ADN’s research focus on *Virulence Management*.

Expected output and publications

This work is expected to be published in a jointly authored paper and will be included as a chapter in my PhD thesis.

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Genetic Footprints of Speciation

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Goal

To set up an individual-based model that allows for different speciation processes to be studied in one modeling environment, and to use this tool to study how patterns of variation in non-coding genetic sequences are influenced by different speciation processes.

Background and motivation

One of the main aims of evolutionary biology is to explain the species diversity that we see today and in the fossil record. Speciation apparently takes place often enough to give rise to a high species diversity, but not so often that we cannot distinguish species anymore. Understanding the process of speciation is therefore a central theme in evolutionary biology. The question of speciation can be split up into the following subquestions (where subquestion (4) can be seen as the main question): (1) Which processes can lead to speciation and what mechanisms drive these processes? (2) Under what circumstances do these mechanisms operate? (3) How will these mechanisms shape observable (genetic, ecological, geographic or other) patterns? (4) What mechanisms and processes have shaped and shape the species diversity that we observe today?

Concerning subquestion (1) Dieckmann et al (2004b) suggest a classification of speciation processes that uses three axes of differentiation: ecological, mating, and spatial differentiation, and also three mechanisms that can drive the differentiation: selection, drift, and external factors. A speciation process is then characterized by the route through the thus-defined three-dimensional space, and by the mechanisms that drive (different parts of) the route. The route to speciation in sexual organisms always starts at the origin of the three-dimensional space (no differentiation in any of the three directions) and ends with at least mating being differentiated. Also, after (successful) speciation, the two new species should be differentiated with respect to at least one of the other two axes, geography or ecology to allow their persistent coexistence. This follows directly from what is maybe the best established law of ecology, the law of competitive exclusion: two species can not coexist if they use exactly the same resources. The classical notions of allopatric and sympatric speciation can be incorporated into this classification. Allopatric speciation starts with an abrupt move on the spatial axis driven by an external factor. Sympatric speciation involves no move on the spatial axis at all. Adaptive speciation involves at least some movement that is driven by selection. It should be clear that, at least in theory, there are many routes to speciation.

The answers to subquestions (2) and (3) can both lead to an answer to (4). For example, if we knew exactly under what circumstances (in terms of parameter ranges) speciation by ecological differentiation occurs (i.e. the answer to subquestion (2)), we could estimate the relative importance of this process for speciation by determining the appropriate parameter values from data. In the same way, if we knew the resulting

genetic pattern of speciation by ecological differentiation (i.e. the answer to subquestion (3)), we could collect the right (genetic) data from pairs of sister species and determine how many of these pairs show the pattern caused by ecological differentiation and thereby estimate the relative importance of this process. Ideally, theoretical (and experimental) studies should provide answers to (2) and (3), and those answers should guide the direction of data collection.

Research on speciation is traditionally much more focused on subquestion (2) than on subquestion (3). Maybe this is simply because, until recently, no one could have imagined the amount of data that is available now or will be available in the near future. What are "observable patterns" has changed dramatically in recent years, especially regarding patterns of DNA sequence variation. Yet, this altered range of options has not yet been exploited for the study of speciation. This project is aimed at finding ways of using patterns of DNA sequence variation (hereafter called genetic patterns) to infer speciation processes. Apart from the availability of large amounts of data, there are two other reasons why now is a good time to look at these genetic footprints of modes of speciation. First, any work in this direction can build upon a framework for individual-based simulations that has been developed by Michael Doebeli, Ulf Dieckmann, and others in recent years. Second, with the power of today's computers, the complexity of the needed simulations should no longer constitute a problem.

Two lines of research are worth noting here. One is the work that was started by Barraclough and Vogler (2000) to try and exploit our knowledge of geographic patterns to infer the predominant speciation process in a clade. The other is the extensive work on how postzygotic isolation can evolve (see Orr (1995), Kondrashov et al. (2002), and Welch (2004)). These two lines of research do not overlap with what I suggest in this proposal but they are based on the same question: what can we learn about past speciation processes by looking at observable patterns today?

Research questions

In this project I aim to look at the effects of different speciation processes on genetic patterns. If I can identify these effects, this should allow me to infer (speciation) process from (genetic) pattern. The single main question is:

- How do different speciation processes shape genetic patterns?

With respect to the speciation processes, this main question can be refined:

- Which aspects of a speciation process have an effect on observable genetic patterns?

It could be, for example, that either the mechanism responsible for speciation or the geographic aspect of the speciation process is most important. Looking at it from the side of the genetic pattern, the main question can also be refined:

- Which aspects of DNA sequence variation are influenced by a speciation process?

This could for example be the number of segregating sites or the shape of the coalescent tree. And it could be necessary to look at many loci, or at rather specific loci. I will perform different neutrality tests (such as the HKA test (Hudson et al. 1987) and Tajima's D test (Tajima 1989)) on data harvested from the simulations to see if such standard tests may point to relevant differences.

Methods and work plan

Methods

The methods that I will use for this project are (i) individual-based simulations to generate data and (ii) standard population genetic methods to analyze them.

Steps

I will carry out the following steps. The first two steps will mainly consist of programming a model that incorporates all the necessary aspects.

1. Allow for different speciation processes to occur in one modeling environment so that direct comparisons between the different processes will be possible (see below for details of the models).
2. Add neutral loci, with appropriate recombination and mutation rates. This has already been modeled by Sergei Semovski and Yurji Bukin from Irkutsk; it should thus be easy to include these features in the model.
3. Run the simulations with different parameter values and harvest the information from the neutral loci. Some careful experimental design will be required in order to be able to explore different parts of the parameter space, while still generating enough data for each combination of parameter values.
4. Analyse the data from step 3 using tools from population genetics.

Model

Initially I will compare (a) strict sympatric speciation due to ecological differentiation in a non-spatial model (as described in Dieckmann and Doebeli 1999) with (b) strict allopatric speciation followed by reinforcement after secondary contact. The model should be built in such a way that scenario (a) and (b) can be run with only small changes to the parameter settings.

The model will describe a population of hermaphrodites that reproduce sexually. Character values will be determined by many additive diploid diallelic loci. There will be three important traits in the model: an ecological trait that determines resource use, a mating trait that determines assortativeness, and a marker trait that determines the preference in case of assortative mating. A fourth trait (a so-called internal trait) will be added later. There will be a resource that is distributed following a Gaussian distribution. In this model, the ecological trait value of the population will first evolve towards the optimum of the resource distribution, i.e., to where the carrying capacity has its maximum. At this optimum, the population may find itself at a fitness minimum if the curvature of the carrying capacity at its maximum is less than that of the competition function. Simply said, under these circumstances it pays off to be different from the rest because individuals gain more from avoiding competition than they lose because further away from the resource maximum there is (obviously) less resource.

As long as mating is random, recombination will always prevent evolutionary branching because it will create intermediate phenotypes. However, if there is linkage disequilibrium between the marker trait and the ecological trait, there is selection pressure for mating to become assortative. This is the case if, for example, birds that eat large seeds have slightly greener feathers and birds that eat smaller seeds have more yellow feathers (because of drift in a finite population). If a mutation now caused a bird to prefer to mate with a similar looking bird, this mutation would give the bird a selective advantage, and assortativeness could evolve, eventually leading to reproductive isolation.

In scenario (b) the population will be split in two by a virtual geographic barrier. Individuals will be assigned to one or the other subpopulation at random. To avoid evolutionary branching within the two identical subpopulations, the parameter determining the 'width' of the competition function will be enlarged. To allow for speciation to occur without ecological differentiation, a so-called internal trait has to be added to the model. This trait has no absolute fitness optimum, but offspring survival is highest when the distance between the trait values of the parents is zero. This trait will also be governed by several diallelic loci with additive effects. Since it would be more realistic if this trait would not be bound in its values, I could allow effect size of the different loci to evolve (in which case the loci do not remain diallelic, but infinitely many alleles could evolve). The mean value of this trait will change due to mutation and drift, not selection, while the variance of the trait value within a population is restricted by selection. If the original population is split in two and the two subpopulations evolve independently, the internal trait will (after some time) have different mean values in the two populations. The distance between those mean values will (in expectation) increase with time. If the two populations come into secondary contact, there will be selection for prezygotic isolation. And if the marker trait (that did not play a role until then) is in linkage disequilibrium with the internal trait, assortative mating can evolve (in the same way as in scenario (a)), so that the two populations will stay reproductively isolated. The internal trait has already been modeled by Géza Meszéna from Budapest, so it should be easily added to the model. In scenario (a) the addition of the internal trait after evolutionary branching will probably not change the process much, although it may slightly facilitate speciation.

Scenario (a) and (b) can both be seen as a two-patch system, in (b) there is no migration between the two patches, whereas in (a) the migration rate is so high that the two patches are effectively one. In reality the migration rate can also take intermediate values, and I will allow for this in my model.

In addition to scenario (a) and (b), it will be interesting to look at the effect of spatial resource heterogeneity (SRH), in times of the two patches differing in resource distribution. Day (2000) has analyzed the effect of SRH on evolutionary diversification in a two-patch system. He finds that, if migration rates are not too high, SRH facilitates evolutionary diversification. Spatial heterogeneity will constitute a third relevant axis of parameter space, the first two being migration and the ratio between the width of the resource distribution and the width of the competition curve.

Neutral loci will also be added to the model. These loci will be linked to the other loci, with a small probability of recombination in each generation. They will consist of a stretch of nucleotides that can mutate according to a simple mutation model (the one-parameter Jukes-Cantor model to start with, giving equal probabilities to every possible single nucleotide mutation). Simulations should run for a while to reach mutation-drift equilibrium, before evolutionary branching will be allowed in the first scenario, or before the population will be split in the second scenario. After the simulations have run, genetic sequences from all individuals will be collected and analyzed using standard population genetic methods.

Schedule

First month: The four steps described before should be carried out one after the other. It will be useful to do a round of these four steps relatively soon to get a first impression of how the model behaves and what needs to be changed.

Second month: With this knowledge I will make changes to the program (improving on steps 1 and 2) before starting a second round of simulations (step 3) and analyses (step 4).

Third month: I will use the last month of the summer program mainly to start writing down the results of this project.

Intermediate result: If the model with two scenarios (a and b) works (that is, step 1 of the work plan is carried out successfully;) I can obtain some intermediate results. Being able to simulate different modes of speciation in one model will allow me to directly compare the behavior of the two models. It would, for example, be interesting to determine whether the time needed for speciation is different in both models. These kinds of relatively simple comparisons have been complicated in the past by the fact that models for distinct modes of speciation differed too much in their set-up.

Relevance and link to ADN's research plan

In ADN, much previous research has been devoted to speciation. This project will build on and extend this work.

Expected output and publications

The study described here is expected to result in a co-authored paper in an international journal and will be integrated as a chapter in my PhD thesis.

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Effect of Habitat Selection Behaviour on Parapatric Speciation

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Goal

To investigate and compare the effect of fixed and evolving habitat selection behaviors on parapatric speciation on spatially structured resource landscapes.

Background and motivation

Bewildering diversity is a characteristic of life on all scales from populations to species to communities. Until recently, evolutionary theory postulated that each branching event has required geographic isolation of populations (Provine 2004), in disagreement with Darwin's (1859) view on speciation. But consider bugs and bats and birds. Insects comprise the vast majority of all known animal species, the ca. 1000 bat species comprise almost a fourth of all mammal species, and the ca. 10 000 bird species comprise well over a third of all terrestrial vertebrate species, spread over all habitable corners of the world with countless subspecies. These groups differ from most of their relatives in that they have wings, giving them superior ability to overcome geographical barriers. The notion of geographical isolation alone causing speciation appears to be at odds with the diversity of these highly mobile taxa.

Evolutionary ecologists studying speciation are starting to concentrate on mechanisms other than geography (Dieckmann et al. 2004, Rundle & Nosil 2005), suggesting that sympatric and parapatric speciation might be more prevalent than previously acknowledged (Via 2004). Also empirical evidence is mounting (e.g. Rolan-Alvarez et al. 2004, Cruz et al. 2004, Smith et al. 2005). Theoretically, speciation in panmictic, sympatric populations has been shown to occur due to frequency-dependent selection, if increased phenotypic difference from the phenotype maximizing carrying capacity reduces competition with the maximizing phenotype more than it reduces carrying capacity (Dieckmann & Doebeli 1999). When the model is extended to include space, this constraint is partially relaxed, so that if gene flow across a spatial resource gradient is restricted by limited movement of individuals and ecological interactions operate on sufficiently small distances, speciation occurs even if the effect of competition between different phenotypes exceeds the benefit gained from different resource use (Doebeli & Dieckmann 2003).

However, as noted above, many of the most species-rich taxa are exceptionally good dispersers. The conditions allowing parapatric speciation in the Doebeli & Dieckmann (2003) model become increasingly restricted with such high mobility. Evolution of habitat selection behaviors could be the key to resolving that contradiction.

If habitat selection behavior evolves, and mating requires sufficient proximity between individuals (which is obligatory for animals with internal fertilization and very likely for many other species), habitat selection leads to non-random mating (Rice 1987). Habitat selection can therefore potentially ease theoretical restrictions on non-allopatric speciation. In addition, because mobility combined with habitat selection would facilitate colonization of suitable unused habitat patches and enhance gene flow between similar but spatially separated patches, it could promote the survival and spreading of habitat-specific adaptations, facilitating evolution of pre-mating reproductive isolation via reinforcement. Mobility could thus become a factor facilitating evolutionary branching, instead of restricting it.

Research questions

The overarching question I will address is whether and how habitat selection behaviors affect conditions for parapatric speciation.

I will first concentrate on how the addition of a fixed habitat selection trait, determined by a single population-wide parameter, changes the conditions for parapatric speciation along a one-dimensional resource gradient as considered by Dieckmann & Doebeli (2003; see below). The most important questions here are whether habitat selection allows branching with larger and more frequent individual movements than the original model, and whether mobility with habitat selection could even facilitate branching.

This model will then be extended to allow evolutionary change in the one-dimensional habitat selection trait. Evolution of habitat preference and specialization were earlier argued to be mutually exclusive (de Meeûs et al. 1993), but this is not the case at least when habitat choice is determined pleiotropically by loci responsible for the specialization (Ravigné et al. 2004). The open question here, also raised by Ravigné et al. (2004), is whether the simultaneous evolution can occur if both traits can evolve simultaneously.

Finally, more complex forms of habitat selection behavior will be allowed to evolve in the multi-dimensional trait space provided by evolving neural nets (see below). These behaviors and their effects will be compared to the results from models with fixed and one-dimensional traits. Important questions are whether or not the evolving behaviors utilize the expanded trait space or resemble the simple one-dimensional traits, and whether they lead to different (e.g. faster) patterns of evolutionary branching.

Should these three sub-projects proceed faster than expected, the next goal could be to expand the approach from the effect of habitat selection on gradients to studying evolution in more complex spatially structured resource landscapes. I am already in the process of investigating habitat specialization and speciation in landscapes of two discrete habitat types, with a direct trade-off between utilization of the two resources. Therefore, investigating speciation with habitat selection in two-resource landscapes with different trade-off geometries (de Mazancourt & Dieckmann 2004) is another avenue of interesting research.

Methods and work plan

The work will build on a spatially explicit, individual-based simulation environment already developed, which describes an evolving population of diploid organisms, placed on spatially structured landscape, with the behavior of individuals controlled by neural nets.

The first task is to modify this simulation environment to reproduce the results of Dieckmann & Doebeli (2003) on speciation along linear environmental gradients without habitat selection behaviors. They model the evolution of ecological and mating preference traits – each defined by a number of diploid, diallelic, freely recombining, additive loci. Individuals reside on a two-dimensional landscape, where the ecological phenotype maximizing local carrying capacity changes with the environmental gradient in one direction, according to a steepness parameter. In other words, because of the environmental gradient, the intermediate phenotype is optimal in the middle of the landscape, and the two extreme phenotypes are optimal at the opposite edges of the landscape.

The effective population size experienced by an individual, divided by its phenotype-specific carrying capacity at its location, determines its death rate. This effective population size, in turn, depends on the number, distances, and phenotypes of surrounding individuals: the width of a spatial interaction kernel determines the competitive impact of individuals given distance away, and the width of a phenotypic interaction kernel determines how competition is reduced with increasing phenotypical difference. This leads to locally frequency-dependent selection: the death rate of an individual depends on the phenotypic composition in its neighborhood. In particular, individuals surrounded by others with differing phenotypes can experience a low death rate, even if the absolute number of neighbors is high.

Individuals give birth at a constant rate. Partners are chosen based on phenotypic difference and spatial distance, so that spatial proximity increases and phenotypic difference either decreases (assortative mating) or increases (disassortative mating) the probability of a partner being chosen, depending on the mating character of the choosing individual. Individuals move at a fixed rate, with movement distances drawn from a given distribution.

The outcome of simulations turns out to be affected by three parameters: the steepness of the environmental gradient, the width of the phenotypic interaction kernel, and the expected lifetime movement distance. Under certain parameter combinations the population branches into two species with different resource optima, inhabiting the adjacent halves of the environmental gradient. This branching requires that the environmental gradient is neither too steep nor too shallow, that competition sufficiently decreases with phenotypic difference, and that individual movement is small enough. Interestingly, if the lifetime movement distance is sufficiently small, branching occurs even when competition between different phenotypes is intense, for a wide range of gradient steepness.

In the first stage of the project, fixed habitat selection behaviors will be introduced in the simulation and results will be compared with the original. The fixed habitat selection behaviors planned to be investigated, in order of ascending complexity, are as follows:

1. Avoidance behavior – by altering the magnitude or the rate of movement, individuals exhibit more movement under poorer resource conditions than under good conditions.
2. Positive taxis – movement directions are biased in the direction of the gradient towards better resource conditions. The strength of this bias depends on the currently experienced resource conditions, so that individuals in poorest conditions exhibit the strongest bias.

3. Avoidance and taxis – a combination of the above, so that individuals at locations with poor resource conditions exhibit more movement *and* strongly bias its direction. By contrast, under good conditions individuals move less and more randomly. This combined behavior should lead to rapid convergence to high-fitness locations.

Furthermore, these behavior models can be extended by taking into account the intensity of competition an individual currently experiences, so that individuals tend to move away from intense competition. This would be predicted to facilitate dispersion but also counter frequency-dependent competition. Accordingly, strong competition-driven habitat selection could impede evolutionary branching.

In the next stage of the project, the avoidance strength and taxis accuracy will be allowed to evolve. Initially these behaviors will be set to describe random movement, and the simultaneous evolution of the three traits – specialization, assortativeness, and habitat selection – will be investigated.

Finally, the habitat selection behaviors will be allowed to evolve via the evolution of individual neural nets, while the other aspects of the model will be retained. These nets will receive information about an individual's surroundings, and will process that information into decisions affecting movement. The structure of the neural nets is controlled by an individual's diploid genome (there are two copies of each neuron). When an individual breeds, a haploid gamete is formed by recombination. Another gamete is drawn from the chosen partner individual, and these combine to form the neural net of a new offspring individual. Random mutations can occur with a given probability, providing new alleles to be shuffled by recombination.

Initially the neural nets cause random behavior, but over time selection leads to increasingly appropriate decisions. This setting will allow flexible, nonlinear, and potentially complex habitat selection behaviors to emerge, which would otherwise be difficult to model. More importantly, as the individuals are initially generalists without habitat selection abilities, the issue of simultaneous independent evolution of habitat specialization and preference (Ravigné et al. 2004) is explicitly investigated here in a multi-dimensional trait space.

Relevance and link to ADN's research plan

The effect of habitat selection on speciation should be profound, as it potentially creates genetic polymorphism, spatial segregation, and reduced gene flow within continuous populations (Ravigné et al. 2004). Thorough investigations into this are however only beginning. The planned work builds on research previously carried out by ADN (Dieckmann & Doebeli 2003). Investigating evolving habitat selection behaviors serves as a natural extension of this earlier research. The final part involving multi-dimensional trait spaces and neural net control is an extension into non-linear and flexible behaviors, and will also help to explore evolution on increasingly complex spatial landscape, a line of research that is already in progress in ADN.

Expected output and publications

I anticipate writing a manuscript on the effects of simple habitat selection behaviors during the Summer Program.

The work involving multi-dimensional trait spaces and neural net control, should it proceed smoothly, is also likely to yield a manuscript during or soon after the Program.

After the Program, should we find such collaboration feasible, I would continue working in collaboration with ADN on exploring evolution in more complex landscapes with spatial and temporal resource dynamics.

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The Influence of Harvesting Pressure on Evolving Food Webs

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Goal

To examine how harvesting pressure influences the evolution of food webs structured by body size.

Background and motivation

Harvesting pressure in fisheries causes drastic ecosystem changes, such as the simplification of trophic structure in food webs (Pauly et al., 1998). While harvesting clearly has ecosystem effects, most fisheries models tend to be single-species models which ignore food web interactions such as predation, competition or indirect effects (Clark, 1990; Hilborn and Walters, 1992). Recently, the importance of understanding harvesting within the context of food web interactions is being recognized (Yodzis, 2001) and incorporated into management models (e.g. EcoSim; Walters et al., 2000). However, the implications of harvesting are not limited to trophic effects, but, when considered over time, can also cause population traits to change and evolve adaptively. Studies have found that harvesting can cause adaptive changes on both short and long time scales. On shorter time scales, changes can occur in species behavior, such as prey preference, foraging time and anti-predator behaviors (e.g. Abrams and Vos, 2003; Matsuda and Abrams, 2004). On longer time scales, changes can occur in life-history traits, such as earlier maturation ages and smaller adult body sizes (Heino, 1998; Heino and Godø, 2002; Gårdmark et al., 2003; Ernande et al., 2004). Yet, while it is known that food web interactions and adaptive responses must be taken into account when considering harvesting pressure (Clark, 1990; Hilborn and Walters, 1992), there have been few studies which take a comprehensive approach incorporating the effects of all three (but see Ernande et al., 2004; Matsuda and Abrams, 2004). Determining how harvesting pressure, multi-trophic interactions and adaptive dynamics interact will thus help in understanding the effects of harvesting pressure on food webs.

For instance, if only food web interactions are considered, increasing harvesting may cause the target species to go extinct (Yodzis, 2001; Matsuda and Abrams, 2005). By contrast, with the inclusion of adaptive dynamics, the target species may not necessarily go extinct; instead, its population traits may change and evolve in response to the fishing pressure. With adaptive dynamics on short time scales, increased harvesting can cause the target species to change its behavior to avoid harvesting or even cause the target species' predator to switch diet (Matsuda and Abrams, 2004). With adaptive dynamics on long time scales, increased harvesting can cause changes in the target species' reaction norms or cause the target species to mature earlier and, consequently, to have smaller adult body sizes (Heino, 1998; Ernande et al., 2004). Hence, the inclusion of adaptive dynamics in food web interactions is critical to understanding the effects of harvesting pressure.

For my proposed project, I intend to study how food webs with adaptive dynamics respond to harvesting pressure. However, as mentioned, adaptive dynamics can occur on short and long time scales. While short and long time scales are equally important, I will focus, for the sake of tractability, only on the inclusion of adaptive dynamics in

the context of long-term evolutionary changes.

Research questions

The main goal of this project is to examine how harvesting pressure influences the evolution of food webs structured by body size. Empirical studies have shown that body size plays an important role in the dynamics and structuring of food webs (e.g. Cohen et al., 1993). Indeed, body size will determine the strength of species interactions through the ratio of predator-prey body sizes or simply through the energetic transfers within the food web. Hence, studying the evolution of body size will help explain how fundamental food web properties can change and respond to disturbances such as harvesting pressure.

In addition, I will investigate how different types of harvesting strategies influence the evolution of food webs. Harvesting typically does not occur at a constant rate, but rather will include some degree of feedback control (Hilborn and Walters, 1992). This can be represented as fixed-quota or fixed-stock strategies. Also, harvesting is not always restricted to a single target species, but can include multiple species at the same or differing trophic levels (Matsuda and Abrams, 2005). Thus, I will study the influence of varying harvesting strategies and of varying the number and type of target species.

Methods and work plan

I intend to study the influence of harvesting pressure on food webs that are assembled based on simple evolutionary and ecological rules, such as foraging optimization (Kondoh, 2003) or trait similarity (Quince et al., 2005). In particular, the food web models I plan on examining use rules based on body size (e.g. Fukami, 2004), adapted from Loeuille and Loreau (2005), with the addition of adaptive dynamics (e.g. Abrams et al., 1993; Dieckmann and Law, 1996).

The basic methodology is to first create food webs that satisfy the following three stability conditions:

1. Demographically stable: Population densities of the species have reached equilibrium.
2. Convergence stable: Absence of directional selection.
3. Locally evolutionary stable: Absence of disruptive selection.

Food webs are evolved from an initial set of “founding” species which are distributed in trait space, in this case body size. The initial species are then allowed to interact according to their population and adaptive dynamics, from which a community that is demographically, convergence and evolutionarily stable should emerge. On this basis, I will examine the influence of harvesting pressure on the resulting food webs. In the first stage, I will examine harvesting pressure on food webs without adaptive responses to harvesting, while in the second stage I will examine webs with adaptive responses to harvesting.

a. Demographic dynamics

The basic population dynamics can be described using a simple equation with linear functional responses,

$$\frac{dN_i}{dt} = N_i \left(\sum_{j=1}^n e_{ij} \gamma_{ij} N_j - m_i - \sum_{j=1}^n \alpha_{ij} N_j - \sum_{j=1}^n \gamma_{ji} N_j \right), \quad (1)$$

where e_{ij} is the conversion efficiency of N_i on N_j ; m_i is the intrinsic mortality rate of N_i ; γ_{ij} and α_{ij} are functions which, respectively, represent the consumption rate and interference competition of species N_i on N_j .

The function describing the consumption rate is based on empirical studies that observed that consumers have the highest consumption rates on prey that satisfy an ideal predator-prey body size ratio (r_o), while deviations away from it have lower consumption rates. A Gaussian function with standard deviation δ_c can describe the consumption rate of a predator with body size B_i on prey with body size B_j ,

$$\gamma_{ij} = \frac{\gamma_o}{\delta_c \sqrt{2\pi}} \exp\left(-\frac{1}{2\delta_c^2} \left(\log_{10}(B_i/B_j) - \log_{10}(r_o)\right)^2\right), \quad (2)$$

where γ_o is a scalar scaling the maximum consumption rate.

Similarly, the function describing the amount of interference competition is based on empirical observations that species with similar body sizes tend to have more interference competition. Again, a Gaussian function with standard deviation δ_I can describe the amount of interference competition from species N_j on species N_i ,

$$\alpha_{ij} = \frac{\alpha_o}{\delta_I \sqrt{2\pi}} \exp\left(-\frac{1}{2\delta_I^2} \left(\log_{10}(B_i/B_j)\right)^2\right), \quad (3)$$

where α_o is a scalar scaling the maximum amount of interference competition.

b. Adaptive dynamics

Adaptive dynamics are incorporated into the food web model using the canonical equation as described by Dieckmann and Law (1996). In this case, the trait which is allowed to evolve is body size,

$$\frac{dB_i}{dt} = k(B_i) \left. \frac{\partial W_i(B'_i, B_i)}{\partial B'_i} \right|_{B'_i=B_i}, \quad (4)$$

where $W_i(B'_i, B)$ is the invasion fitness of the individuals with body size B_i in the environment determined by the resident trait values B , and $k(B_i)$ scales the rate of change of the trait B_i . I will use two alternative forms for $k(B_i)$ to compare the assumptions of adaptive dynamics (AD) and the assumptions of quantitative genetics (QG). To examine the assumptions of AD, I will use a form for $k(B_i)$ that is proportional to the population densities N_i . To examine the assumptions of QG, I will use a form for $k(B_i)$ that is independent of population densities.

c. Harvesting

Following the creation of stable food webs, I intend to study the effects of harvesting pressure. I will examine three types of harvesting strategies (Hilborn and Walters, 1992): constant rate, fixed-stock, and fixed-quota strategies. For a constant rate strategy, the harvested population of species N_i can be expressed as

$$H_i = \mu_c N_i, \quad (5)$$

where H_i is the amount harvested from population N_i and μ_c is a scalar scaling the harvesting rate. For a fixed-stock strategy, the harvested population of species N_i can be expressed as

$$H_i = N_i - \mu_s, \quad (6)$$

where μ_s represents the minimum population density to be maintained. Finally, for a fixed-quota strategy, the harvested population of species N_i can be expressed simply as

$$H_i = \mu_q, \quad (7)$$

where μ_q represents the maximum quota that can be harvested in a population.

d. Measuring the effects of harvesting

To measure the effects of harvesting, I will compare the changes in population density in all species, keeping track especially of instances when species go extinct. Taking the perspective of fisheries, I will also study the changes in the average yield from the harvested species (Y_i), which can be calculated as

$$Y_i = \frac{1}{T} \int_0^T H_i dt, \quad (8)$$

where T is the time interval for which the average yield is calculated.

In order to gain some understanding of the influence of harvesting on population dynamics, I will examine changes in stability in terms of persistence (minimum persistence of the species) and variability (global stability as characterized by the dominant Lyapunov exponent).

Relevance and link to ADN's research plan

This project fits into to ADN's research focus on Fisheries-Induced Adaptive Change. It will also contribute to ADN's new theme on Evolving Biodiversity.

Expected output and publications

This work will be included as a chapter in my PhD thesis. I also intend to publish this work as a co-authored research article.

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