Modelling the struggle for existence in structured populations

Vol. 2: Annexes

Mémoire pour l’obtention de l’Habilitation à diriger des recherches
Soutenance : 10 janvier 2012 à Paris

David Claessen

Laboratoire “Ecologie & Evolution”,
UMR 7625 CNRS-UPMC-ENS
and
Centre d’Enseignement et des Recherches sur l’Environnement et la Société - Environmental Research and Teaching Institute (CERES-ERTI),
Ecole Normale Supérieure,
24 rue Lhomond,
75005 Paris
This document contains reprints of articles discussed in “Vol. 1: Synthesis”, organised by section. Section numbers refer to the sections in Vol. 1.

**Contents**

A Annexes

A.1 Size structure (section 2) ..................................................... 3
A.2 Adaptive dynamics (section 3) .............................................. 65
A.3 Current and future research (section 4) .............................. 203
A.4 Other work (section 5) ......................................................... 221
A Annexes

A.1 Size structure (section 2)


Bistability in a size-structured population model of cannibalistic fish—a continuation study

David Claessen and André M. de Roos

Institute for Biodiversity and Ecosystem Dynamics, University of Amsterdam, P.O. Box 94084, 1090 GB Amsterdam, The Netherlands

Received 21 February 2002

Abstract

By numerical continuation of equilibria, we study a size-structured model for the dynamics of a cannibalistic fish population and its alternative resource. Because we model the cannibalistic interaction as dependent on the ratio of cannibal length and victim length, a cannibal experiences a size distribution of potential victims which depends on its own body size. We show how equilibria of the resulting infinite-dimensional dynamical system can be traced with an existing method for numerical continuation for physiologically structured population models. With this approach we found that cannibalism can induce bistability associated with a fold (or, saddle-node) bifurcation. The two stable states can be qualified as 'stunted' and 'piscivorous', respectively. We identify a new ecological mechanism for bistability, in which the energy gain from cannibalism plays a crucial role: Whereas in the stunted population state cannibals consume their victims, on average, while they are very small and yield little energy, in the piscivorous state cannibals consume their victims not before they have become much bigger, which results in a much higher mean yield of cannibalism. We refer to this mechanism as the 'Hansel and Gretel' effect. It is not related to any individual 'choice' or 'strategy', but depends purely on a difference in population size distribution. We argue that studying dynamics of size-structured population models with this new approach of equilibrium continuation extends the insight that can be gleaned from numerical simulations of the model dynamics.

© 2003 Elsevier Science (USA). All rights reserved.

Keywords: Size-dependent cannibalism; Physiologically structured population model; Fold bifurcation; Continuation; Mean yield; Eurasian perch; Infinite-dimensional dynamical system; Infanticide

1. Introduction

Theoretical studies have shown that the population dynamic consequences of cannibalism may be manifold. Gurtin and Levine (1982) showed that cannibalism can regulate a population that would otherwise grow exponentially. Cannibalism may have various effects on the stability of populations; on the one hand it may induce population cycles (Diekmann et al., 1986; Hastings, 1987; Magnússon, 1999) or chaos (Costantino et al., 1997), on the other hand it may dampen cycles that are caused by other interactions (van den Bosch and Gabriel, 1997; Claessen et al., 2000). Cannibalism is also known to induce multiple stable states (Botsford, 1981; Fisher, 1987; Cushing, 1991, 1992). A striking example of the latter is the so-called life boat mechanism, which enables a cannibalistic population to persist under conditions where a non-cannibalistic, but otherwise identical population would go extinct (van den Bosch et al., 1988; Henson, 1997). Finally, cannibalism can have major impacts on population size distribution and individual life history (Fisher, 1987; Claessen et al., 2000, 2002, and below).

Although there are exceptions, cannibals are generally larger than their victims (Polis, 1981). As a consequence, many theoretical studies of cannibalism use physiologically structured population models (e.g., Diekmann et al., 1986; van den Bosch et al., 1988; Cushing, 1992; Claessen et al., 2000), in which the roles of cannibals and victims are determined on the basis of stage, age or size...
Our aim here is to complement our previous results with an analysis of equilibrium curves and thus to provide more insight into the population dynamics observed in simulations. Since we cannot explicitly express the equilibrium of our model in terms of its parameters, we study equilibrium curves by numerical continuation (i.e., by tracing an equilibrium while varying one or more parameters). For models of ODEs or discrete maps numerical methods for continuation of equilibria and stability analysis are readily available (e.g., Kuznetsov, 1995), but this is not the case for physiologically structured population models (PSPMs' hereafter, Metz and Diekmann, 1986). One difficulty with PSPMs is that they are in principle infinite dimensional, as functions of individual state (e.g., size) enter the definition of the population state. Our continuation approach is based on a general method for numerical equilibrium continuation of PSPMs, recently introduced by Kirkilionis et al. (2001). Although the method is developed for finite-dimensional environments (e.g., the abundances of \( k \) food sources), we show how it can be used to approximate an infinite dimensional environment (e.g., the continuous size distribution of prey) that is needed to describe size-dependent cannibalism.

This article covers both new ecological results and a new (numerical) method of analysis and therefore it is structured as follows. In Section 2, we define our model of size-dependent cannibalism. In Section 3, we first give a non-technical outline of the continuation approach. The rest of that section (starting with Section 3.2) is more technical and gives a detailed description of the continuation approach. Section 4 describes the results of the model analysis. The discussion (Section 5) treats the ecological results as well as the merits of the continuation method.

2. The model

Our model describes the dynamics of a size-structured, cannibalistic population and its unstructured, alternative resource population. The parameter values we use (Table 1) are based on piscivorous fish, in particular Eurasian perch (\( P. f. \)), and zooplankton (\( D. \) spp.) (Claessen et al., 2000). In our model, we assume that the physiological state of an individual is completely determined by its body length \( x \). Vital rates such as food ingestion, metabolism, reproduction and mortality are assumed to depend entirely on body length and the condition of the environment. The population size distribution is denoted by \( n(x) \) and the density of the alternative resource by \( R \). All individuals are born with the same length \( x_b \), and are assumed to mature upon reaching the size \( x_f \). Reproduction is assumed to be continuous (in time) which implies that...
the size distribution \(n(x)\) is continuous. Note that in our notation we ignore all time dependencies (e.g., \(n(x)\) rather than \(n(t, x)\)) because we consider equilibria only.

The assimilation rate is assumed to follow a size-dependent, type II functional response

\[
F(x) = c_a \frac{\gamma(x)}{1 + H(x)\gamma(x)},
\]

where \(c_a\) is the assimilation efficiency, \(\gamma(x)\) is the sum of the encounter rates with conspecific and alternative prey mass, and \(H(x)\) is the size-dependent digestion time per gram of prey mass (Table 2). The encounter rate with alternative prey mass is assumed to be  

\[
\gamma_a(x) = A(x)R.
\]

We assume that the attack rate on the alternative resource \(A(x)\) is dome shaped, positive for the length interval \((0, x_p)\) and reaches a maximum at length \(x_p/2\). At length \(x_p\) the function \(A(x)\) and its slope become zero (Table 2).

The encounter rate with conspecific prey mass is obtained by integration over the size distribution of potential victims. We assume that a cannibal of length \(x\) can capture victims with lengths \(y\) in the range \(\delta x < y < \varepsilon x\), and that the encounter rate with conspecific prey mass hence is

\[
\gamma_c(x) = \int_{\delta x}^{\varepsilon x} \beta x^2 T(x, y)w(y)n(y)\,dy.
\]
The term $\beta x^2$ is the maximum cannibalistic attack rate of a cannibal of length $x$, reached only for the optimal victim size $y = \phi x$ (with $\delta < \phi < a$). The parameter $\beta$ hence reflects the species-specific tendency to cannibalize, which we refer to as the cannibalistic voracity. The tent-shaped function $T(x, y)$ accounts for the effect of suboptimal victim sizes $y \neq \phi x$. It takes values between 0 and 1 if $\delta x < y < a x$, and is zero outside this range (Table 2). We refer to the parameters $\delta$ and $a$ as the lower and upper limits of the cannibalism window, respectively (Claessen et al., 2000). Thus, the product $\beta x^2 T(x, y)$ equals the attack rate of a cannibal of length $x$ on a victim of length $y$. The mass of an individual of length $x$ is assumed to scale with body volume, $w(x) = \dot{x} x^3$.

We use a quadratic relation for the maximum cannibalistic attack rate, even though a sigmoidal function would describe the observed maximum cannibalistic attack rate better (L. Persson, pers. com.). Such a sigmoidal function would, however, be computationally more costly. Data show that small individuals (age 0–1 years, length 7–50 mm) cannibalize little while the maximum attack rate levels off at very large sizes (>250 mm). In Claessen et al. (2000) we used a function that describes the leveling-off well ($x^{0.6}$), but overestimates the cannibalistic potential of small individuals. This had no important consequences since that model assumed pulsed reproduction, such that cannibalism was practically impossible up to the age of 1 year. In the present context with continuous reproduction, the fact that the function $x^{0.6}$ overestimates cannibalism by very small individuals becomes problematic. Hence, we chose to use a quadratic relation, although it overestimates cannibalism by very large individuals. With a more realistic sigmoidal function, similar results would be obtained as long as the accelerating part of the function resembles the quadratic relation; but the maximum size in the population would be smaller.

We assume that a fraction $\kappa$ of assimilated energy is allocated to growth and maintenance (Kooijman and Metz, 1984), and the remainder to reproduction. The growth rate in mass is obtained by subtracting the metabolic rate from the energy intake rate. Assuming that the metabolic rate scales with body volume like $\rho x^3$, the growth rate in length becomes

$$\dot{g}(x) = \frac{1}{3\lambda x^2}(\kappa F(x) - \rho x^3). \quad (3)$$

The length for which the metabolic rate equals the intake rate allocated to growth ($\kappa F(x) = \rho x^3$) is referred to as the maximum length, denoted by $X_{\text{max}}$.

We assume that all individuals allocate the fraction $1 - \kappa$ of the assimilation rate to reproduction. For juveniles ($x < x_J$), this energy is assumed to be used for development of reproductive organs (Kooijman, 1993). For adults, the per capita birth rate is calculated by dividing the investment in reproduction by the energy cost of producing a single newborn:

$$b(x) = \begin{cases} c_r (1 - \kappa) F(x) w(x) & \text{if } x \geq x_f, \\ 0 & \text{otherwise,} \end{cases} \quad (4)$$

where the conversion efficiency $c_r$ takes into account losses due to egg respiration.

The mortality rate is assumed to be the sum of a constant background mortality rate $\mu_0$, a size-dependent mortality rate caused by cannibalism, and a starvation mortality:

$$\mu(x) = \mu_0 + \mu_c(x) + \mu_s(x). \quad (5)$$

In accordance with (2), the cannibalistic mortality rate is defined as

$$\mu_c(x) = \int_{x/e}^{x/\delta} \frac{\beta x^2 T(y, x)}{1 + H(y) \gamma(y)} n(y) dy. \quad (6)$$

In equilibrium individuals cannot grow beyond the maximum sustainable size, so for an equilibrium analysis we do not have to consider starvation mortality. However, in population cycles individuals may go through periods of food shortage and starvation. For such cases we assume that starvation mortality rate increases linearly with the difference between the metabolic rate and the food assimilation rate

$$\mu_s(x) = \begin{cases} s [\rho x^3 - \kappa F(x)] & \text{if } \kappa F(x) < \rho x^3, \\ 0 & \text{otherwise,} \end{cases}$$

where $s$ is a proportionality constant. The individual-level model is summarized in Table 2 and the PDE formulation for the population-level model is presented in Table 3.

We assume that the alternative resource population is unstructured. In our model it follows semi-chemostat dynamics extended with a term to account for the effect of consumption by the structured population,

$$\frac{dR}{dt} = r(K - R) - \int_{x_0}^{x_{\text{max}}} \frac{A(x) R}{1 + H(x) \gamma(x)} n(x) dx$$

with $A(x)$, $H(x)$ and $\gamma(x)$ as defined in Table 2.

| PDE | $\frac{\partial n}{\partial t} = \frac{\partial m}{\partial x} = -\mu(x)n(x)$ |
| Boundary condition | $g(x_0) n(x_0) = \int_{x_0}^{x_{\text{max}}} b(x) n(x) dx$ |
| Resource dynamics | $\frac{dR}{dt} = r(K - R) - \int_{x_0}^{x_{\text{max}}} \frac{A(x) R}{1 + H(x) \gamma(x)} n(x) dx$ |

The individual-level functions are listed in Table 2. Parameters are listed in Table 1.

*aNote that the time argument has been left out from variables and functions.*
3. The continuation approach

3.1. Non-technical outline

As mentioned in the Introduction, in this article we study our model (Table 3) by numerical continuation of equilibrium curves. Continuation methods for ODEs are well established but for PSPMs they are still a very recent development. We use the continuation method of Kirkilionis et al. (2001) which is based on the fundamental distinction in PSPMs between the state of an individual and the condition of its environment. The idea is that if the environment is prescribed properly, the life history of an individual (in terms of the development of its body size, survival, energy reserves, etc.) can be calculated without direct knowledge about other individuals. In this view, ‘environment’ mediates all interactions that affect the fate of an individual. The set of rules which dictates how the state of an individual changes in response to the environment is referred to as the individual level or i-level model (Metz and Diekmann, 1986). With such a distinction between environment and individual state, an equilibrium of the PSPM requires an environmental condition which gives rise to a life history consistent with this environmental condition, in the sense that the population growth rate equals zero and, moreover, the extant population generates the environmental condition by feedback.

We illustrate this with an example. Consider a size-structured population in which individuals interact with each other only through exploitative competition, i.e., through their impact on food density (e.g., de Roos et al., 1992; de Roos, 1997, or our model with $\beta = 0$). The environment of an individual is then fully characterized by the food density. Once a constant food density is given, the life history of an individual is determined as well, because it depends on the food density only. This shows that if the environment is known, individuals can be considered to be independent. Once the life history is known, we can formulate the necessary conditions for equilibrium. In this example, they are the requirements (i) that on average an individual exactly replaces itself and (ii) that the total population consumption rate equals renewal rate of the food resource. Denoting the per capita lifetime reproduction by $R_0$, the total consumption rate by $X$ and the renewal rate by $Y$, these conditions can be specified as $R_0 - 1 = 0$ and $X - Y = 0$, respectively. These are two equations in the two unknowns $P$, the population birth rate and $R$, the resource density.

We refer to the variables $P$ and $R$ as input ($I$) variables, and to $R_0 - 1$ and $X - Y$ as output ($O$) variables (Diekmann et al., 1998, note that our definition of input and output is different). Quite generally the life history and the feedback yield a map $f : \mathbb{R}^k \to \mathbb{R}^k$, $I \mapsto O$, (7) which we call the ‘input–output map’. An equilibrium can be found via an input $I^* \in \mathbb{R}^k$ for which the equilibrium and feedback conditions $f(I^*) = 0$

hold. In our example this is a two-dimensional map, with the food density $R$ and the population birth rate $P$ as input variables and $R_0 - 1$ and $X - Y$ as output variables. The population birth rate is an input variable because it determines the total population size and hence the total population consumption rate.

The condition $f(I^*) = 0$ can now be used in numerical continuation. Applying the method of Kirkilionis et al. (2001) we would trace the equilibrium food density and the total population birth rate while varying one free parameter. In this case the continuation problem is hence only two dimensional, although the population size distribution is still an infinite-dimensional object.

As pointed out above, in our model of size-dependent cannibalism the interactions depend directly on body size. This means that the definition of environment should include functions of body size, such as the size-dependent mortality rate due to cannibalism, denoted $\mu_\alpha(x)$ where $x$ is body size. The input variable for our model of cannibalism is therefore infinite dimensional. Kirkilionis et al. (2001) developed their continuation method for cases in which the environment is finite dimensional. In Sections 3.5 and 3.6 we show in detail how our model with an infinite-dimensional interaction environment can be studied with the method of Kirkilionis et al. (2001).

We are not able to evaluate the stability of equilibria during the continuation. For information on stability we therefore rely on EBT simulations of the model as summarized in Table 3. In a biologically meaningful way this method discretizes the continuous population distribution by subdividing the population into cohorts of individuals of similar age. The method is described in detail in de Roos et al. (1992) and de Roos (1997).

Sections 3.2–3.6 treat the new continuation methodology in detail. The biologically interested readers may skip these sections and continue with Section 4.

3.2. Life history as an input–output map

In this section we show how elements from the i-level model outlined in Section 2 can be used to construct a life history if the appropriate input is given. We subdivide the life history into three aspects: survival, growth and reproduction. The probability to survive to age $a$ is denoted $S(a)$ and is the solution of the ODE

$$\frac{dS}{da} = -\mu(x(a))S(a), \quad S(0) = 1,$$  

(8)
where the function $\mu(x)$ is the size-dependent mortality rate. The growth trajectory, denoted $x(a)$, is the solution of
\[
dx{a} = g(x(a)), \quad x(0) = x_0, \tag{9}
\]
with $g(x)$ the growth rate in length. The expected, cumulative reproduction up to age $a$, denoted $B(a)$, is the solution of
\[
\frac{dB}{da} = b(x(a))S(a), \quad B(0) = 0, \tag{10}
\]
in which $b(x)$ is the size-dependent, per capita birth rate. The expected, lifetime reproductive output, denoted $R_0$, is then given by
\[
R_0 = B(\infty). \tag{11}
\]
Due to the occurrence of $x(a)$ in (8) and (10), (9) has to be solved first, then (8), and finally (10). Alternatively, the ODEs (8)–(10) can be solved simultaneously.

Together, $S(a)$, $x(a)$ and $R_0$ define a life history. It should be noted that the rates $\mu(x)$, $g(x)$ and $b(x)$ may depend on the environment. If the latter is known the life history is set. Below we make the dependence on the environment explicit.

### 3.3. In the absence of cannibalism

In equilibrium and in the absence of cannibalism (i.e., $\beta = 0$) the mortality rate (5) reduces to the constant background mortality rate, $\mu(x) = \mu_0$ (due to the assumption of equilibrium there is no starvation). The growth rate $g(x)$ and the fecundity $b(x)$ depend on the alternative resource density only ((3) and (4)). This implies that the resource density $R$ alone is required as input to integrate (8)–(10).

In equilibrium each individual must exactly replace itself which translates into the condition $R_0 = 1$. Additionally, the total consumption rate of the size-structured population must equal the renewal rate of the alternative resource which constitutes the second equilibrium condition. To obtain the total consumption rate from the life history, we use the fact that in equilibrium the consumption rate of the entire population equals the total (lifetime) consumption of a single individual multiplied with the population birth rate. This follows from the fact that in equilibrium we can write the population age distribution as $m(a) = S(a)P$, where $P$ is the population birth rate. We can use this to calculate integrals over the size axis in terms of integrals over the age axis,
\[
\int_{x_0}^{x_{\text{max}}} z(x)n(x) \, dx = P \int_0^\infty z(x(a))S(a) \, da, \tag{12}
\]
where $z(x)$ is some weighing function, e.g., the per capita consumption rate of alternative resource.

We denote the expected, cumulative consumption up to age $a$ with $\theta(a, R)$. It can be calculated in parallel with (8) and (9) by integrating
\[
\frac{d\theta}{da} = \frac{A(x(a))R}{1 + \gamma(x(a))H(x(a))}S(a), \quad \theta(0, R) = 0 \tag{13}
\]
(cf.(1)). The total population consumption rate of alternative resource is then the product of $P$ and $\theta(\infty, R)$. Note that $P$ is required as an input variable, since it cannot be derived from the life history.

Now we can formulate the input ($I$) and output variables ($O$) used for the input–output map (7). As input we need the resource density $R$ and the population birth rate $P$,
\[
I_1 = R, \tag{14}
I_2 = P. \tag{15}
\]
The output variables are the equilibrium conditions,
\[
O_1 = R_0(I) - 1, \tag{16}
O_2 = r(K - I_1) - I_2(\infty, I_1). \tag{17}
\]
Then, for the non-cannibalistic case we have a two-dimensional map, $f : \mathbb{R}^2 \rightarrow \mathbb{R}^2$ like (7). The equilibrium condition $f(I^*) = 0$ can be used to continue an equilibrium $I^*$ with one free parameter by applying the method of Kirkilionis et al. (2001).

### 3.4. In the presence of cannibalism

With cannibalism ($\beta > 0$) the rates $\mu(x)$, $g(x)$ and $b(x)$ in (8)–(10) all depend on the environment in a size-dependent manner. This dependence results from the occurrence of the cannibalistic mortality rate $\mu_c(x)$ in (5) and of the encounter rate with conspecific prey $\gamma_c(x)$ in (3) and (4). It implies that, in addition to $I_1$ and $I_2$ (14) and (15), these two functions are required as input for the calculation of a life history,
\[
I_\mu(x) = \mu_c(x), \tag{18}
I_\gamma(x) = \gamma_c(x). \tag{19}
\]
On the basis of this extended input $I$ we can again calculate the life history with (8)–(10). From the obtained survival function $S(a)$, the growth trajectory $x(a)$ and the population birth rate $P$ it is possible to construct the population size distribution $n(x)$, using (12). This enables us to use (2) and (6) to compute the functions $\mu_c(x)$ and $\gamma_c(x)$ that correspond to this extended input. If we denote the result of these calculations by $I_\mu(x, I)$ and $I_\gamma(x, I)$, emphasizing their dependence on the input, we obtain two more output relations:
\[
O_\mu(x) = I_\mu(x) - \mu_c(x, I'), \tag{20}
O_\gamma(x) = I_\gamma(x) - \gamma_c(x, I'). \tag{21}
\]
With these input and output variables we can now define an input–output map like (7), such that \( f(I/C) = 0 \) defines an equilibrium point. However, because \( I \) and \( O \) contain functions of body size, the map is infinite dimensional.

### 3.5. Continuation

For continuation purposes the infinite-dimensional input–output map has to be approximated by a finite-dimensional one. In order to obtain a finite-dimensional map, we represent the functions \( \mu_c(x) \) and \( \gamma_c(x) \) by their values at the fixed sizes \( x_1, x_2, \ldots, x_k \). The values \( \mu_c(x) \) are used as input, \( I_3 = \mu_c(x_1), I_4 = \mu_c(x_2), \ldots, I_{k+2} = \mu_c(x_k). \) \( \gamma_c(x) \) is approximated by \( \gamma_c(x) \) at \( x \) equal to \( x_1, x_2, \ldots, x_k \).


equation here

3.5. Continuation

We choose \( x_k \) such that \( x_{\text{max}} < x_k \) to account for the entire range of body sizes. In the calculation of the life history (8)–(10), the value of \( \mu_c(x) \) is approximated by linear interpolation between the values at the two nearest \( x_i \) and \( x_{i+1} \). Because we assume that cannibals are always larger than their victims, the function \( \gamma_c(x) \) is not actually needed as input; when during the integration of (8)–(10) the growth rate of a cannibal must be calculated, the size distribution of its potential victims can be reconstructed from results obtained previously in the integration (Section 3.6).

The output variables are then defined by (16) and (17) together with

\[
\begin{align*}
O_3 &= I_3 - \mu_c(x_1, I), \\
O_4 &= I_4 - \mu_c(x_2, I), \\
&\quad \vdots \\
O_{k+2} &= I_{k+2} - \mu_c(x_k, I).
\end{align*}
\]

In Section 3.6 we give more details of the finite-dimensional approximation and show how to calculate the new estimates \( \mu_c(x_i, I) \). The input–output map (7), the input and output variables ((14)–(17) and (22)–(23)), and the life history as specified with (8)–(11) and (13) were used for numerical continuation to obtain the results in Section 4. We can apply numerical continuation techniques (Kuznetsov, 1995), for example to continue the equilibrium with \( \beta \) as bifurcation parameter. An initial estimate of \( I^* \) for the case \( \beta = 0 \) was obtained from numerical integration.

### 3.6. Discretization

As mentioned in Section 3.5, for numerical continuation the function \( \mu_c(x) \) is represented by its values at the fixed sizes \( x_1, x_2, \ldots, x_k \). Here we show how in principle from given estimates \( \mu_c(x_i) \) and \( \gamma_c(x_i) \) new estimates can be calculated. To facilitate the distinction between the size \( x(a) \) of the ‘focus’ individual, whose life history is calculated, and the fixed sizes used in the discretization, we denote the fixed sizes by \( y_i \) rather than \( x_i \). To avoid excessive notation we suppress the dependence of functions on the input variable \( I \).

During the integration of (8)–(10), we can calculate the cumulative cannibalistic mortality rate inflicted on a victim of length \( y_i \), denoted by \( M_i(a) \), as

\[
\frac{dM_i(a)}{da} = \frac{\beta x(a)^2 T(x(a), y_i)}{1 + H(x(a)\gamma(x(a)))} S(a) P, \quad M_i(0) = 0.
\]

A new estimate of \( \mu_c(y_i) \) is then given by \( \mu_c(y_i)' = M_i(\infty) \). Note that \( x \) refers to cannibal length, and that the function \( \gamma(x) \) occurs in the right-hand side of this differential equation. The latter means that the current, discretized estimate \( \gamma_c(y_i) \) is used in the calculation of the new estimate \( \mu_c(y_i)' \). Also, the current estimate \( \mu_c(y_i) \) is used in this calculation, because it affects \( S(a) \).

In a similar way the cumulative mass-encounter rate, \( G_i(a) \), with a cannibal of fixed length \( y_i \) can be obtained by solving

\[
\frac{dG_i(a)}{a} = \frac{\beta y_i^2 T(y_i, a)}{1 + H(y_i)\gamma(y_i)} w(x(a)) S(a) P, \quad G_i(0) = 0.
\]

Note that \( x \) is now victim length. The new estimate of \( \gamma_c(y_i) \) is then given by \( \gamma_c(y_i)' = G_i(\infty) \). Again, the current estimates \( \gamma_c(y_i) \) and \( \mu_c(y_i) \) have to be used in the right-hand side of this ODE.

Since the functions \( \mu_c(x) \) and \( \gamma_c(x) \) are represented at \( k \) fixed body lengths, we need to solve \( 2k \) ODEs simultaneously with (8)–(10). Although this is possible, it is computationally costly if the number of discretization points \( x_1, x_2, \ldots, x_k \) is large. An alternative method is based on the idea that during the calculation of the life history we can reconstruct the entire population size distribution (see Section 3.3 and below). This allows for the derivation of a discrete approximation to the function \( n(x) \) while integrating the ODEs (8)–(10). The values \( \gamma_c(y_i) \) and \( \mu_c(y_i) \) of the cannibalistic encounter rate and cannibalistic mortality rate at length \( y_i \) can subsequently be obtained by substituting for \( n(x) \) its discrete approximation into (2) and (6), respectively. This method is computationally more efficient and was used to obtain the results in Section 4.

Theoretically, the precise choice of the discretization points does not matter although the accuracy of the approximation obviously depends on their resolution. In our calculations we chose them equidistant as \( y_i = x_{\text{hi}} + i\Delta \) with \( \Delta = 1 \) mm (note that \( x_{\text{hi}} = 7 \) mm and \( x_{\text{max}} \) often lies between 350 and 350 mm). After some initial experimentation we found that \( \Delta = 1 \) mm generally
gave a reliable result without causing too much computational effort. With too large a value of \( \lambda \) the continuation process failed to find an equilibrium.

Analogous to the representation of the size distribution in the EBT method for numerical integration (de Roos et al., 1992), the continuous population size distribution is approximated by a number \( (q) \) of delta-functions: we divide the size distribution into \( q \) length classes of width \( \Delta \) and represent each length class by the total number of individuals it contains, denoted \( N_i \),

\[
N_i = \int_{x_b + (i-1)\Delta}^{x_b + i\Delta} \rho(x) \, dx \quad \text{for } i = 1 \ldots q,
\]

and by the average length of these individuals, denoted \( X_i \),

\[
X_i = \frac{1}{N_i} \int_{x_b + (i-1)\Delta}^{x_b + i\Delta} x \, \rho(x) \, dx \quad \text{for } i = 1 \ldots q.
\]

The calculation of the life history provides us with exact values for \( N_i \) and \( X_i \). To calculate \( N_i \) we integrate the product of survival function and population birth rate,

\[
d\sigma = S(a)P, \quad \sigma(0) = 0,
\]

simultaneously with the integration of (8)–(10). The integration proceeds in stages over the length intervals \([x_b + (i-1)\Delta, x_b + i\Delta]\) and is stopped exactly at reaching the upper bound of this interval using an ODE integration technique with event localization (Hairer et al., 1993). Let \( a_i \) refer to the age at which the length \( x_b + i\Delta \) is reached, with \( i = 1, \ldots, q \) and \( a_0 = 0 \). The number of individuals \( N_i \) is then obtained as

\[
N_i = \sigma(a_i) - \sigma(a_{i-1}) \quad \text{for } i = 1, \ldots, q.
\]

For the calculation of the average length in each size class \( X_i \), we keep track of the integral of the product of length and survival,

\[
d\pi = x(a)S(a), \quad \pi(0) = 0.
\]

Then the average length is given by

\[
X_i = \frac{(\pi(a_i) - \pi(a_{i-1}))P}{N_i}
\]

The quantities \( N_i \) and \( X_i \) for \( i = 1, \ldots, q \) constitute an exact, discrete representation of the population size distribution \( \rho(x) \). Using this representation the value of \( \mu_e(y_i) \) can be approximated as

\[
\mu_e(y_i) \approx \sum_{j=1}^{q} \beta X_j^2 T(X_j, y_i) N_j
\]

where

\[
T(X_j, y_i) = A(X_j)R + \beta X_j^2 \sum_{k=1}^{q} T(X_j, X_k) X_k^2 N_k
\]

(cf. de Roos et al., 1992).

Similarly, an estimate of \( \gamma_e(y_i) \) can be obtained as

\[
\gamma_e(y_i) \approx \beta y_i^2 \sum_{j=1}^{q} T(y_i, X_j) X_j^2 N_j
\]

(cf. (2)). Note that the function \( T(y, x) \) equals 0 for \( y > x \). Therefore, when the value \( \gamma_e(y_i) \) is needed in the integration of the life history ODEs, all the quantities \( N_i \) and \( X_i \) that are involved in non-zero terms in the summation above have been calculated already. Hence, the values \( \gamma_e(y_i) \) can be obtained from the set of values \( N_i \) and \( X_i \) that are previously obtained in the integration and do not have to be specified as input variables.

4. Results

An obvious way to study the effect of cannibalism on population dynamics is to vary the strength of cannibalism, tuned by the parameter \( \beta \) (cf. Cushing, 1991; Claessen et al., 2000). However, recent model analysis has shown that the size-dependent nature of cannibalism, reflected in the cannibalism window \( T(x, y) \), has important implications as well (Claessen et al., 2002). In particular it appears that population dynamics are very sensitive to the lower limit of the cannibalism \( (\delta) \) but less so to the maximum \( (\epsilon) \) and the optimum \( (\phi) \) of the window (although \( \epsilon \) has a major effect on life history). Since empirical work has shown that the value of \( \delta \) varies between species of cannibalistic fish (Mittelbach and Persson, 1998), these results allow for between-species comparison of the effect of cannibalism on population dynamics (e.g., Claessen et al., 2002). Motivated by these findings we choose \( \beta \) as bifurcation parameter for \( \delta = 0.06 \) (the default for Eurasian perch) but repeat the continuation for various fixed values of \( \delta \).

The results of a continuation analysis with \( \beta \) as free parameter for a fixed \( \delta = 0.06 \) are shown in Fig. 1, in which the equilibrium state is characterized by the resource density \( R \), the population birth rate \( P \) and the ultimate length \( x_{\text{max}} \). The figure shows that initially \( P \) increases rapidly with \( \beta \), which is associated with an increase in per capita fecundity as well as an increase of the number of adults. The initial decrease of the alternative resource density is associated with an increase of the number of small individuals, which consume the resource (result not shown). The resource density then increases up to a maximum which reflects that the total consumption rate decreases up to that point. After \( \beta = 0.144 \), both \( P \) and the number of adults decline, whereas the average per capita fecundity increases with \( \beta \). The lower panel shows that the maximum size in the population always increases with \( \beta \), which relates to combined effect of more alternative
resource and the energy gain from cannibalism. The vertical, dotted line in Fig. 1 marks a critical value of $\beta$ where $R$ attains a maximum and the equilibrium curve changes slope abruptly ($\beta = 0.3316$). This point corresponds to the point where the maximum length in the population ($x_{\text{max}}$) equals the size for which the attack rate on the alternative resource becomes zero. This occurs at length $x_p$ which equals 160 mm (Tables 1 and 2).

To better understand the abrupt change around the critical value of $\beta$ we have a closer look at the population structure for two fixed values of $\beta$, below and above the critical value, respectively. For $\beta = 0.25$ and 0.4 the population structures are represented in Fig. 2. The figure shows the population size distribution $n(x)$, but also the input function $\mu_i(x)$ (6). For both values of $\beta$ the population size distribution is U-shaped. The reason is the high growth rate at intermediate sizes ($g(x)$, Fig. 2), which causes accumulation of individuals close to the ultimate size. In the lower panels the contributions from feeding on conspecifics and on the alternative resource to the individual growth rate $g(x)$ are indicated. With a $\beta$ below the critical value even the largest individuals feed on the alternative resource (Fig. 2a), whereas for a $\beta$ beyond it the largest individuals feed exclusively on conspecifics (Fig. 2b). The size interval for which individuals feed exclusively on conspecifics, that is $(x_p, x_{\text{max}})$, is referred to as the ‘piscivory niche’ (cf. Claessen et al., 2002). The size interval with a positive food intake rate from the alternative resource is referred to as the ‘planktivory niche’. Thus, the vertical dotted line in Fig. 1 marks the opening of the piscivory niche.

### 4.1. The effect of the cannibalism window

In our model the body length of the smallest victims that a cannibal can take is defined as a fraction $\delta$ of its own length ((2) and Table 2). For five values of $\delta$, Fig. 3 shows results of continuations with $\beta$ as free parameter. Note that Fig. 1 was made with $\delta = 0.06$, the estimate for Eurasian perch (Claessen et al., 2000). For reference, in Fig. 3 also the constant maturation size ($x_f$) and the maximum size of planktivory ($x_p$) are indicated, together with the maximum size ($x_{\text{max}}$) which depends on $\beta$. We make four observations from Fig. 3:

1. If $\beta$ is decreased to zero, the maximum length approaches $x_f$. 

![Fig. 1. One-parameter continuation of the equilibrium with $\beta$ (the cannibalistic voracity) as free parameter. Continuation started in the absence of cannibalism ($\beta = 0$, $R = 7.44 \times 10^{-5}$ and $P = 1.29 \times 10^{-5}$). Parameters: $\delta = 0.06$, Table 1.](image1)

![Fig. 2. Aspects of the equilibrium size-structure. (a) $\beta = 0.25$, (b) $\beta = 0.4$. Upper panels: population size-distribution $n(x)$. Middle panels: the interaction variables (22), representing the cannibalistic mortality rate $\mu_i(x)$. Lower panels: the growth rate subdivided by food type. The net growth rate (top border of colored area) is obtained by adding the intake rate (colored area) to the (negative) metabolic rate (lower border of colored area). For each $x$ the height of the colored area corresponds to the total energy intake rate. The contributions from planktivory (blue area) and piscivory (red area) are indicated. With $\beta = 0.25$, individuals cannot grow beyond the “planktivory niche”—even the largest individuals consume the alternative resource. With $\beta = 0.4$ individuals $x > 160$ mm are in the “piscivory niche”—they consume conspecifics exclusively.](image2)
2. If \( b \) is increased from zero and \( d \) is large, the maximum size \( x_{\text{max}} \) increases and at high \( b \) individuals reach giant sizes.

3. If \( b \) is increased from zero and \( d \) is small, the maximum size approaches \( x_p \), or a value just below it.

4. If \( b \) is increased from zero and \( d \) is intermediate the intersection of \( x_{\text{max}} \) with \( x_p \) is followed by a fold bifurcation. For any \( b \) in between this fold bifurcation and a second one (at lower \( b \)) there exist three equilibria.

Below we address these four results separately.

**Result 1.** To understand the first result, consider a population that consists of individuals that start reproducing at age \( A \) with constant rate \( \delta \). Assume a constant mortality rate \( \mu \). The lifetime reproduction is

\[
R_0 = \int_A^\infty \mu e^{-\mu a} \, da = \frac{\delta}{\mu} e^{-\mu A}
\]

The condition \( R_0 = 1 \) implies that the relation \( A = \frac{1}{\mu} \ln \left( \frac{\mu}{\delta} \right) \) must hold in equilibrium. From this we can see that if the mortality rate becomes very small, the equilibrium condition requires that \( A \) becomes very large and/or \( \delta \) becomes very small, i.e., population regulation occurs either through decreasing juvenile survival or adult fecundity.

Now consider our full model. One effect of letting \( \beta \) go to zero is that the total mortality rate decreases toward the background mortality rate. Of course, in this model both the maturation age and the birth rate are not parameters but they depend on the resource density \( R \). The age at maturation decreases with \( R \) and fecundity at a given size increases with \( R \). This correlated dependence of age at maturation and fecundity implies that if we assume \( \beta = 0 \) and let the background mortality rate \( \mu_0 \) become very small, the equilibrium condition can be fulfilled only by decreasing the resource density. The resource density in equilibrium, however, should be at least sufficiently high to attain an ultimate size larger than the maturation size. Analogous to the age-structured case discussed above, we can hence expect that with decreasing \( \mu_0 \) the resource density approaches the minimum density required to reach \( x_f \). In turn, this implies that the maximum size approaches \( x_f \) as the mortality rate approaches zero. We have verified this by choosing the background mortality rate as free parameter and assuming \( \beta = 0 \). Continuation shows that if \( \mu_0 \) is decreased and \( \beta = 0 \), the resource density indeed approaches the level at which growth becomes zero at size \( x_f \) (see also de Roos et al., 1990).

In conclusion, if we let the cannibalistic voracity \( \beta \) decrease to zero, the maximum length in the population approaches the maturation length because the total mortality rate becomes very small. This result therefore depends on our assumption of a low background mortality rate.

**Result 2.** In Fig. 3a and b the maximum size in the population is positively correlated with \( \beta \) over the entire range of \( \beta \). Before the intersection of \( x_{\text{max}} \) and \( x_p \) this is partly because the density of the alternative resource increases with \( \beta \) (cf. Fig. 1). Beyond this point, however, it is entirely due to the cannibalistic energy gain which increases with \( \beta \). The latter is true despite that the total biomass of the population decreases with \( \beta \).

**Result 3.** The convergence of the maximum length \( x_{\text{max}} \) to a value just below \( x_p \) when the cannibalistic voracity \( (\beta) \) is increased from zero (e.g., Fig. 3c) is easily explained if we assume that cannibals do not gain energy by eating conspecifics. A negligible energy gain from cannibalism may result if cannibals consume very small victims only, which is a possibility if \( \delta \) is sufficiently small. Killing conspecifics without an energy gain is often referred to as ‘infanticide’ (Hausfater and Hrdy, 1984). Under this assumption, increasing \( \beta \) merely increases the mortality rate of victims, resulting in a lower overall population density. As a consequence, the rate of consumption of alternative resource by the
populations decreases with $b$, and hence the density of this resource increases. As the resource approaches its carrying capacity the consumers attain a maximum size which turns out to be close to the size where the attack rate on this resource becomes zero, i.e., close to $x_p$.

If we make the assumption of infanticide and also that the resource is at its carrying capacity, then with the parameters in Table 1 the maximum size is $x_{\text{max}} = 151.86$ mm. In our model and assuming $d = 0$, the maximum size converges to $155.13$ mm when $b$ is increased (Fig. 3c). The difference between these two values is due to the gain from cannibalism in the latter case. Even though the density of victims decreases to zero when $b$ becomes very large, the per capita energy gain from cannibalism at a given size turns out to converge to a constant value. The reduced victim density appears to be exactly balanced by the increased cannibalistic attack rate, which scales with $b$.

With a gain from cannibalism, the asymptotic value of $x_{\text{max}}$ when $b$ becomes very large depends on $d$. For example, with $\delta = 0.03$ this value is $x = 156.89$, and with $\delta = 0.04$ it is even larger (cf. Fig. 3d and e). A heuristic explanation of this is that if $\delta$ is small, many victims will be killed while they are still very small and hence have low energetic value. Cannibals with a higher $\delta$ ‘spare’ the smallest victims, but consume them when they have become more nutritious. Thus, at the same overall rate of killing, the gain from cannibalism at a given size turns out to be larger with a larger $\delta$ because the captured victims are larger.

The fact that the asymptotic value (for $\beta \to \infty$) of $x_{\text{max}}$ increases with $\delta$ implies that there is a critical value of $\delta$, for which the maximum size in the population equals the maximum size for planktivory ($x_p$). A population with a maximum size so small that it is in the planktivory niche ($x_{\text{max}} < x_p$) we characterize as ‘stunted’. Above this critical $\delta$ the curve of $x_{\text{max}}$ intersects with $x_p$ at some value of $\beta$. For example, with $\delta = 0.042$ this intersection occurs at $\beta = 0.6555$ (Fig. 3c). Although in Fig. 3d it seems that with $\delta = 0.04$ the maximum size approaches an asymptotic value below $x_p$, it intersects with $x_p$ at $\beta = 3.40$. What happens after intersection with $x_p$ is the subject of the next paragraph.

**Result 4.** Whereas with $\delta = 0.1$ and 0.05 the intersection with $x_p$ merely increases the slope of the equilibrium curve, with $\delta = 0.042$ and 0.04 the intersection is followed by a fold bifurcation (Fig. 3). In the latter two cases, the fold bifurcation occurs at $\beta = 0.7131$ and 6.139, respectively. In both cases, the maximum size at the fold bifurcation is just above the maximum size in the planktivory niche, $x_{\text{max}} \approx 163$. There is a second fold bifurcation at a lower $\beta$, and in between the two folds there are three equilibria for each parameter combination.

If the size distribution of a population extends into the piscivory niche (i.e., $x_{\text{max}} > x_p$) we say the population is in the ‘piscivorous’ state, as opposed to the stunted population state in which piscivory (i.e., cannibalism) is only a minor contribution to the energy budget of individuals (e.g., Fig. 2a). Fig. 3 shows that the transition from the stunted to the piscivorous population states can be associated with a fold bifurcation. An important consequence of the occurrence of fold bifurcations in the equilibrium curve is bistability. That is, for a given set of parameters (i.e., a given species in given conditions) the population can be in either one of the two states; stunted or piscivorous. In the next section we study the stunted and piscivorous states in more detail, and address the question of what determines whether the intersection of $x_{\text{max}}$ and $x_p$ is followed by a fold bifurcation or not.

**4.2. Comparison of ‘stunted’ and ‘piscivorous’ population states**

In the regions with bistability (Fig. 3c and d) numerical integration of the model (Table 3) shows that the equilibria at the upper branch, corresponding with piscivorous population states (i.e., largest $x_{\text{max}}$), are stable near the left fold bifurcation only (Section 4.4). Equilibria at the middle branch (i.e., intermediate $x_{\text{max}}$) are found to be always unstable, while equilibria at the lower branch, corresponding to stunted population states, turn out to be stable. In this section we therefore restrict ourselves to comparing stunted population states from the lower equilibrium branch with the stable, piscivorous equilibrium states near the left fold bifurcation at the upper branch.

For a specific choice of the parameters in a region with bistability ($\delta = 0.04$ and $\beta = 0.7$, cf. Fig. 3d) we compare the stunted and the (upper) piscivorous population states ($x_{\text{max}} = 158.1$ and 295.4 mm, respectively). Fig. 4 shows different aspects of the size structure of the two states. First of all, the size distribution (Fig. 4a) is U-shaped in both cases but obviously wider in the piscivorous state. The total biomass of the structured population in the piscivorous state is approximately twice the total biomass of the population in the stunted state (not shown). Fig. 4a shows that this difference is associated with (i) a larger density of small individuals in the piscivorous state, and (ii) the existence of individuals in the piscivory niche. The aspects (i) and (ii) are not independent since more individuals in the piscivory niche leads to a higher population fecundity, and thus to a higher inflow of small individuals.

The different population size distributions in the two equilibrium states give rise to different size-specific, cannibalistic mortality rates (Fig. 4b). In the piscivorous
state the peak occurs at a higher victim size than in the stunted state ($x = 54.5$ versus $31.5$ mm; respectively). The magnitude of the peaks are comparable in both states ($0.096$ and $0.108$ day$^{-1}$ in piscivorous and stunted, respectively). Yet the shift to larger victim sizes gives cannibals in the piscivorous state a much larger gain from cannibalism than in the stunted state. Even if we discount the effect of the higher victim density in the piscivorous state, the expected benefit from cannibalizing a single individual is higher in the piscivorous population (i.e., $x = 158.1$ mm). Thicker solid: the largest cannibals in the piscivorous population ($x = 295.4$ mm). Thin solid: cannibals of length $x = 158.1$ mm in the piscivorous population. The distribution is scaled such that the area under the curve equals one.

The distribution is defined as a relative frequency (i.e., area under the curve equals one), and is weighted by victim mass. Fig. 4d shows the distribution of ingested conspecific prey mass over victim length for three types of individuals. It shows that for the largest cannibals in both population states the bulk of the ingested conspecific prey mass comes from victims that are smaller than 50 mm: 96% in the stunted state, and 69% in the piscivorous state. Yet in the piscivorous state the victim distribution is wider and the peak occurs at a larger victim length than in the stunted state. Even cannibals of the same size take larger victims in the piscivorous state than in the stunted state. This reflects that on average cannibals ‘spare’ victims until they have reached a larger size in the piscivorous state. Of course this is not the result of a choice of the cannibals but is entirely due to the different population size distributions in the two states. Combined with the higher victim density (Fig. 4a), this explains the larger ingestion rate from cannibalism and, eventually, the larger growth rate of cannibals in the piscivorous state than in the stunted state (Fig. 4c).

4.3. Costs and benefits of cannibalism

To quantify the verbal argument of ‘sparing’ victims by cannibals, we compare the costs and benefits of cannibalism for equilibria at the different values of $\delta$ and $\beta$ shown in Fig. 3. (Diekmann et al., 2003) derive expressions for the costs and benefits in case only adult individuals cannibalize juvenile conspecifics. We follow their derivation, adding corrections for the fact that in our model cannibalism by juvenile individuals can take place as well. Consider a newborn individual. One effect of cannibalism is that its survival probability is reduced, such that the survival probability up to a specific age is always lower in the presence of cannibalism. Let $S_c(a)$ denote the probability that the newborn individual has escaped cannibalism upon reaching age $a$. $S_c(a)$ can be calculated analogously to (8) by integration of the ODE:

$$\frac{dS_c}{da} = -\mu_c(x(a)) S_c(a),$$

with initial condition $S_c(0) = 1$. Note that in this ODE we only take into account the cannibalistic mortality $\mu_c(x(a))$. The costs of cannibalism can be represented by the probability that the newborn individual at some moment in its life falls victim to a cannibal (and hence does not die of background mortality). These
costs equal
\[ 1 - S_c(\infty). \] (33)

Considering a newborn individual the benefits can be represented by the expected per capita biomass loss due to cannibalism, given as
\[ \int_{0}^{\infty} \mu_c(x(a))w(x(a))S(a) \, da. \]

The value of this integral, and those following below, can be calculated using the approach presented in Section 3. This expected biomass loss to cannibalism can be expressed in terms of the quantity of food which is required for the production of a single offspring, that is, in units of
\[ c_p = \frac{c_r(1 - \kappa)c_a}{\lambda x_h^b}. \]

In this expression, \( c_a \) converts the amount of ingested biomass into the amount of assimilated biomass, \( 1 - \kappa \) equals the fraction of assimilated energy that an adult spends on reproduction and \( c_r \) represents the conversion efficiency with which offspring with weight \( \lambda x_h^b \) are produced from the energy allocated to reproduction. Since cannibalism by juvenile individuals occurs as well, not all biomass loss due to cannibalism is directly converted into new offspring. To correct for juvenile cannibalism we calculate the fraction of cannibalistic biomass loss ingested by juveniles as
\[ C_j = \frac{\int_{0}^{\infty} F_c(x(a))S(a) \, da}{\int_{0}^{\infty} F_c(x(a))S(a) \, da} \] (34)
in which \( F_c(x(a)) \) represents the cannibalistic assimilation rate:
\[ F_c(x) = c_a \frac{c_r(x)}{1 + H(x)Y(x)} \]
(cf. (1) and (2)) and \( a_j \) is the age at which the maturity threshold is reached, i.e.,
\[ x(a_j) = x_f. \]

Taken together, the benefits of cannibalism in terms of newly produced offspring equal
\[ \Phi = c_p(1 - C_j) \int_{0}^{\infty} \mu_c(x(a))w(x(a))S(a) \, da. \] (35)

We refer to the difference between the benefits and costs of cannibalism \( \Phi - (1 - S_c(\infty)) \) as the net benefit. This net benefit measures the balance between the additional reproduction and additional mortality due to cannibalism. It should be noted that this quantity only measures instantaneous effects of cannibalism; the indirect effect of additional growth is not included, but may be important since growth influences mortality, food intake and fecundity.

Fig. 3. First, this figure shows that the net benefit is always negative. Second, it shows that the net benefit sharply decreases from zero and reaches a minimum for values of \( \beta \) around 0.05. Third, for higher values of \( \beta \) the net benefit ultimately approaches zero, but remains significantly lower if \( \delta = 0 \). Fourth, if the equilibrium curve is folded, the figure clearly shows that in the piscivorous state the net benefit is close to zero but significantly higher than in the stunted state.

Obviously, for \( \beta = 0 \) both costs and benefits equal 0. As Fig. 5c shows, for low values of \( \beta \) up to 80% of all biomass loss due to cannibalism is ingested by juvenile individuals. These juvenile individuals do not produce offspring, but use the energy for the development of reproductive organs (see Section 2). In terms of new offspring the benefits of cannibalism are therefore low, while the costs in terms of the expected reduction in survival are increasing. Even though the net benefit in terms of additional reproduction and mortality is negative, cannibalism can be seen as a positive density-dependent mechanism for these values of \( \beta \) on the grounds that a cannibalistic population (i.e., one with \( \beta > 0 \)) can persist at lower values of alternative resource than a non-cannibalistic population (i.e., one with \( \beta = 0 \); see Fig. 5b). This positive effect apparently operates more strongly for larger values of \( \delta \) and is entirely due to the indirect benefit of cannibalism increasing juvenile (and adult) growth.

For larger values of \( \beta \) the density of alternative resource needed for persistence of a cannibalistic population is always larger than the resource density required by its non-cannibalistic counterpart (Fig. 5b). Hence, cannibalism acts as a negative density-dependent mechanism. The higher resource densities observed for higher \( \beta \) values imply that the net benefit of cannibalism in terms of additional reproduction and mortality is necessarily negative. This negative net benefit becomes negligible, however, with a piscivorous population state for very high values of \( \beta \). For these values of \( \beta \) adult reproduction on the basis of alternative resource intake becomes negligible (results not shown). Hence, the model approximates a situation in which the alternative resource is used for juvenile growth and development only, while adults have an exclusively cannibalistic diet.

Overall Fig. 5a shows that the net benefit of cannibalism is larger if \( \delta \) is larger, that is, if cannibals ‘spare’ victims until they have become more nutritious. Interestingly, this effect can also occur due to the population size distribution alone, as is shown for the cases with bistability (Fig. 5a). Here, a wider size distribution implies a larger net benefit. The occurrence of bistability and the associated two fold bifurcations suggests the presence of a positive feedback mechanism. A likely candidate for this mechanism is the fact that the
benefits of cannibalizing a large victim are higher in the sense that it leads to a higher production of new offspring as well as to increased growth of the cannibals, which in turn means that they will capture even larger victims. Since the piscivorous state depends on a high population fecundity, a high net benefit may be essential in maintaining this population state. In the piscivorous state even adult individuals are increasingly likely to become a victim of cannibalism (Fig. 5d). In comparison with the stunted population state, the piscivorous state is also characterized by that a larger fraction of the cannibalistic intake is ingested by juvenile individuals (Fig. 5c). Hence, cannibalism by both large, adult and small, juvenile individuals is higher in the piscivorous state at the expense of cannibalism by large juveniles and small adults.

4.4. Comparison with the EBT method

With the EBT method we have examined dynamics of our model for specific parameter values (e.g., Fig. 6). First of all, the congruence of the two methods confirms the validity of the continuation method laid out in Section 3. Second, the EBT method allows us to judge the local stability of the equilibria in different parts of the equilibrium curve. Close to the left fold bifurcation (e.g., $\beta_{fold} = 0.519$ in Fig. 3d), equilibria on the upper branch are stable whereas equilibria on the lower branch from the fold bifurcation (i.e., intermediate $x_{max}$) are not. This behavior is consistent with the exchange of stability which generically occurs at fold bifurcations (Hale and Koçak, 1991).

Fig. 6 also shows that the piscivorous equilibrium destabilizes at $\beta \approx 0.755$ and that a limit cycle exists. The amplitude of the cycle increases with $\beta$ and the cycle disappears at $\beta \approx 0.875$. Beyond this point trajectories started from the piscivorous state end up near the stunted state. With other choices of parameters a similar pattern was found (results not shown).

4.5. Robustness

We tested the robustness of our results by trying many different parameter combinations. Different values of $\xi$, $\mu_0$, $K$ and $x_p$ all gave the same qualitative pattern. An interesting result is that when $x_p$ is smaller, the $\delta$-range of bistability is larger (and shifted to higher $\delta$).

5. Discussion

5.1. Population dynamical consequences of cannibalism

It is well known that cannibalism may induce alternative stable states in structured population models (Botsford, 1981; Fisher, 1987; van den Bosch et al., 1988; Cushing, 1991). Fisher (1987) has found bistability in a discrete-time model of a fish population with size-dependent cannibalism and competition. The ultimate size that individuals reach differs between the two stable states. Interestingly, in the equilibrium with large individuals the population density is low whereas in the equilibrium with small individuals the density is high, a result opposite to our results. The ‘stunted’ state in his model is maintained by severe intracohort competition among young-of-the-year (YOY) individuals, which retards their own growth at high densities. Cannibalism is restricted to 1-year-old individuals on YOY and increases with the body size of cannibals. In this model, bistability occurs only if the rate of cannibalism increases sufficiently abruptly with cannibal length (Fisher, 1987). This can be understood as follows. A lower YOY density leads to less competition and hence larger, but fewer, cannibals next year. If the rate of cannibalism increases sufficiently rapidly with cannibal length, this outweighs the effect of the reduced number of cannibals, and the net effect of a lower YOY density is an increased YOY mortality. Thus, a sufficiently strong dependence of cannibalistic attack rate on cannibal body size leads to a positive feedback loop between current and next year YOY density, which can induce bistability.

A major conceptual difference between the way cannibalism is modeled in Fisher’s model and our model concerns the energy gain from cannibalism. Cannibalism in the model of Fisher (1987) can be considered infanticide (Hausfater and Hrdy, 1984) because it does not provide a direct energy gain. Consequently, individual growth and fecundity are not directly affected by cannibalism, only indirectly through competition.

In our model the energy gain from cannibalism creates a positive feedback loop via fecundity and growth. Basically, a high victim density may result in a large intake rate from cannibalism, leading to high fecundity, which in turn leads to a high victim density. The immediate effect of a large intake rate on instantaneous fecundity is complemented by an indirect effect via individual growth. A high intake results in a high individual growth rate, and hence large body sizes. Increased body size strengthens the feedback loop in two ways which relate to absolute and relative body size, respectively. First, with absolute body size the handling time per unit of prey mass decreases and the maximum cannibalistic attack rate increases (Table 2). Consequently, the intake rate generally increases with body size. Since fecundity is proportional to the intake rate (4), it also increases with body size. Second, the effect of relative body size is that larger individuals exploit their victims at a larger size and hence more efficiently. The net benefit of cannibalism is higher in the piscivory state than in the stunted state (Fig. 5), because in the
piscivory state cannibals ‘spare’ victims until they have become bigger. We refer to this effect as the ‘Hansel and Gretel’ effect. A cannibalistic selection mechanism that depends on the population size distribution, size-dependent cannibalism and individual growth: The net benefit of cannibalism increases as the size distribution of cannibals shifts to larger sizes. Positive feedback may arise from the positive mutual effect of the gain from cannibalism on individual growth. The feedback is amplified by the positive relation between body size and fecundity. The novelty in our model that allows for this mechanism to emerge is the combination of a number of elements that had already been studied before, but not in conjunction: energy gain from cannibalism, size-dependent cannibalism and food-dependent growth. If any of these elements is lacking, the Hansel and Gretel effect cannot be found.

Based on empirical data (Mittelbach and Persson, 1998; Persson et al., 2000) we assume in our model that a cannibal cannot capture conspecifics with body lengths smaller than a fraction $\delta$ of its own length. We can compare the results with different values of $\delta$ in the light of the ‘Hansel and Gretel’ effect. A cannibalistic population with a higher value of $\delta$ consumes on average larger victims, resulting in a higher net benefit of cannibalism (Fig. 5). If one would start with a population in the piscivorous state (e.g., with $\delta = 0.042$, $\beta = 0.6$) and if by some process the range of potential victims would be enlarged by decreasing the lower limit

Figs. (a) The net benefit of cannibalism as a function of the cannibalistic voracity $\beta$. The net benefit equals the difference between the cannibalistic benefits, i.e. the expected number of offspring per newborn individual resulting from cannibalism and the cannibalistic costs, i.e. probability that an individual during its lifetime falls victim to a cannibal (see Section 4.3). (b) Resource density. (c) Fraction of the biomass loss to cannibalism, which is ingested by juvenile individuals. (d) Probability for a just maturing individual to die of cannibalism during its adult life. Thick solid: $\delta = 0.1$; Dashed: $\delta = 0.05$; Thin solid: $\delta = 0.042$; Dotted: $\delta = 0.04$; Dot-dashed: $\delta = 0.0$.
of the cannibalism window, then at some critical point (in this case, $\delta = 0.033$) the piscivorous state would disappear through a fold bifurcation, leaving the population in the stunted state. This scenario illustrates the crucial role of postponing cannibalism for the maintenance of the piscivorous population state. As an example of such a process, removal of submerged vegetation reduces the opportunity of YOY to hide from cannibals and may effectively lead to a lower $\delta$. Alternatively, individuals with a lower $\delta$ (but otherwise the same) have larger access to food; one might expect, therefore, that natural selection favors individuals with lower $\delta$, resulting in evolution toward the fold bifurcation.

In our discussion we frequently make use of the distinction between cannibalism and infanticide, based on the presence or absence of an energy gain to cannibalism, respectively. From a modeling perspective, ignoring the energy gain from cannibalism can be a welcome simplification, which may explain its frequent usage (e.g., Hastings, 1987; Costantino et al., 1997; van den Bosch and Gabriel, 1997; Briggs et al., 2000). From a biological point of view infanticide may be a reasonable approximation to cannibalism. However, our results indicate that ‘infanticide’, in the form of a small or even negligible gain from cannibalism, may be the result of population dynamics, rather than a generic aspect of the species under study. In our model, if the cannibalistic population is in the ‘stunted’ state the energy gain from cannibalism plays an insignificant role. However, perturbing the system may cause the population to reach the piscivorous state, a phenomenon which would have been impossible to predict under the assumption of infanticide. Thus, a model that assumes infanticide as a stand-in for cannibalism may miss some interesting results.

5.2. Continuation versus simulation

In Section 3 we have described a method for numerical continuation of a physiologically structured population model with a complex (infinite dimensional) interaction environment. In Section 4, we have shown results obtained with this method. We think our results clearly illustrate how using this method can increase the level of understanding about the dynamics of a structured population model, as compared to studying simulations alone. First, since both stable and unstable equilibria can be continued, two fold bifurcations could be localized. Moreover, continuation revealed that both fold bifurcations lie on the same equilibrium curve (although the second fold bifurcation may disappear to infinity).

Second, Fig. 6 suggests that from numerical simulations alone one might conclude that piscivorous equilibria exist only in a small interval of parameter values, and do not exist at high values of the cannibalistic voracity ($\beta$). Our continuation study shows, however, that the equilibria do exist but that they are unstable after a limit cycle arises.

Third, the fact that we study equilibria allowed us to use the conditions for equilibrium to interpret observed patterns. We applied this in the explanations of results 1 and 3 (see Section 4.1). It allowed us to compare biologically meaningful summary statistics, such as the cannibalistic net benefit, over the entire range of parameter values. The latter would have been impossible in simulations of our model since equilibria are unstable for large portions of parameter space (e.g., Fig. 6), and the interpretation of ‘net benefit’ in the context of population cycles is a lot more complicated than in the case of equilibria. In short, the discovery of the ‘Hansel and Gretel effect’ as an explanation of bistability in cannibalistic populations has to be attributed to this new method.

Acknowledgments

We thank Lennart Persson for comments on an earlier version of this paper and Odo Diekmann for many inspiring discussion about the costs and benefits of cannibalism, as well as suggestions for improvement of the text. The manuscript has benefited from constructive comments from two anonymous referees and Philipp Getto. A.M. de Roos is financially supported by a grant from the Netherlands Organization for Scientific Research (NWO).
References


Henson, S., 1997. Cannibalism can be beneficial even when its mean yield is less than one. Theoret. Population Biol. 51 (2), 109–117.


Temperature-Driven Regime Shifts in the Dynamics of Size-Structured Populations

Jan Ohlberger, Eric Edeline, Leif Asbjørn Vollestad, Nils C. Stenseth, and David Claessen

1. Centre for Ecological and Evolutionary Synthesis (CEES), Department of Biology, University of Oslo, P.O. Box 1066 Blindern, 0316 Oslo, Norway; 2. Université Pierre et Marie Curie–Paris 6, Unité Mixte de Recherche (UMR; Centre National de la Recherche Scientifique) 7618, Laboratoire Biogéochimie et Ecologie des Milieux Continentaux, 46 rue d’Ulm, F-75230 Paris Cedex 05, France; 3. Environmental Research and Teaching Institute, Laboratoire d’Écologie et Evolution (UMR 7625), Ecole Normale Supérieure, 24 rue Lhomond, F-75230 Paris Cedex 05, France

Submitted May 18, 2010; Accepted October 19, 2010; Electronically published January 12, 2011

Online enhancement: appendix.

Abstract: Global warming impacts virtually all biota and ecosystems. Many of these impacts are mediated through direct effects of temperature on individual vital rates. Yet how this translates from the individual to the population level is still poorly understood, hampering the assessment of global warming impacts on population structure and dynamics. Here, we study the effects of temperature on intraspecific competition and cannibalism and the population dynamical consequences in a size-structured fish population. We use a physiologically structured consumer-resource model in which we explicitly model the temperature dependencies of the consumer vital rates and the resource population growth rate. Our model predicts that increased temperature decreases resource density despite higher resource growth rates, reflecting stronger intraspecific competition among consumers. At a critical temperature, the consumer population dynamics destabilize and shift from a stable equilibrium to competition-driven generation cycles that are dominated by recruits. As a consequence, maximum age decreases and the proportion of younger and smaller-sized fish increases. These model predictions support the hypothesis of decreasing mean body sizes due to increased temperatures. We conclude that in size-structured fish populations, global warming may increase competition, favor smaller size classes, and induce regime shifts that destabilize population and community dynamics.

Keywords: cannibalism, competition, global warming, population dynamics, PSPM.

Introduction

Understanding and predicting the ecological impacts of climate change has become a major goal of today’s science. Climate change is believed to have severe impacts on virtually all ecosystems and biota (Stenseth et al. 2002; Par-
dynamics of size-structured populations depend on the strengths and nature of inter-cohort and inter-age-class competition and that changes in the size scaling of physiological rates can alter the dynamics between population oscillations and stability (Persson et al. 1998). Further, the population dynamics depend on the potential for intra-specific predation or cannibalism (Claessen et al. 2000, 2002), a common phenomenon in animals (Fox 1975; Smith and Ray 1991). It has been shown theoretically and empirically that the interplay between these two size-dependent interactions can lead to major shifts in population dynamics (Persson et al. 2003). Although it is known that individual-level processes critically depend on temperature, its effects on size-dependent competition and cannibalism have not been studied before.

In a previous attempt, Vasseur and McCann (2005) used a simple bioenergetics model to determine the influence of temperature on a consumer-resource interaction in order to predict the consequences of temperature changes on the dynamics and persistence of consumer populations. Their results indicate that warming is likely to destabilize consumer-resource interactions and that the qualitative response of a population depends on whether individual metabolic rate increases faster or slower with temperature than ingestion rate. Their model is a first step toward a bioenergetics theory of the impact of climate change on food web dynamics, but it has two critical shortcomings, which are the absence of size structure and the simplifying assumption about the temperature dependence of biological rates that does not allow for a higher metabolic efficiency of large compared to small individuals in cold environments (Kozlowski et al. 2004). Van de Wolfshaar et al. (2008) present the first size-structured population model that accounts explicitly for temperature effects on vital rates. They show that the temperature and size dependence of vital rates may have fatal consequences for winter survival of both individuals and the population as a whole. However, they did not study the effect of changing temperature on population dynamics.

Here we investigate the impact of temperature on intraspecific competition and cannibalism, levels of resource exploitation, and population size structure and dynamics. We calibrate our model to Eurasian perch (Perca fluviatilis L., subsequently referred to as “perch”), a common freshwater fish for which abundant physiological data and a validated population model exist. The basic model for our study is described in Claessen et al. (2000) and builds on a model presented in Persson et al. (1998). Their models correctly predicted growth trajectories and population dynamics observed in natural populations (Sanderson et al. 1999; Persson et al. 2000, 2004; Svanbäck and Persson 2009), thereby showing that the studied effects of intraspecific interactions on population dynamics reflect common characteristics of size-structured populations. To this model we add general temperature dependencies of individual physiological rates. With this study, we aim for a mechanistic understanding of population-level consequences of increasing temperatures due to global warming.

**Model Description**

We model the effects of temperature on the dynamics of a cannibalistic consumer population using a physiologically structured population model based on individual-level processes (Metz and Diekmann 1986; de Roos et al. 1992). The individual-level processes are determined by the consumers’ physiological state and interactions with their environment (consumer-resource interactions), whereas the population state is the distribution of individuals over all possible individual states. Our model is based on the size-structured consumer-resource model described by Persson et al. (1998), with added cannibalistic interactions among the consumers, as presented by Claessen et al. (2000). This model has been calibrated to perch using empirically derived functions and parameter values to describe the physiological state of an individual fish.

We extend this size-structured model by simulating the effects of temperature on both (i) the consumers’ individual physiological state through rates of food intake and metabolism and (ii) the population growth rate of the resource. Data for the effects of temperature on consumption and metabolism as well as the allometric scaling of these processes were taken from a bioenergetics model (Karas and Thoresson 1992) based on experimental data for perch (Lessmark 1983). All model equations and parameters are listed in tables 1 and 2, and a full description is presented in the appendix in the online edition of the *American Naturalist*. The model is simulated with the “escalator boxcar train” method (de Roos et al. 1992) using the software EBTtool (http://staff.science.uva.nl/~aroos).

**Individual-Level Processes**

Total body mass is composed of two physiological variables: irreversible mass, consisting of structural mass such as bones and vital organs that cannot be starved away, and reversible mass, consisting of energy reserves such as fat, muscle, and gonad tissue. Energy is acquired through food intake, lost through respiration, and converted to reversible and irreversible mass according to previously described allocation rules (Persson et al. 1998). The amount of net energy an individual can allocate to somatic or reproductive growth is the difference between energy acquired from food intake, accounting for conversion efficiency, and energy required for metabolism (fig. 1; tables 1, 2). Perch consumption consists of piscivory (cannibal-
<table>
<thead>
<tr>
<th>Symbol</th>
<th>Value</th>
<th>Unit</th>
<th>Interpretation</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Variables:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$s_i$</td>
<td>...</td>
<td>g</td>
<td>Irreversible mass of cohort $i$</td>
<td></td>
</tr>
<tr>
<td>$y_i$</td>
<td>...</td>
<td>g</td>
<td>Reversible mass of cohort $i$</td>
<td></td>
</tr>
<tr>
<td>$N_i$</td>
<td>...</td>
<td>individuals L$^{-1}$</td>
<td>Abundance of cohort $i$</td>
<td>Mitchell et al. 2004</td>
</tr>
<tr>
<td>$k$</td>
<td>...</td>
<td>...</td>
<td>Number of cohorts</td>
<td></td>
</tr>
<tr>
<td>$R$</td>
<td></td>
<td>L$^{-1}$</td>
<td>Resource population density</td>
<td></td>
</tr>
<tr>
<td>Environment:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$T$</td>
<td>Varied</td>
<td>°C</td>
<td>Temperature</td>
<td>Le Cren 1958</td>
</tr>
<tr>
<td>$S$</td>
<td>120</td>
<td>days</td>
<td>Length of growing season</td>
<td></td>
</tr>
<tr>
<td>Ontogeny and mortality:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$w_s$</td>
<td>.0018</td>
<td>g</td>
<td>Egg mass</td>
<td>Claessen et al. 2000</td>
</tr>
<tr>
<td>$x_i$</td>
<td>4.6</td>
<td>g</td>
<td>Maturation bone mass</td>
<td>Claessen et al. 2000</td>
</tr>
<tr>
<td>$q_i$</td>
<td>.74</td>
<td>...</td>
<td>Juvenile maximum condition</td>
<td>Claessen et al. 2000</td>
</tr>
<tr>
<td>$q_s$</td>
<td>1.37</td>
<td>...</td>
<td>Adult maximum condition</td>
<td>Claessen et al. 2000</td>
</tr>
<tr>
<td>$k_i$</td>
<td>.5</td>
<td>...</td>
<td>Gonad-offspring conversion</td>
<td>Claessen et al. 2000</td>
</tr>
<tr>
<td>$\lambda_1$</td>
<td>52.3</td>
<td>mm g $^{-\lambda_1}$</td>
<td>Allometric size scalar</td>
<td>Le Cren 1958</td>
</tr>
<tr>
<td>$\lambda_2$</td>
<td>.303</td>
<td>...</td>
<td>Allometric size exponent</td>
<td>Le Cren 1958</td>
</tr>
<tr>
<td>$\mu_0$</td>
<td>.01</td>
<td>day$^{-1}$</td>
<td>Background mortality rate</td>
<td>Claessen et al. 2000</td>
</tr>
<tr>
<td>$q_s$</td>
<td>.2</td>
<td>...</td>
<td>Starvation condition</td>
<td>Claessen et al. 2000</td>
</tr>
<tr>
<td>$s$</td>
<td>.2</td>
<td>day$^{-1}$</td>
<td>Starvation coefficient</td>
<td>Claessen et al. 2000</td>
</tr>
<tr>
<td>Consumption:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\alpha$</td>
<td>.62</td>
<td>...</td>
<td>Allometric exponent of planktivory</td>
<td>Claessen et al. 2000</td>
</tr>
<tr>
<td>$\hat{A}$</td>
<td>3.0E+4</td>
<td>L day$^{-1}$</td>
<td>Maximum zooplankton attack rate</td>
<td>Claessen et al. 2000</td>
</tr>
<tr>
<td>$w_{opt}$</td>
<td>8.2</td>
<td>g</td>
<td>Optimal forager size</td>
<td>Claessen et al. 2000</td>
</tr>
<tr>
<td>$\sigma$</td>
<td>.6</td>
<td>...</td>
<td>Allometric exponent of piscivory</td>
<td>Claessen et al. 2000</td>
</tr>
<tr>
<td>$\beta$</td>
<td>0, 100</td>
<td>L day$^{-1}$ mm$^2$</td>
<td>Cannibalistic voracity (with/without)</td>
<td></td>
</tr>
<tr>
<td>$\delta$</td>
<td>.06</td>
<td>mm$^{-1}$</td>
<td>Minimum victim/cannibal ratio</td>
<td>Claessen et al. 2000</td>
</tr>
<tr>
<td>$\epsilon$</td>
<td>.45</td>
<td>mm$^{-1}$</td>
<td>Maximum victim/cannibal ratio</td>
<td>Claessen et al. 2000</td>
</tr>
<tr>
<td>$\phi$</td>
<td>.2</td>
<td>mm$^{-1}$</td>
<td>Optimal victim/cannibal ratio</td>
<td>Claessen et al. 2000</td>
</tr>
<tr>
<td>$\xi_1$</td>
<td>5.0</td>
<td>day g$^{-(1+\xi_1)}$</td>
<td>Allometric scalar of handling</td>
<td>Claessen et al. 2000</td>
</tr>
<tr>
<td>$\xi_2$</td>
<td>-.8</td>
<td>...</td>
<td>Allometric exponent of handling</td>
<td>Claessen et al. 2000</td>
</tr>
<tr>
<td>$\theta_1$</td>
<td>2.8</td>
<td>...</td>
<td>Allometric scalar of $Q$,</td>
<td>Karås and Thoresson 1992</td>
</tr>
<tr>
<td>$\theta_2$</td>
<td>.072</td>
<td>...</td>
<td>Allometric exponent of $Q_s$</td>
<td>Karås and Thoresson 1992</td>
</tr>
<tr>
<td>$\gamma_{a, max}$</td>
<td>32.0</td>
<td>g$^{-\gamma_{a, max}}$ °C</td>
<td>Allometric scalar of $T_{max}$</td>
<td>Karås and Thoresson 1992</td>
</tr>
<tr>
<td>$r_{a, max}$</td>
<td>-.029</td>
<td>...</td>
<td>Allometric exponent of $T_{max}$</td>
<td>Karås and Thoresson 1992</td>
</tr>
<tr>
<td>$\gamma_{a, opt}$</td>
<td>28.0</td>
<td>g$^{-\gamma_{a, opt}}$ °C</td>
<td>Allometric scalar of $T_{opt}$</td>
<td>Karås and Thoresson 1992</td>
</tr>
<tr>
<td>$r_{a, opt}$</td>
<td>-.043</td>
<td>...</td>
<td>Allometric exponent of $T_{opt}$</td>
<td>Karås and Thoresson 1992</td>
</tr>
<tr>
<td>Metabolism:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\rho_1$</td>
<td>.033</td>
<td>g$^{(1-\rho_1)}$ day$^{-1}$</td>
<td>Allometric scalar of metabolism</td>
<td>Claessen et al. 2000</td>
</tr>
<tr>
<td>$\rho_2$</td>
<td>.77</td>
<td>...</td>
<td>Allometric exponent of metabolism</td>
<td>Claessen et al. 2000</td>
</tr>
<tr>
<td>$\theta_m$</td>
<td>2.0</td>
<td>...</td>
<td>Allometric scalar of $Q_m$</td>
<td>Karås and Thoresson 1992</td>
</tr>
<tr>
<td>$\theta_{m}$</td>
<td>.073</td>
<td>...</td>
<td>Allometric exponent of $Q_m$</td>
<td>Karås and Thoresson 1992</td>
</tr>
<tr>
<td>$\gamma_{m, max}$</td>
<td>36.5</td>
<td>g$^{-\gamma_{m, max}}$ °C</td>
<td>Allometric scalar of $T_{max}$</td>
<td>Karås and Thoresson 1992</td>
</tr>
<tr>
<td>$r_{m, max}$</td>
<td>-.013</td>
<td>...</td>
<td>Allometric exponent of $T_{max}$</td>
<td>Karås and Thoresson 1992</td>
</tr>
<tr>
<td>$\gamma_{m, opt}$</td>
<td>32.0</td>
<td>g$^{-\gamma_{m, opt}}$ °C</td>
<td>Allometric scalar of $T_{opt}$</td>
<td>Karås and Thoresson 1992</td>
</tr>
<tr>
<td>$r_{m, opt}$</td>
<td>-.029</td>
<td>...</td>
<td>Allometric exponent of $T_{opt}$</td>
<td>Karås and Thoresson 1992</td>
</tr>
<tr>
<td>Resource:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$K$</td>
<td>100</td>
<td>L$^{-1}$</td>
<td>Carrying capacity</td>
<td>Claessen et al. 2000</td>
</tr>
<tr>
<td>$M$</td>
<td>3.0E-5</td>
<td>g</td>
<td>Wet mass of zooplankton</td>
<td>Claessen et al. 2000</td>
</tr>
<tr>
<td>$r_s$</td>
<td>.58</td>
<td>day$^{-1}$</td>
<td>Maximum population growth rate</td>
<td>Mitchell et al. 2004</td>
</tr>
<tr>
<td>$r_{s, max}$</td>
<td>32.40</td>
<td>°C</td>
<td>$T_{max}$ for zooplankton growth</td>
<td>Mitchell et al. 2004</td>
</tr>
<tr>
<td>$r_{s, opt}$</td>
<td>27.54</td>
<td>°C</td>
<td>$T_{opt}$ for zooplankton growth</td>
<td>Mitchell et al. 2004</td>
</tr>
<tr>
<td>$Q_z$</td>
<td>1.799</td>
<td>...</td>
<td>Slope of zooplankton growth with $T$</td>
<td>Mitchell et al. 2004</td>
</tr>
</tbody>
</table>
Table 2: Model equations

<table>
<thead>
<tr>
<th>Name</th>
<th>Equation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Standardized weight</td>
<td>$w = x(1 + q_i)$</td>
</tr>
<tr>
<td>Length</td>
<td>$l = \lambda w^\delta$</td>
</tr>
<tr>
<td>Zooplankton attack rate</td>
<td>$A_i(x, y, T) = \frac{\lambda_w^x \exp \left(1 - \frac{w}{w_m} \right)^\delta}{w_m^\delta} r_i(x, y, T)$</td>
</tr>
</tbody>
</table>
| Cannibalistic attack rate| $A_c(x, y, T) = \begin{cases} 
\beta c^{x-y} (e^{\varphi c} - 1) & \text{if } \delta c < v < \varphi c \\
\beta c^{x-y} & \text{if } \varphi c < v < \delta c \\
0 & \text{otherwise}
\end{cases}$                             |
| Zooplankton encounter    | $\eta_i(x, y, T) = A_i(x, y, T) R_m$                                    |
| Cannibalistic encounter  | $\eta_i(x, y, T) = \Sigma_i A_i(x, y, y', T)(x + y')N_e$                |
| Total encounter rate     | $\eta(x, y, T) = \eta_i(x, y, T) + \eta_i(x, y, T)$                     |
| Handling time            | $H_i(x, y, T) = \xi_i w^x \left(1 - \frac{1}{w_m} \right)$              |
| Food intake rate         | $I_i(x, y, T) = \eta(x, y, T) \left(1 + H_i(x, y, T) \eta(x, y, T) \right)$ |
| Acquired energy          | $E_i(x, y, T) = k_i I_i(x, y, T)$                                       |
| Maintenance requirements | $E_m(x, y, T) = \rho_i (x + y)^{\alpha} r_i(x, y, T)$                    |
| Energy balance           | $E_i(x, y, T) = E_i(x, T) - E_m(x, y, T)$                               |
| Energy allocation rule   | $\kappa(x, y) = \begin{cases} 
\frac{1}{(1 + q_i)q_i} & \text{if } x \leq x_i \\
\frac{1}{(1 + q_i)q_i} & \text{if } x > x_i
\end{cases}$  |
| Fecundity                | $F(x, y) = \begin{cases} 
k(x, y)^{x-y}(x, y) & \text{if } x > x_i \text{ and } y > q_i x \\
0 & \text{otherwise}
\end{cases}$ |
| Total mortality          | $\mu(x, y, T) = \mu_i(x, y) + \mu_i(x, y, T)$                           |
| Starvation mortality     | $\mu_s(x, y) = \begin{cases} 
\delta(q_x y - 1) & \text{if } y < q_x \\
0 & \text{otherwise}
\end{cases}$  |
| Cannibalistic mortality  | $\mu_c(x, y) = \Sigma_i A_i(x, y, y, T) N_e$                            |
| Resource dynamics        | $\frac{dR}{dt} = r_i(T)(K - R) - R \Sigma_i A_i(x, y, y, T) N_e$       |
| Temperature dependence   | $r(x, y, T) = V(x, y, T) \left(1 - V(x, y, T)^{\frac{1}{T}} \right)$    |
|                         | $V(x, y, T) = \frac{(T_{\text{opt}}(x, y) - T)}{(T_{\text{opt}}(x, y) - T_{\text{opt}}(x, y))}$ |
|                         | $X(x, y, T) = W \left(1 + \left(1 + \frac{40}{T} \right)^{0.7} \right) \frac{1}{400} \cdot ^2$ |
|                         | $W = \left( T_{\text{opt}}(x, y) - T_{\text{opt}}(x, y) \right) \ln Q(x, y)$ |
|                         | $Y = \left( T_{\text{opt}}(x, y) - T_{\text{opt}}(x, y) + 2 \right) \ln Q(x, y)$ |
| Acquired energy          | $T_{\alpha, \text{opt}}(x, y) = \gamma_{\alpha, \text{opt}}(x + y)^{\alpha_{\text{opt}}}$ |
|                         | $T_{\alpha, \text{max}}(x, y) = \gamma_{\alpha, \text{max}}(x + y)^{\alpha_{\text{max}}}$ |
|                         | $Q_{\alpha}(x, y) = \theta_{\alpha}(x + y)^{\theta_{\alpha}}$          |
| Metabolism               | $T_{\alpha, \text{opt}}(x, y) = \gamma_{\alpha, \text{opt}}(x + y)^{\alpha_{\text{opt}}}$ |
|                         | $T_{\alpha, \text{max}}(x, y) = \gamma_{\alpha, \text{max}}(x + y)^{\alpha_{\text{max}}}$ |
|                         | $Q_{\alpha}(x, y) = \theta_{\alpha}(x + y)^{\theta_{\alpha}}$          |
| Resource growth          | $T_{\alpha, \text{max}}$, $T_{\alpha, \text{opt}}$, and $Q$, directly estimated (see table 1) |

* The parameter values 40 and 400 had to be changed to 20 and 200, respectively, in the temperature-dependence term for intake rate according to Karás and Thoresson (1992).
Figure 1: Illustration of the energy flow in a physiologically structured population model. The amount of net energy an individual can allocate to somatic or reproductive growth is the difference between energy acquired from food intake, accounting for conversion efficiency (losses), and energy required for metabolism.

Figure 2: Temperature-dependence terms (A) for perch consumption (solid lines), perch metabolism (dashed lines), and zooplankton growth rate (dotted lines); and the individual net energy gain (B) as a function of temperature for perch of body weight 0.1 g (solid lines), 1 g (dashed lines), and 10 g (dotted lines). Parameter values were taken from Karås and Thoresson (1992) and Lessmark (1983) for perch and estimated for zooplankton, based on data from Mitchell et al. (2004). Thin dotted lines (A) indicate calibration to a value of 1 at 20°C (see text). The body size of perch was set to 8.2 g, the optimal size for prey attack. The net energy gain for differently sized perch (B) was calculated at a zooplankton density of 2 individuals L⁻¹, which resembles rather low resource levels where exploitative competition in perch is expected to be high.
run the model for 500 years, sampling the population state yearly during the last 250 years (to omit transient dynamics). The obtained population data are plotted against temperature to obtain the bifurcation plot. This allows one to assess the amplitude and periodicity of population cycles, since a regular cycle at a given temperature will be characterized by a fixed number of dots spaced vertically (see figs. 3, 4). We performed bifurcation analyses over temperature for a noncannibalistic population (β = 0; see eq. [A19] in the appendix) and for various degrees of cannibalism (β = 0, 5, 10, 25, 50, 100, 300). Complementary bifurcation analyses over the cannibalistic voracity β were performed for discrete temperatures (12°–22°C, in 2°C steps). To further evaluate the model behavior, we used bifurcation analyses of maximum attack rate, digestion rate, and metabolic rate. To analyze the dynamics for a given temperature in detail, we ran our model at a fixed temperature for 100 years with a daily output and sampled during the last 25 years.

### Sensitivity Analysis

First, we analyzed the sensitivity of our results to changes in all parameters specifying the temperature dependence (all parameters new to our model; tables 1, 2), in order to account for the uncertainty in the measured parameters. We did this by performing bifurcation analyses for the cannibalistic and the noncannibalistic populations (see figs. 3, 4), for each parameter value increased or decreased by 10% (60 perturbations in total). We then examined changes in the population dynamics, both qualitatively (types and sequence of dynamics) and quantitatively (temperature at the onset of generation cycles).

Second, we analyzed the effect of changes in the scaling parameter of the attack rate (α) on our results for the noncannibalistic population. We did this, because it is known that α has a strong effect on the population dynamics and the potential for generation cycles because it determines the size dependence of the critical resource density (Persson et al. 1998). Values of α for freshwater fish preying on zooplankton that are reported in the literature range from 0.47 to 0.68, including for species such as pikeperch (Stizostedion lucioperca), roach (Rutilus rutilus), bream (Abramis brama), bluegill sunfish (Lepomis macrochirus), and arctic char (Salvelinus alpinus; Persson and de Roos 2006). Hence, we run our model within this range of α values to mimic the size scaling of attack rates of a variety of other species for which data were available (see table A2).

Third, we analyzed the effects of changes in the “cannibalism window” (δ, ε) on our results for the cannibalistic population. The cannibalism window strongly determines the intraspecific interactions and thus the dynamics of cannibalistic populations (Claessen et al. 2002). Changes in δ and ε were tested at the same time to mimic two other cannibalistic species for which empirical data exist (Persson et al. 2004), yellow perch (Perca flavescens), and pike (Esox lucius; see table A2).
Temperature Affects Population Dynamics

Results

Our model predicts that temperature has a major impact on the dynamics of size-structured populations. With increasing temperature, resource density decreases due to increasing food intake and increasing population birth rate in the consumer, which eventually shift the population dynamics from a stable fixed point to competition-driven generation cycles, irrespective of the degree of cannibalism (figs. 3, 4). These shifts in population dynamics are typically associated with an increase in competition (Persson et al. 1998; Claessen et al. 2000). A direct consequence of the different temperature dependencies of the vital rates at the individual level is that energy gain increases faster with temperature for small individuals (fig. 2B). Therefore, cool conditions favor big individuals, while warm conditions favor small ones. Increasing temperature hence reinforces the competitive advantage of small over large individuals, which enhances the mechanism that causes generation cycles (Persson et al. 1998). Below we present a detailed analysis of the model behavior for a cannibalistic and a noncannibalistic population.

Population Dynamics

The effect of temperature on the consumer and resource dynamics is exemplified in figure 4. It shows the bifurcation plots over temperature for a noncannibalistic (fig. 4A, 4B) and a cannibalistic (fig. 4C, 4D) consumer population and the respective resource densities.

In the noncannibalistic case (fig. 4A, 4B), the population dynamics shift from fixed-point dynamics to recruit-driven generation cycles at ~16.5°C. The fixed point, or stable equilibrium, at low temperature is characterized by small fluctuations in consumer and resource densities (fig. 3A, left). The pulses of newborns are almost constant over time, and the population consists of multiple coexisting cohorts. At low temperatures, the competitive asymmetry between small and large individuals is reduced and the total population fecundity is relatively low. Consequently, newborn cohorts are never sufficiently numerous to outcompete the adult cohorts, resulting in a uniform consumer age and size structure. Although the fixed point destabilizes slightly in a narrow, low-temperature range (12.2°C–13.5°C; fig. 4A, 4B), the population dynamics remain qualitatively similar to the FP dynamics in terms of size structure and level of competition. By contrast, as temperature increases, the resource density declines (fig. 4A) and competition among cohorts increases. Up to ~16.5°C, increasing temperature is associated with a considerable decline in resource density but not in consumer density (fig. 4A, 4B), indicating that the total food intake of the consumer increases. At this threshold temperature, total food intake and, as a consequence, population birth rate have increased sufficiently to allow certain newborn cohorts to deplete the resource to levels where they can still grow at a slow rate but mature individuals starve to death. Consequently, the population dynamics shift to recruit-driven generation cycles in which a single recruit year class dominates the population as it grows slowly throughout the years until it matures, reproduces, and is outcompeted by its offspring, the new dominating cohort (fig. 3A, right). These generation cycles have a 6-year periodicity (“horizontal” lines in fig. 4A, 4B), and they correspond to the recruit-driven single-cohort cycles first described by Persson et al. (1998). Interestingly, in the 13.5°C–16.5°C temperature range, the system is bistable, meaning that both types of dynamics (FP and GC) can be observed, depending on the initial conditions. In this range the amplitude of FP dynamics is sufficiently small to prevent the occurrence of high newborn pulses that would cause a shift to generation cycles. Beyond ~16.5°C, the amplitude of FP dynamics is such that high newborn pulses occur frequently, resulting in a rapid shift to GCs, whereas below ~13.5°C the GC dynamics are unstable because the newborn pulses are insufficient to outcompete the mature cohorts.

In the cannibalistic case (fig. 4C, 4D), population dynamics shift from a fixed point at temperatures below 13.5°C to irregular cannibal-driven dynamics at 13.5°C–15°C, to a mixture of cannibal-driven dynamics and generation cycles (CD-GC) around 15.5°C, and finally to sta-
ble generation cycles above 16.5°C. The temperatures at which these shifts occur depend on the degree of cannibalism in the population. Here we illustrate the dynamics for a cannibalistic voracity of \( \beta = 100 \) (see Claessen et al. 2000). At very low temperatures, we see fixed-point dynamics, but as temperature increases above 13.5°C, the fixed point destabilizes. At this point, juveniles can grow sufficiently quickly to cannibalize the recruits and thereby dominate the dynamics, which leads to the occurrence of cannibal-driven cycles at temperatures around 15°C. Figure 3B (left) shows an example of a 5-year CD cycle. The cycle is initiated by two high pulses of newborns that deplete the resource momentarily before being decimated quickly by cannibals. The resource level is thus relatively high throughout the cycle, indicating the absence of strong competition. The two abundant year classes nevertheless dominate the population structure, in particular by their strong cannibalistic impact on the survival of subsequent cohorts. Once background mortality has sufficiently reduced the abundance of the dominant year classes, new dominant year classes can arise. CD dynamics are thus driven not by competition for the resource but instead by cannibalism, which strongly reduces the abundance of the newborns, enabling the coexistence of many age classes. As temperature increases further (above 16.5°C), the population dynamics shift to stable generation cycles with a 7-year periodicity (“horizontal” lines in fig. 4C, 4D), which correspond to dwarfs-and-giants cycles (Claessen et al. 2000). These cycles are mainly driven by competition, in the same way as the GCs occur without cannibalism. The difference is that the first offspring cohort (fig. 3B), produced by the slowly growing “dwarf” cohort, is almost entirely cannibalized by its parental cohort. As a consequence, the resource level recovers quickly after the first offspring pulse (fig. 3B), ensuring the survival of the dwarf cohort, which subsequently reproduces a second offspring cohort the following year. This time, however, the dwarfs have become too big to consume the newborns, and the old dwarfs are thus outcompeted by this new dwarf cohort. The first offspring cohort, meanwhile, has reached a body size that allows them to cannibalize the new dwarf cohort and thereby grow to giant sizes (up to 800 mm; fig. 5) by the end of the cycle. The temperature range 15.5°–16.5°C represents mixed dynamics, in which CD dynamics and GC dynamics are both unstable and the system alternates between them (Claessen et al. 2000).

In both the cannibalistic and the noncannibalistic cases, increasing temperature is associated with a decreasing average zooplankton density with increasing variability (fig. 5A, 5B) despite a higher population growth rate. These changes reflect the above-described increased food consumption and thus competition within the consumer pop-

ulation as well as the onset of generation cycles at high temperatures.

**Individual Life-History Traits**

As a consequence of increased competition and the shift to generation cycles at high temperatures, average perch body size tends to decrease with temperature (fig. 5C). The trend is continuous and monotonic without cannibalism but discontinuous and oscillatory with cannibalism due to the occasional occurrence of giants at intermediate to high temperatures. Consequently, the relationship between maximum and average size and temperature is hump shaped in cannibalistic populations, and the variation in body size increases (fig. 5D). The lower temperature threshold for the occurrence of giants (length >250 mm) is ~14.5°C. Without cannibalism, growth is limited by planktivory and the fish never reach sizes above 200 mm, but as cannibalism becomes stronger, the fish grow larger and reach sizes of up to 800 mm within the 15.5°–20°C temperature range. Moreover, the number of coexisting year classes and the maximum age in the population decrease with temperature, most dramatically in the range 14°–16°C with cannibalism and around 16°C without cannibalism (fig. 6A).

**Sensitivity Analysis**

Our sensitivity analysis of the parameters in the temperature-dependence term shows that the model is qualitatively robust with respect to types and sequence of the dynamics for most parameters and that the onset temperature for generation cycles is more sensitive to perturbations in the noncannibalistic compared to the cannibalistic case (table A1). In the cannibalistic case, all but two of the possible perturbations result in the same sequence and types of dynamics (FP, CD, GC) as in the nonperturbed model, and in all but one case, the onset of generation cycles occurs within a similar temperature range of 15.5°–17°C. In the noncannibalistic case, all but one of the perturbations result in the same sequence of dynamics as in the nonperturbed model (FP, GC). However, the threshold for the onset of generation cycles appears to be more variable, with values ranging from 12.5° to 18°C. In both cases, four perturbations result in the population becoming extinct at relatively low temperatures (12.5°–15°C). Changes in those parameters related to the resource growth rate have virtually no effect on the population dynamics of the consumer. The perturbations slightly alter the population numbers but never shift the onset temperature for GCs by more than ~0.2°C. Interestingly, the strongest effects come from changes in the parameters related to consumption. Some of the pertur-
bations remove the FP dynamics at low temperatures, but they never remove the generation cycles at high temperatures within the 12°C–22°C range.

The sensitivity analysis of the scaling parameter of the attack rate (α) indicates that, in the noncannibalistic case, the dynamics do not change qualitatively when using known α values for other freshwater fish but that the temperature threshold above which the GCs are predicted varies between the mimicked species. It should be noted that, similar to the Eurasian perch parameterization, these systems are bistable, meaning that the types of dynamics at intermediate temperatures depend on the initial conditions. According to these tests, a lower α (as reported for pikeperch) results in lower onset temperatures, whereas a slightly higher α (as reported for arctic char and bluegill sunfish) results in higher onset temperatures for GCs. However, at even higher α (as reported for bream and roach), the FP dynamics first destabilize to multicohort, small-amplitude GCs and then to single-cohort GCs at intermediate temperatures, around 12.5°C–15°C (see table A2).

Finally, the sensitivity analysis of the cannibalism window shows that the values of the minimum and maximum victim/cannibal ratios (δ and ε) known for yellow perch result in the same dynamics as and a slightly lower onset temperature than in Eurasian perch, whereas when using the values for pike, the dynamics are similar but clear generation cycles never emerge (see table A2).

**Discussion**

Our model predicts that a temperature increase can induce regime shifts in the dynamics of size-structured populations and that global warming might destabilize ecosystems. As temperature increases, the consumer population shifts from stable dynamics with several coexisting year classes to competition-driven generation cycles in which population numbers fluctuate more severely in both frequency and amplitude. Additionally, our model predicts that warming increases the proportion of younger, smaller individuals. The driving force behind these changes is size-dependent, intraspecific competition for food, which changes with temperature.

*Temperature and Size Effects on the Individual Level*

Smaller individuals are generally competitively superior to larger ones because metabolic rate increases faster than foraging ability with body size (Persson 1987; Werner 1988). As a consequence of the different scaling relationships of the physiological rates, critical resource densities generally increase with increasing size, resulting in asymmetrical intraspecific competition (Persson et al. 1998). Thus, small individuals can usually grow at resource levels where large individuals starve to death. Indeed, this seems to be the case for all freshwater fish species for which the respective data exist (Persson and de Roos 2006). The size dependence of intraspecific competition, however, changes with temperature. The net energy gain of an individual increases faster with temperature at smaller sizes (fig. 2B), thereby magnifying the competitive advantage of small over large individuals. This effect is reflected in the general observation that optimum growth temperatures (which for immature fish can be assumed to be equal to those of the net energy gain) decrease with increasing body size (Kozłowski et al. 2004). This has been reported for several fish species (e.g., Karás and Thoresson 1992; Björnsson and Steinarsson 2002; Imsland et al. 2006) and other ectotherms (e.g., amphipods; Panov and McQueen 1998). Thus, this functional form of the temperature-size relationships can be assumed to be valid for other fish species and possibly ectotherms in general.

*Temperature-Driven Regime Shifts in Population Dynamics*

In line with the expected temperature effects on the individual level, we find more stable dynamics with several coexisting cohorts and a constant size distribution at lower temperatures where the difference between the size-dependent scaling of metabolic rate and intake rate, that is, the net energy gain, is smaller. At high temperatures, where the metabolic advantage of small individuals increases, recruit-driven single-cohort cycles appear in populations without cannibalism (Persson et al. 1998), and dwarfs-and-giants cycles appear in cannibalistic populations (Claessen et al. 2000). Thus, generation cycles consistently occur within our modeled populations at temperatures above ~16.5°C. Their onset is ultimately caused by an increase in total fecundity and thus population birth rate (fig. 6B), which results in stronger recruit year classes that deplete the resource to levels below the critical resource density for mature fish, which consequently starve to death (figs. 3, 4).

The impact of temperature on size-structured population dynamics are mediated through changes in physiological rates at the individual level that alter density-dependent interactions (competition and cannibalism) at the population level. However, abrupt transitions in population dynamics from stable to unstable states, as observed in our model, cannot be entirely understood based on individual-level processes that change with temperature in a continuous manner. The observed regime shifts in population dynamics within a few tenths of a degree suggest the existence of temperature thresholds, reflecting complex feedbacks between physiological rates, resource...
levels, population fecundity, and density-dependent processes. Such transitions might be less abrupt in natural systems due to the use of alternative food sources, interactions with other abiotic factors, or the spatial variation of competition between age classes within different microhabitats. Nonetheless, destabilization due to increasing temperature is a robust pattern that has never been studied empirically or theoretically. Similar to these size-structured consumer-resource models, theoretical models of cannibalism in size-structured populations yield some general predictions about the effects of cannibalistic interactions (Claessen et al. 2003). When cannibals and victims share a common resource, two mechanisms of size-dependent interaction may arise: either the cannibals control the victims or the victims control the cannibals. In the former case many cannibals of relatively small sizes cannibalize most of the victims, whereas in the latter case the victims can outcompete the cannibals so that most of them die and only a few can grow to giant sizes. Data from several fish species support these predictions, and it has been shown empirically that perch populations may actually switch between these two types of dynamics (Persson et al. 2003).

**Generality of the Observed Dynamics**

Theoretical models of varying complexity, from stage-structured to fully size-structured consumer-resource models, suggest that single-generation cycles are to be expected whenever intraspecific competition is asymmetric, with small individuals being competitively superior over large ones in exploiting the common resource (de Roos and Persson 2003). Accordingly, recruit- or juvenile-driven generation cycles have been observed in fish species, including roach (*Rutilus rutilus*) and vendace (*Coregonus albula*; Persson and de Roos 2006), as well as in *Daphnia* (McCauley et al. 1999; Nilsson et al. 2010). The similarity between different types of population models as well as the corresponding empirical data suggests that these effects of intraspecific competition on population dynamics reflect a general characteristic of size-structured populations. However, whether the occurrence of generation cycles depends on environmental temperature has never been studied empirically or theoretically. Similar to these size-structured consumer-resource models, theoretical models of cannibalism in size-structured populations yield some general predictions about the effects of cannibalistic interactions (Claessen et al. 2003). When cannibals and victims share a common resource, two mechanisms of size-dependent interaction may arise: either the cannibals control the victims or the victims control the cannibals. In the former case many cannibals of relatively small sizes cannibalize most of the victims, whereas in the latter case the victims can outcompete the cannibals so that most of them die and only a few can grow to giant sizes. Data from several fish species support these predictions, and it has been shown empirically that perch populations may actually switch between these two types of dynamics (Persson et al. 2003).
Sensitivity Analysis

The observed bifurcations in the noncannibalistic population are qualitatively robust to perturbations of the temperature-related parameters, but the onset temperature for generation cycles varies considerably. In contrast, the bifurcations for the cannibalistic population are qualitatively and quantitatively robust to parametric perturbations. The higher sensitivity in the noncannibalistic population may be explained by its bistability over a wide temperature range, as this means that the transition to GCs takes place whenever the fixed point is sufficiently perturbed. A slight increase in the bifurcation parameter may then be enough to trigger an attractor change, even if the FP attractor is still stable (but has a small basin of attraction).

Our sensitivity analysis of the bifurcations shows that our model generally yields the same results and conclusions if we mimic the size scaling of the attack rate or the cannibalism window of other fish species for which data exist. The types of dynamics that occur with increasing temperature are consistent for the different parameter values tested, with fixed-point dynamics at low and generation cycles at high temperatures. Nonetheless, the thresholds at which the dynamics shift vary depending on parameter value or mimicked species. That, however, does not challenge the conclusions drawn from our model results.

Temperature Effects on Age and Size Structure

The observed increase in competition and the destabilization of the population dynamics result in a higher proportion of smaller-sized fish and thus reduced average body size at high temperatures. This trend is continuous in the noncannibalistic population but discontinuous in the cannibalistic population (fig. 5C). These findings support the age-structure shift hypothesis according to which global warming might increase the proportion of young age classes and thus lead to a reduction in average body size in aquatic animals (Millien et al. 2006; Daufresne et al. 2009). Our model provides a mechanistic explanation for these findings by showing that increased temperature increases a metabolism-associated competitive advantage of small individuals over large ones. Warming-induced shifts towards smaller-sized populations represent a threat to population persistence since smaller size decreases diet breadth and fecundity but increases competitive ability and often predation risk (Arendt 2007). Therefore, warming might destabilize not only population dynamics but also whole ecosystems. It should be noted, however, that such ecological effects might be altered by evolutionary responses, since changes in size structure may also cause genetic changes related to life-history traits (Roff 2002).

The predictions of our model match well with observed changes in perch life-history traits with latitude. Size-dependent competition and cannibalism are barely considered in studies on life-history variation over latitudinal or temperature ranges (e.g., Bergmann’s rule; Ray 1960; Atkinson and Sibly 1997). However, both interactions control individual growth and body size (Fox 1975; Claessen et al. 2000) and may change with environmental temperature. In perch, various life-history traits have been shown to correlate with latitude and most likely temperature (Belk and Houston 2002; Heibo et al. 2005). For instance, Heibo et al. (2005) found a latitudinal cline for maximum age in perch that could be attributed to either an accelerated growth rate or a longer growing season at low latitudes and high temperatures (fig. 6A). This decline corresponds well to our model predictions of reduced maximum age and number of coexisting cohorts in the population due to the shift toward competition-driven, recruit-dominated dynamics. Since perch populations are potentially cannibalistic, the model performs well in predicting the general decline in maximum age. The cannibalistic case describes the smooth decline as seen in the data much better than the noncannibalistic case (fig. 6A).

Interestingly, maximum body size does not seem to correlate with latitude in perch (Belk and Houston 2002; Heibo et al. 2005). Instead, the biggest perch are found in lakes at intermediate degrees of latitude, thus corresponding to intermediate water temperatures (Heibo et al. 2005). This also matches with our model prediction of maximum body sizes for a cannibalistic population at intermediate temperatures (fig. 5D), whereas maximum size decreases continuously with temperature in the noncannibalistic population. Hence, cannibalism may invalidate the temperature to maximum size relationship. Since most families of freshwater teleosts have been described as at least partly cannibalistic (Smith and Reay 1991), this could explain why Bergmann’s rule does not apply to most freshwater fish (Belk and Houston 2002). Our results suggest that maximum body size in size-structured populations is determined by a combined effect of temperature and size-dependent competition and cannibalism. Our study suggests that the ecological mechanism causing the decline in mean body size and age at high temperatures is change in size-dependent intraspecific competition, whereas the mechanism explaining the occurrence of the largest individuals at intermediate temperatures is change in the cannibalistic interactions.

Conclusions

Due to the generality of both the size and the temperature dependence of the individual physiological rates that we
use in our model, we suggest that the predictions presented broadly apply to size-structured fish populations that are highly dependent on environmental temperature and possibly to aquatic ectotherms in general. We conclude from our model analysis that global warming might destabilize aquatic ecosystems through the onset of generation cycles that are characterized by strong recruit year classes with high intraspecific competition.

Acknowledgments

We thank Ø. Langangen and L. Rogers for helpful comments and discussions. We further want to thank L. Persson and two anonymous reviewers for their valuable and helpful comments on earlier versions of this manuscript. J.O. was supported by the Research Council of Norway.

Literature Cited


Associate Editor: Daniel Roelke
Editor: Ruth G. Shaw
Queries for ecol-92-12-09

This manuscript/text has been typeset from the submitted material. Please check this proof carefully to make sure there have been no font conversion errors or inadvertent formatting errors. Allen Press.
Stage-specific biomass overcompensation by juveniles in response to increased adult mortality in a wild fish population

JAN OHILBERGER,1,5 ØYSTEIN LANGANGEN,1 ERIC EDELING,2 DAVID CLAESSEN,3 IAN J. WINFIELD,4 NILS CHR. STENSETH,1 and L. ASBJORN VOLLESTAD1

1Centre for Ecological and Evolutionary Synthesis (CEES), Department of Biology, University of Oslo, P.O. Box 1066 Blindern, 0316 Oslo, Norway
2UPMC-Paris6, UMR (CNRS) 7618, Laboratoire Biogéochimie et Ecologie des Milieux Continentaux, 46 rue d’Ulm, F-75230 Paris Cedex 05, France
3Environmental Research and Teaching Institute, Laboratoire d’Ecologie et Evolution (UMR 7625), Ecole Normale Supérieure, 24 rue Lhomond, F-75230 Paris Cedex 05, France
4Centre for Ecology and Hydrology, Lancaster Environment Centre, Library Avenue, Bailrigg, Lancaster, Lancashire LA1 4AP United Kingdom

Abstract. Recently developed theoretical models of stage-structured consumer–resource systems have shown that stage-specific biomass overcompensation can arise in response to increased mortality rates. We parameterized a stage-structured population model to simulate the effects of increased adult mortality caused by a pathogen outbreak in the perch (Perca fluviatilis) population of Windermere (UK) in 1976. The model predicts biomass overcompensation by juveniles in response to increased adult mortality due to a shift in food-dependent growth and reproduction rates. Considering cannibalism between life stages in the model reinforces this compensatory response due to the release from predation on juveniles at high mortality rates. These model predictions are matched by our analysis of a 60-year time series of scientific monitoring of Windermere perch, which shows that the pathogen outbreak induced a strong decrease in adult biomass and a corresponding increase in juvenile biomass. Age-specific adult fecundity and size at age were higher after than before the disease outbreak, suggesting that the pathogen-induced mortality released adult perch from competition thereby increasing somatic and reproductive growth. Higher juvenile survival after the pathogen outbreak due to a release from cannibalism likely contributed to the observed biomass overcompensation. Our findings have general implications for predicting population and community level responses to increased size-selective mortality caused by exploitation or disease outbreaks.

Key words: biomass model; compensatory response; infectious disease; life history; maturation; mortality; pathogen; perch (Perca fluviatilis); reproduction; stage structure.

INTRODUCTION

Increasing mortality is expected to decrease population density because individuals are removed from the population. However, besides the negative direct effect on population numbers, mortality also releases the surviving individuals from competition for resources. If growth, maturation, and/or reproduction are food-dependent processes, this indirect density-dependent effect may lead to higher growth rates, faster maturation, and/or increased adult fecundity. Such indirect effects of mortality may result in increased stage-specific biomass production thereby compensating for the removal of individuals from the population (Werner and Gilliam 1984, De Roos et al. 2007). Experimental studies on laboratory populations have shown that stage-specific biomass remains unchanged or increases if not all individuals are subjected to increased mortality rates, for instance in water fleas (Slobodkin and Rickman 1956), blowflies (Nicholson 1957), soil mites (Cameron and Benton 2004), and fish (Schröder et al. 2009). Differences in mortality rates between size classes or life stages of natural populations are commonly observed in nature and may arise from size-specific predation (Brooks and Dodson 1965, Werner and Gilliam 1984), harvesting by humans (Law 2000, Fenberg and Roy 2008), or parasite infections (Ohlberger et al. 2011).

Recently developed theoretical models of stage-structured consumer–resource systems have shown that compensation (unchanged biomass) or overcompensation (increased biomass) in stage-specific biomass can arise in response to increased mortality rates (De Roos et al. 2007, 2008b). This phenomenon occurs in populations in which one or more of the life stages (e.g., juveniles and adults) are limited by intraspecific competition at high population densities. Biomass compensation or overcompensation emerges in the life
stage in which individual performance is not strongly limited by competition, independent of which stage experiences increased levels of mortality. When adults experience strong competition and dominate the population biomass regulation of reproduction occurs, whereas when juveniles predominate and compete intensely for resources regulation of maturation occurs (De Roos et al. 2007, 2008b, Huss and Nilsson 2011). If mortality increases in reproduction-regulated systems, the net effect of the release from competition is an accumulation of juvenile biomass, because the increase in individual fecundity is more important than the increase in juvenile growth and maturation. Stage-specific biomass overcompensation can have important consequences for interspecific interactions at the community level as it may affect, for instance, the performance of predators that specialize on a specific life stage (Persson et al. 2007, De Roos et al. 2008a). We may thus hypothesize that in natural systems density-dependent biomass overcompensation is an important process underlying population level responses to increased mortality caused by selective harvesting, predator increases or disease outbreaks. However, besides theoretical and experimental work on this topic, evidence of stage-specific overcompensation in natural populations is rare. Moreover, in cannibalistic populations, such as perch (Perca fluviatilis; Craig 1978, Le Cren 1992), the compensatory response to increased adult mortality might be modified through changes in cannibalistic interactions between the life stages.

In this paper, we investigate whether a massive pathogen outbreak in a natural fish population induced a compensatory response in stage-specific biomass production through indirect density-dependent effects. The perch population in Windermere (UK) experienced a considerable increase in mortality due to the outbreak of a perch-specific pathogen in 1976. Although the pathogenic organism itself has never been unambiguously identified, secondary infections have been described in detail (Pickering and Willoughby 1977). The pathogen induced a massive (98%) mortality in 1976 and the following years, with much higher prevalence among large, mature compared to small, immature individuals (Bucke et al. 1979) and with an estimated 10-fold increase in mortality after 1976 (Langangen et al. 2011). The population age structure remained severely truncated up to the early 2000s, indicating that the pathogen affected the perch population for many years (Edeline et al. 2008, Ohlberger et al. 2011b). However, population numbers did not collapse after the disease outbreak (Langangen et al. 2011), suggesting the incidence of a compensatory response at the population level.

We first present a stage-structured biomass model parameterized to our study system to predict the effect of the estimated increase in adult mortality rates on perch population biomass and age structure. The model predicts biomass overcompensation by juveniles in response to high adult mortality due to a shift in food-dependent growth and reproduction rates. Incorporating cannibalism into the model reinforces this compensatory response by further releasing adults from resource competition and by releasing juveniles from intraspecific predation. Second, using a 60-year time series of scientific monitoring of Windermere perch, we demonstrate that the high adult mortality after the pathogen outbreak was indeed associated with juvenile biomass overcompensation, with the consequence of a nearly unchanged average total population biomass. Third, a comparison of individual gonad weights showed that age-specific adult fecundity and growth increased after the disease outbreak, thereby indicating a competitive release. We argue that changes in intraspecific competition and cannibalism mediated the observed compensatory response by the juvenile stage in this population.

**Material and Methods**

**Model description**

We used a consumer–resource biomass model with a stage-structured consumer population as presented by De Roos et al. (2007, 2008b). This model is a simplified representation of a physiologically structured population model that accounts for a complete size-structure of the consumer population based on size-dependent individual vital rates, and thus directly translates individual-level assumptions to the population level. Juvenile (J) and adult (A) consumers feed on an unstructured resource (R) according to a type II functional response at maximum ingestion rate ($I_{max}$). The resource population follows semi-chemostat dynamics, in the absence of consumers, with turnover rate ($\delta$) and maximum biomass ($R_{max}$). The change in resource biomass is described by its intrinsic growth rate and the loss through consumption by juveniles and adults

$$\frac{dR}{dt} = \delta(R_{max} - R) - \frac{R}{R + 1}(I_{max}J + qI_{max}A)$$

where ($q$) scales the ingestion rate of adults to account for differences in foraging abilities of the two life stages, that is, whether adults are competitively superior to juveniles in terms of resource use ($q > 1$) or vice versa ($q < 1$). The net biomass production ($v$) of juveniles (j) and adults (a) depend on the assimilation efficiency ($\sigma$), food-dependent ingestion rate and maintenance rate ($T$). It is assumed that basic metabolic demands are met before energy is allocated to somatic or reproductive growth and that all surplus energy is allocated to somatic growth in juveniles, while all surplus energy is allocated to reproduction in adults. All consumers experience a mortality rate ($\mu$). The change in juvenile biomass is described as

$$\frac{dJ}{dt} = v^+J(R)A + vJ(R)J - \gamma(V^+J(R))J - \mu J$$
where \( v_j \) is the net biomass production of juveniles

\[
v_j(R) = \sigma \frac{qI_{\text{max}} R}{R + 1} - T
\]

with

\[
v_j^* (R) = v_j(R) \text{ if } R > \frac{1}{\sigma qI_{\text{max}} / T - 1}, \quad 0 \text{ otherwise. }
\]

Maturation rate (\( \gamma \)) depends on the newborn–adult size ratio (\( z \)), the net biomass production (which depends on resource abundance), and juvenile mortality rate

\[
\gamma (v_j^* (R)) = (v_j^* (R) - \mu_j) / (1 - z^{1-\mu_j/v_j^* (R)}).
\]

The change in adult biomass is described as

\[
\frac{dA}{dt} = \gamma (v_j^* (R))J + v_a(R)A - v_j^* (R)A - \mu_aA
\]

where \( v_a \) is the net biomass production of adults according to

\[
v_a(R) = \sigma qI_{\text{max}} \frac{R}{R + 1} - T
\]

with

\[
v_a^* (R) = v_a(R) \text{ if } R > \frac{1}{\sigma qI_{\text{max}} / T - 1}, \quad 0 \text{ otherwise. }
\]

We incorporate inter-stage cannibalism into the model by assuming that adults feed unselectively on the alternative resource and on juveniles. In contrast to the non-cannibalistic model, the change in resource biomass is then described as

\[
\frac{dR}{dt} = \delta (R_{\text{max}} - R) - \frac{R}{R + 1} (I_{\text{max}}J) - \frac{R}{R + J + 1} (qI_{\text{max}}A)
\]

the change in adult biomass is described as

\[
v_a(R, J) = \sigma qI_{\text{max}} \frac{R + J}{R + J + 1} - T
\]

with

\[
v_a^* (R, J) = v_a(R, J) \text{ if } R + J > \frac{1}{\sigma qI_{\text{max}} / T - 1}, \quad 0 \text{ otherwise}
\]

and juvenile mortality is described as

\[
\mu_j = \mu + \frac{qI_{\text{max}} A}{R + J + 1}.
\]

All other equations and all parameters are the same as presented for the non-cannibalistic version of the model.

Model parameterization

We parameterized the stage-structured biomass model to perch using observational data and published literature values. Maximum mass-specific ingestion rate (0.032) was calculated based on the mean body mass (90 g) of adult perch from our data. As in De Roos et al. (2008a), values for mass-specific maintenance and background mortality rates follow standard quarter-power scaling laws of adult body size with proportionality constants 0.01 and 0.001, respectively. Maximum ingestion rate was assumed to be 10 times larger than maintenance rate and 100 times larger than background mortality rate. The newborn–adult size ratio was set to 0.1 based on the size when Windermere perch start feeding on their main larval prey Daphnia (\( \sim 10 \) mm; Guma’a 1978) and the mean size at 50% maturation probability of male perch in Windermere (\( \sim 100 \) mm; Ohlberger et al. 2011b). Based on data on asymmetric competition between perch life stages (Persson and De Roos 2006, Ohlberger et al. 2011a) and in accordance with Nilsson et al. (2010), we set the default value for \( q \) to 0.8. Assimilation efficiency was set to 0.5 for adult perch in approximation to Karás and Thoresson (1992). Resource turnover rate and maximum density were set to 0.1 and 2.0 (De Roos et al. 2008a). The last two parameters scale the total biomass, but not the juvenile to adult biomass ratio.

To compare model results and observational data, we simulate a low and a high mortality case for a cannibalistic and a non-cannibalistic population. Based on the estimated increase in adult mortality in Windermere perch after the pathogen outbreak, we increase the background mortality rate in adults by a factor 10 (Langangen et al. 2011). In order to account for uncertainty in the estimated parameter values, we run the model 1000 times by re-sampling all parameters simultaneously and randomly within a uniform \pm 20% range of the default value. We used the equilibrium value of each run as model output.

Data collection

Windermere, a glacial valley lake in the English Lake District, UK, is divided into a north and south basin by shallows and islands (Le Cren 2001). The perch populations in the two basins are effectively independent according to capture–mark–recapture and genetic data (Kipling and Le Cren 1984, Bodaly et al. 1989). Perch is the most abundant fish species and is preyed upon by pike (Esox lucius), the top predator in the lake. The scientific monitoring of perch (and pike) was initiated in the mid-1940s and continues to date with very little change in gear type and fishing methods. Perch trapping takes place for 6 weeks during spring on the spawning grounds with standard traps that are unselective for perch of 90–300 mm total length (Le Cren et al. 1977). Individual fish (1942–2003, \( N = 105 763 \)) are measured and pike can be found in Le Cren (2001).
Data analyses

We estimated perch biomasses based on population numbers that were taken from recent population estimations for the years 1943–2002 (Langangen et al. 2011). These estimates were obtained by fitting age-structured population models to the 60-year time series of catch-at-age data, thus including long time series before and after the outbreak of the perch-specific pathogen in 1976. The age-structured model takes into account the full cohort and the non-linearity between catch and effort for estimating the population numbers (Langangen et al. 2011). Age-specific biomasses in each year were calculated by multiplying the population estimates by the average sex, age, and basin-specific body masses from the catch data. Since only very few 1-year-old fish were caught in the traps, their numbers were estimated assuming the same mortality for age 1 fish before and after the disease outbreak (this age group was much less affected by the pathogen; Bucke et al. 1979). This is a conservative approach to estimating age 1 fish biomass, because assuming higher mortality after 1976 increases the estimated number and thus biomass for this age-class after the disease outbreak. Biomasses for the 1-year-old fish were estimated by fitting von Bertalanffy growth curves to the observed mass data on older age classes using a length–weight relationship obtained from years in which individual length and weight were recorded (44 out of 60, including years before and after the disease outbreak). Stage-specific biomasses were calculated as juvenile and adult biomasses for each year from the 1–2 and the 3–6 year-old fish, respectively. Windermere perch typically mature at age 2 or age 3, with males maturing on average slightly earlier and at a smaller size than females (Craig 1977, Ohlberger et al. 2011b). Due to the abrupt (10-fold) increase in mortality and the corresponding change in the demography of the perch population in 1976 (Edeline et al. 2008, Langangen et al. 2011, Ohlberger et al. 2011b), we pooled the biomass data into two periods, a low mortality phase before (34 years) and a high mortality phase after the disease outbreak (25 years) and calculated mean biomasses for the two phases.

We used analysis of covariance (ANCOVA) to compare regressions of gonad weight to age relationships for female perch before and after the disease outbreak in both basins. Gonad weight data were log-transformed, because they were not normally distributed (Shapiro-Wilk normality test: $W = 0.8829, P < 0.001$). Total population fecundity could not be estimated reliably because the catch data are biased towards mature fish (trapping on spawning grounds), which introduces high levels of uncertainty when estimating age-class specific proportions of mature vs. immature fish, and due to a shift in age at maturity before vs. after the disease outbreak.

Fig. 1. Model results for (a) non-cannibalistic and (b) cannibalistic populations showing the juvenile (triangles), adult (circles), and total population (squares) biomasses at low and high mortality. High mortality refers to a 10 times higher adult mortality compared to juveniles, while all other parameters were the same in both cases. Symbols represent mean values and lower and upper error bars represent 5% and 95% quantiles from 1000 runs of simultaneously and randomly resampled parameters within a $\pm 20\%$ range of the default value.

RESULTS

The stage-structured consumer–resource model predicts biomass overcompensation by the juvenile stage in response to increased adult mortality for non-cannibalistic and cannibalistic populations (Fig. 1a, b). When background mortality in adults is low, adult biomass is higher than juvenile biomass, whereas juvenile biomass is higher when adult mortality is increased by a factor of 10. In the non-cannibalistic population, juvenile biomass production increases in response to high adult mortality due to higher total reproduction and higher juvenile growth rates (Fig. 1a). Total reproduction increases without but slightly decreases with cannibalism, while “per biomass” reproduction increases in both cases. In
the cannibalistic population, however, the release from predation on juveniles reinforces the compensatory response. As a consequence, total population biomass even increases at high compared to low adult mortality (Fig. 1b). Biomass overcompensation by juveniles, that is, a decrease in adult biomass and a corresponding increase in juvenile biomass, was found in all 1000 model runs in both cases. The described pattern is therefore remarkably robust to parameter perturbations (±20%), showing that these model results apply to a broad parameter space.

Our data analysis shows that the biomass of perch in both Windermere basins decreased considerably in the age classes 3–6, but increased in the age classes 1–2 after the invasion of the pathogen (Fig. 2a, b). Although biomasses of 3–6 year-old fish decreased by 88% and 77%, total population biomasses decreased by only 14% and 10% in the north and south basins, respectively. Hence, biomasses of 1–2 year-old fish increased nearly as much as the biomasses of adult fish decreased, demonstrating an overcompensatory response by the juvenile stage to the increased adult mortality. While the older age classes (3–6) were more abundant before, the younger age classes (1–2) were more abundant after the disease outbreak, and this pattern was relatively constant within each period despite large fluctuations in total population numbers (Fig. 2c, d). The juvenile-to-adult biomass ratio shifted from smaller than one to larger than one around 1976. The biomasses of 1–2 year-old fish (in percentage of total population biomass) increased from <20% before to >80% after the disease outbreak in both basins. Our analysis also showed biomass overcompensation by juveniles when 2-year-old male perch were classified as adults. The comparison of gonad weight data before and after the pathogen outbreak showed that age-specific fecundity increased significantly after 1976 (Fig. 3a, b). In both basins, the gonad weight to age relationships differed significantly in slope and intercept between the two periods (ANCOVA; north, \( F_{1,251} = 81.2, P < 0.001 \); south,


\[ F_{1,413} = 31.8, P < 0.001 \]. In contrast, the gonad mass to body mass relationships did not differ significantly (north, \( F_{1,251} = 0.44, P = 0.506 \); south, \( F_{1,413} = 2.71, P = 0.101 \)), indicating enhanced adult growth after the disease outbreak.

**Discussion**

Our data analysis demonstrates strong stage-specific overcompensation in juvenile perch in response to the drastically increased adult mortality induced by the disease outbreak, with a nearly unchanged average total population biomass. The stage-structured biomass model that we parameterized to our study system predicts biomass overcompensation in response to increased adult mortality due to food-dependent shifts in growth and reproduction and, in the presence of cannibalism, due to changes in cannibalistic interactions between the life stages. The increase in age-specific adult fecundity and size at age (with an unaltered mass–fecundity relationship) after the pathogen outbreak indicates that higher growth and reproduction rates contributed to the observed biomass overcompensation in Windermere perch. Enhanced adult growth rates may result from a competitive release in reproduction-regulated systems with indeterminate growth. Published data on zooplankton abundance in Windermere (George and Hewitt 1998) suggest a slight increase in resource levels from the mid-1970s onward. However, these data are only from the north basin, do not cover the entire time series, and their interpretation is complicated by the fact that phosphorus levels in the lake have also increased at the same time (Parker and Maberly 2000). Besides a release from competition for common resources, reduced inter-stage cannibalism may also have contributed to the observed overcompensation in juvenile biomass. It is known that perch undergo ontogenetic niche shifts from feeding on zooplankton to feeding on macroinvertebrates and finally to piscivory and cannibalism (Craig 1978, Le Cren 1992). Cannibalism may enhance the compensatory response in juvenile biomass production by releasing adults from competition thereby increasing energy availability for growth and reproduction, and/or by releasing juveniles from predation thereby increasing juvenile survival. Although small perch constitute a small proportion of total diet of large perch in Windermere (McCormack 1970), we suggest that a release from inter-stage cannibalism after the disease outbreak contributed to the observed biomass overcompensation by juveniles, because adult biomass decreased considerably and because perch cannibalize less when other resources are abundant (McCormack 1970).

Several factors and processes that may be of importance in generating the observed response in Windermere perch were not included into the model. First, we do not consider evolutionary change in life-history traits, such as changes in size at maturity. Indeed, length at maturity in perch has previously been shown to have slowly increased before but rapidly decreased after the pathogen outbreak, which may have contributed to a shift in reproduction rate (Ohlberger et al. 2011a). Although the smaller size at maturation increased the proportion of mature 2-year-old fish, this effect can be assumed to be negligible for the biomass analyses, because the increase was only a few percent (Ohlberger et al. 2011a). Second, Windermere perch were commercially fished at the beginning of our time series (see Appendix), continuing until 1948 in the north and until 1964 in the south basin (Le Cren et al. 1977). However, fishing effort was relatively low (7–28% of the fish were removed in any given year) compared to those years prior to our time series and tests have shown that our results do not change qualitatively when using only data collected after commercial fishing had stopped. Third, an introduced roach (Rutilus rutilus) population has expanded in Windermere since the early 1990s (Winfield et al. 2008), which might conceivably have contributed to a change in perch age-structure through intensified interspecific competition. Roach were not caught by gill net surveys in 1979 and 1980 (Craig and Fletcher 1981), and they subsequently comprised only about 4% of the fish community in 1995 and about 20% in 2000 (based on fish numbers from extensive gill net surveys; I. J. Winfield, unpublished data). Changes in the perch biomass distribution, however, occurred around the disease outbreak and perch population numbers did not decline in response to the roach expansion.

We used a simple stage-structured biomass model that predicts the observed biomass overcompensation in the juvenile stage as a result of a shift in food-dependent reproduction and growth rates and/or due to changes in cannibalism and thus juvenile survival. Here, we assume that all surplus energy in adults is diverted to reproduction. We have evaluated this assumption by introducing a parameter that scales the energy allocation to growth and reproduction and by studying model behavior as a function of this parameter. Biomass overcompensation by juveniles becomes weaker as more energy is allocated to growth in adults, but a compensatory effect occurs for all nonzero values of the allocation parameter. Hence, relaxing this assumption does not change the model results qualitatively. In our model we also assume asymmetric competition between life stages. This assumption is supported by physiological observations and modeling results suggesting that smaller-sized perch tend to be more efficient than larger ones in exploiting a common resource (Persson and De Roos 2006, Ohlberger et al. 2011a). De Roos et al. (2007) showed that populations are likely to be reproduction limited when juveniles are superior to adults with respect to resource competition (or have lower mortality) and that in such systems increases in mortality induce a compensatory effect in the juvenile stage. The extent of the compensatory response depends on the degree to which mortality rate increases, because juvenile biomass changes with adult mortality according
to a hump-shaped relationship (De Roos et al. 2007, Schröder et al. 2009). In other words, if adult mortality increases above a certain level, juvenile biomass also decreases and the population will eventually go extinct. In perch, the pathogen-induced increase in mortality rate was such that the compensatory response by the juveniles nearly matched the loss of biomass in the adult stage. As a consequence, average total population biomass remained nearly unchanged.

Compensatory responses in biomass distributions between ontogenetic life stages can have far-reaching consequences for population structure, community dynamics, and food web stability. On the population level, increased size-selective mortality changes the size structure of the population and may promote stage-specific biomass overcompensation if growth and reproduction are food-dependent processes (De Roos et al. 2007, 2008b). On the community level, changes in size-structure affect resource, competitor and predator populations through interspecific interactions. For instance, predators may either benefit or suffer from changes in prey size distribution, depending on which size-class or life stage they target. A predator may thus promote its own population growth by feeding on the size-class that shows the overcompensatory response in biomass (emergent Allee effect; De Roos and Persson 2002), or it may facilitate the existence of another predator by feeding on a different size class (emergent facilitation; De Roos et al. 2008a, Huss and Nilsson 2011). Accordingly, Persson et al. (2007) showed experimentally that culling old stunted fish of a prey population, Arctic charr (Salvelinus alpinus), caused an increase in the availability of small-sized fish and allowed the previously almost extinct predator, brown trout (Salmo trutta), to recover. In Windermere, the invasion of the perch-specific pathogen may have had a similar positive effect on the predator pike. Pike feed on most of the perch size-classes, and juvenile pike potentially compete with large perch for macroinvertebrates and small perch (Frost 1954, Edeline et al. 2008). Abundance of adult pike increased considerably around 5–10 years after the pathogen outbreak in perch (see Appendix; Langangen et al. 2011), and it was shown that the disease changed the effect of perch on pike recruitment from
negative to positive (Edeline et al. 2008, Langangen et al. 2011), thereby increasing pike population growth rate. Taken together, this suggests that the disease facilitated the high pike abundance by decreasing competition and increasing the biomass production of its prey. In contrast to theoretical models assuming adverse effects of pathogens and predators on a shared prey population (Hatcher et al. 2006), our results indicate that pathogens and predators might also enhance each other’s performance. As a consequence of these changes in community structure, predation by pike on perch may have increased considerably and thus contributed to the high levels of mortality among adult perch that have been observed since the outbreak of the pathogen.

The presented findings, together with recent theoretical developments and experimental work, have implications for both disease control and fisheries management. Our study suggests that natural populations may respond to increased mortality rates caused by infectious diseases or harvesting by stage-specific biomass overcompensation. This has management implications for at least two reasons: biomass overcompensation may result in counterintuitive measures necessary to restore fish stocks (Persson et al. 2007), and it may render marine protected areas less efficient than expected based on models without overcompensation (Claessen et al. 2009). The population-level response depends on the increase in mortality rates, the competitive interactions within and between life stages, and the environmental conditions that determine resource availability. More studies addressing such applied issues, particularly regarding the interplay between exploitation and environmental conditions, are clearly needed.

Acknowledgments

We would like to thank two anonymous reviewers who provided constructive comments on an earlier version of this manuscript. We are grateful to the Freshwater Biological Association for their joint stewardship of the long-term Windermere data. This work was supported by the Research Council of Norway and the Natural Environment Research Council of the UK.

Literature Cited


APPENDIX

A figure showing time series of population numbers and total fishing effort for pike and the two perch populations in the north and south basins of Windermere, modified from Langangen et al. (2011) (Ecological Archives XXXXX).
Population and Life-History Consequences of Within-Cohort Individual Variation

Manuela González-Suárez,1,* Jean-François Le Galliard,1,2 and David Claessen1,3

1. Centre National de la Recherche Scientifique, Université Pierre et Marie Curie, École Normale Supérieure (CNRS/UPMC/ENS), Unité Mixte de Recherche 7625, Écologie et Évolution, Université Pierre et Marie Curie, 7 Quai St. Bernard, 75005 Paris, France; 2. CNRS/UPMC/ENS, Unité Mixte de Service 3194, Centre de Recherche en Écologie Expérimentale et Prédicitive (CEREEP)—Écotron Île-de-France, École Normale Supérieure, 78 rue du Château, 77140 St-Pierre-lès-Nemours, France; 3. Centre d’Enseignement et de Recherches sur l’Environnement et la Société—Environmental Research and Teaching Institute (CERES-ERTI), École Normale Supérieure, 24 Rue Lhomond, 75005 Paris, France

Submitted March 10, 2011; Accepted June 17, 2011; Electronically published August 24, 2011

Online enhancements: appendixes, zip file. Dryad data: http://dx.doi.org/10.5061/dryad.jh87h.

ABSTRACT: The consequences of within-cohort (i.e., among-individual) variation for population dynamics are poorly understood, in particular for the case where life history is density dependent. We develop a physiologically structured population model that incorporates individual variation among and within cohorts and allows us to explore the intertwined relationship between individual life history and population dynamics. Our model is parameterized for the lizard Zootoca vivipara and reproduces well the species’ dynamics and life history. We explore two common mechanisms that generate within-cohort variation: variability in food intake and variability in birth date. Predicted population dynamics are inherently very stable and do not qualitatively change when either of these sources of individual variation is introduced. However, increased within-cohort variation in food intake leads to changes in morphology, with longer but skinnier individuals, even though mean food intake does not change. Morphological changes result from a seemingly universal nonlinear relationship between growth and resource availability but may become apparent only in environments with strongly fluctuating resources. Overall, our results highlight the importance of using a mechanistic framework to gain insights into how different sources of intraspecific variability translate into life-history and population-dynamic changes.

Keywords: demography, Jensen’s inequality, phenotypic plasticity, physiologically structured population models, stochasticity, Zootoca vivipara.

Introduction

Differences in age or stage are important sources of heterogeneity among individuals that are captured by structured population models (Caswell 2001). However, significant demographic variation can also be found within the same stage or age class (Huss et al. 2007). Heterogeneity among individuals may be caused by diverse factors, including differences in size or quality at birth or variation in resource abundance or climatic conditions (McNamara and Houston 1996). At the same time, some of these factors, for example, resource abundance, are affected by population size and structure. For instance, in organisms with food-dependent growth rates, when the population is dense, food levels decrease, thereby reducing individual growth rates. Population dynamics may hence determine which life histories are realized via changes in the environment. In turn, variation in individual life histories influences population dynamics (Benton et al. 2006). Thus, there is a feedback between life histories and population dynamics that is mediated by the environment.

Ecologists have long been interested in understanding how variation among individuals influences population dynamics and community ecology (recently reviewed by Bolnick et al. [2011]). A first set of studies has focused on the one-way influence of individual variation on population dynamics and, in particular, on the questions of whether variation stabilizes or destabilizes dynamics (e.g., Lomnicki 1978; Ebenman and Persson 1988; Grimm and Uchmanski 2002; Filin and Ovadia 2007) and how variation affects the risk of extinction (Kendall and Fox 2002; Vindenes et al. 2008). These questions have been explored with diverse population models (unstructured, structured, and individual based) and assumptions of various forms of variation among individuals, including differences in social rank, body size, or vital rates. A main conclusion of past analyses is that the relationship between individual variation and population stability is complex and usually nonmonotonic. For example, among-cohort variation can
have a stabilizing effect when population dynamics are inherently unstable, but the effect is destabilizing for inherently stable dynamics (Lindström and Kokko 2002). Likewise, individual variation in body size in grasshoppers appears to destabilize dynamics under a deterministic, constant environment, whereas the effect is stabilizing for a changing, stochastic environment (Filin and Ovadia 2007).

Another important conclusion is that variation in vital rates can lead to higher or lower extinction risks, depending on the mean-variance relationship in vital rates (Kendall and Fox 2002). Similarly, Vindenes et al. (2008) found that demographic stochasticity may increase, decrease, or remain unaltered, depending on the specific vital-rate values explored and how these rates vary over time among individuals. Altogether, these studies have shown that there is no unique effect of individual variation in population dynamics; instead, the predicted effects depend on how variation is modeled. Therefore, models that generate variation in vital rates or life history without considering the actual mechanisms generating this variation likely provide limited insight into how variation affects natural populations. Instead, biologically relevant conclusions require models that consider and explicitly include the mechanisms that give rise to individual variation.

Variation among individuals not only affects population stability but also can influence demographic traits such as growth, survival, and fecundity rates via feedbacks from population dynamics to individual life history. A second set of studies of individual variation, based on physiologically structured population models (hereafter PSPMs), acknowledges explicitly that the relationship between individual life history and population dynamics is intertwined (Metz and Diekmann 1986). PSPMs show that realized life histories and population dynamics are emergent properties of individual-level processes involving energy acquisition and allocation, social interactions, and so on. The main implications emerging from this work are that (1) the feedback between life history and population dynamics is likely to result in population cycles referred to as generation or cohort cycles (e.g., de Roos et al. 1992), (2) size-dependent trophic interactions influence the types of dynamics observed (e.g., Claessen et al. 2000), (3) realized life histories depend strongly on population dynamics (e.g., Claessen et al. 2000; de Roos et al. 2002), and (4) food-dependent growth may induce bistability and potentially a catastrophic population collapse (e.g., Persson et al. 2007). Traditional PSPMs account for variation among cohorts caused by environmental conditions experienced during life and plasticity in realized life histories (e.g., food-dependent growth) but ignore within-cohort variation. One exception is a study that demonstrates that stochastic within-cohort variability may dampen the amplitude of single generation cycles or lead to small-amplitude, stochastic fluctuations (van Kooten et al. 2007). Except for this study, the population-dynamic consequences of the life-history–population feedback in the context of within-cohort variation remain largely unexplored.

Our study aims to address this gap in our knowledge by exploring the question of how explicit consideration of mechanisms that generate within-cohort variability affects predicted population dynamics and life history while accounting for feedbacks. We use a detailed, predominantly mechanistic framework inspired by empirical understanding and knowledge about what causes individual variation in natural populations. In particular, we develop a PSPM that accounts for feedback of the population on life history, keeps tracks of each individual explicitly, and incorporates stochastic variation in individual fates (see also de Roos et al. 2009). Introducing within-cohort individual variation, we relax a common and strong assumption of the PSPM framework and obtain a model that is basically an explicit individual-based model (IBM). A common difference between studies using PSPMs and those using IBMs is that the former focus on long-term population dynamics and feedback, whereas the latter usually focus on a one-way effect of individual variability on population dynamics. Here, we try to unify these two approaches, making use of concepts and tools developed in the context of PSPM theory to analyze the interaction between individual variation and population feedback. Our model is based on the European common lizard Zootoca vivipara, for which there is strong evidence of phenotypic plasticity in growth rates, survival, and reproduction (Sorci et al. 1996; Lorenzon et al. 2001; Le Galliard et al. 2010).

We first present a deterministic model without individual variation and explore general model behavior under the assumption of a constant environment. Second, we develop four stochastic model versions aimed at unraveling the effects that two distinct mechanisms generating variation among individuals have on population dynamics and realized individual life histories. Important factors leading to individual differences include heritable life-history strategies (i.e., foraging or mating strategies), genetic variation in birth size or body condition, the consequences of differences in time of birth, and variation in climatic conditions, food availability, or habitat quality (reviewed by Le Galliard et al. 2010 for the common lizard). Among these, we consider stochastic variation in date of birth, which exposes different individuals to distinct environmental conditions during early life history (potentially affecting development) and generates a range of coexisting ages in each year class. In addition, we explore stochastic, among-individual variation of food availability as the most important and direct determinant of variability in individual resource acquisition and thus a potentially critical
factor leading to differences in individual growth and reproduction. The two mechanisms considered here, variation in birth date and variation in food availability, are major sources of individual variation in natural populations of reptiles and other species (Madsen and Shine 2000; Shine and Olsson 2003; Reznick et al. 2006; Sargent et al. 2007). We do not explore the role of heritable variation, although it is likely important, because mechanistically including heritable sources of variation requires an understanding of quantitative genetics that is not currently available for our model species. Finally, it is important to emphasize that although our model is species specific, our approach to modeling mechanisms of individual variation is very general. Therefore, our modeling exercise is likely relevant for a wide range of species.

Model Formulation and Parameterization

PSPMs explicitly link individual-level processes (growth, reproduction, mortality) and population dynamics (de Roos 1997). The life history of an individual is represented by a dynamic energy budget model that describes the acquisition of energy and its allocation to maintenance, growth, and reproduction, depending on individual physiological traits, or i-states, and the current state of the environment. Population functions, such as the total population biomass and the population size distribution, are derived by bookkeeping individual contributions, including birth, growth, and death. The mechanistic approach of PSMPs allows a unique understanding of causal relationships between individual-level processes and the resulting population dynamics. In addition, the PSPM framework allows a close connection between model and empirical system: the main model ingredients pertain to the individual level and can be parameterized with experimental data, while outputs, such as population dynamics and realized life history, are observable in the field.

Our PSPM of the common lizard is similar to earlier models of freshwater fish (Persson et al. 1998) and ungulates (de Roos et al. 2009) in its definition of the state of individuals (structural vs. reserves body mass), the use of a simple energy budget model to compute individual growth, and the specification of size-dependent functions for metabolic, food intake, and survival rates. Unique aspects, inspired by the biology of our model species, pertain to assumptions about energy-allocation rules, density dependence, the influence of weather conditions, and the sources of within-cohort variation among individuals (in the stochastic versions). These novelties are presented below, whereas a complete model description can be found in appendix A in the online edition of the American Naturalist.

Model Species

Zootoca vivipara is a small (newborn to adult snout-vent length: 20–70 mm), diurnal, nonterritorial Lacertidae species found in humid habitats across northern Eurasia. This lizard is an active forager that preys on a diversity of invertebrate species (Avery 1966). Populations may be oviparous or ovoviviparous, but we parameterize and validate our model with data from the latter (table 1). Natural populations can be divided into three main age classes: juveniles (<1 year old), yearlings (1–2 years old), and adults (>2 years old). Zootoca vivipara hibernate from September–October to March–April, exhibiting little winter activity and very low winter mortality (Bauwens 1981). Therefore, we explicitly model only the active season, ~200 days per year. Mating occurs soon after emergence from hibernation, but females retain the eggs in the abdominal cavity until embryonic development is completed. Females lay an average of five (range 1–12) noncalcified eggs from early June to early July, and eggs generally hatch within minutes of laying. Additional information on the species’ life history can be found in Massot et al. (1992) and references therein. We model only the female portion of the population and assume that individuals interact only by competition for a food resource.

Model Outline

The state of individuals is defined by three i-state variables: age, structural mass (i.e., bone, organs), and reserves mass (i.e., adipose and reproductive tissues). We assume that energy acquisition, growth, survival, and reproduction are functions of body mass defined by an energy budget model (app. A). Food intake and metabolism also depend on environmental conditions, that is, sunshine duration, to reflect the importance of weather on lizard life history (Adolph and Porter 1993). Food intake is also a function of a density-dependent scaling function $D(B)$ that provides feedback from population dynamics to the individual process of food consumption. Whereas PSPMs usually model the dynamics of the resource population explicitly, we cannot accurately model prey dynamics because the common lizard feeds on a large variety of prey and its functional response is not well understood (Avery 1966; González-Suárez et al. 2011). In the absence of enough empirical data to adequately define the consumer-resource interaction in this species, we model density dependence in a phenomenological way, using a simple function $D(B)$ that reflects our general knowledge of the species’ feeding biology. An individual’s feeding rate is obtained by multiplying its empirical, size-dependent feeding rate under standard conditions (see González-Suárez et al. 2011) by the function $D(B)$, which is a decreasing function of the
Table 1: Parameters of a physiologically structured population model of the lizard Zootoca vivipara

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Value</th>
<th>Units</th>
<th>Definition</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\lambda_1$</td>
<td>61.693</td>
<td>mm (g$^{3/11002}$)$^{-1}$</td>
<td>Allometric scalar</td>
<td>Unpublished data 1</td>
</tr>
<tr>
<td>$\lambda_2$</td>
<td>.303</td>
<td>...</td>
<td>Allometric exponent</td>
<td>Unpublished data 1</td>
</tr>
<tr>
<td>$W$</td>
<td>2.941</td>
<td>...</td>
<td>Dry to wet mass conversion</td>
<td>Avery 1971</td>
</tr>
<tr>
<td>$\gamma_1$</td>
<td>34.449</td>
<td>mg g$^{-2/11002}$ day$^{-1}$</td>
<td>Food intake scalar</td>
<td>Unpublished data 2</td>
</tr>
<tr>
<td>$\gamma_2$</td>
<td>.69</td>
<td>...</td>
<td>Food intake exponent</td>
<td>Unpublished data 2</td>
</tr>
<tr>
<td>$\delta$</td>
<td>4$^b$</td>
<td>h day$^{-1}$</td>
<td>Hours of sunshine per day</td>
<td>Standard conditions</td>
</tr>
<tr>
<td>$B_0$</td>
<td>1,500$^b$</td>
<td>g</td>
<td>Population biomass resulting in observed food intake rates</td>
<td>Free parameter</td>
</tr>
<tr>
<td>$\delta$</td>
<td>1$^b$</td>
<td>...</td>
<td>Density-dependent function coefficient</td>
<td>Free parameter</td>
</tr>
<tr>
<td>$\omega$</td>
<td>.303</td>
<td>...</td>
<td>Dry to wet mass conversion</td>
<td>Avery 1971</td>
</tr>
<tr>
<td>$q_{nt}$</td>
<td>.197</td>
<td>...</td>
<td>Neonate body condition</td>
<td>Nagy 1983</td>
</tr>
<tr>
<td>$q_{nt}$</td>
<td>.205</td>
<td>...</td>
<td>Body condition after reproduction</td>
<td>Nagy 1983</td>
</tr>
<tr>
<td>$q_s$</td>
<td>.084</td>
<td>...</td>
<td>Starvation body condition</td>
<td>Nagy 1983</td>
</tr>
<tr>
<td>$\tau$</td>
<td>.0226</td>
<td>kJ mg$^{-1}$</td>
<td>Prey mass conversion factor</td>
<td>Avery 1971</td>
</tr>
<tr>
<td>$\nu$</td>
<td>.82</td>
<td>...</td>
<td>Assimilation efficiency coefficient</td>
<td>Avery 1975</td>
</tr>
<tr>
<td>$\sigma$</td>
<td>1.0</td>
<td>kJ day$^{-1}$</td>
<td>Basal activity scope</td>
<td>Grant and Porter 1992</td>
</tr>
<tr>
<td>$\Phi$</td>
<td>.0286</td>
<td>g kJ$^{-1}$</td>
<td>Energy-to-structural-mass conversion coefficient (with costs of synthesis)</td>
<td>Avery 1971; Peterson et al. 1999</td>
</tr>
<tr>
<td>$\Psi$</td>
<td>.0210</td>
<td>g kJ$^{-1}$</td>
<td>Energy-to-reserves-mass conversion coefficient (with cost of synthesis)</td>
<td>Peterson et al. 1999; Kooijman 2000</td>
</tr>
</tbody>
</table>

Note: Values derive from a literature survey or unpublished data sets or cannot be parameterized a priori (“free parameter”). See “Model Parameterization.” All mass is given as dry mass.

* Default values defined for free parameters or standard conditions.

population’s weighted abundance $B$ (app. A). Although the exact mechanisms by which increased abundance affects individual food intake, and hence body growth, remain unknown, $D(B)$ appears to be a reasonable representation that largely captures the observed density-dependent response in body growth of $Z$. vivipara (app. B in the online edition of the American Naturalist).

Understanding how assimilated energy is actually channeled in an organism is complicated, and numerous energy allocation rules have been proposed (Kooijman 2000; Claessen et al. 2009). We assume that individuals follow a “net allocation model” (Kooijman 2000) before first reproduction and a “gross-production allocation model” (Kooijman 2000) after the first reproduction event (see app. A for details). These two allocation models reflect observed differences in prioritization between reproductive lizards,
which prioritize reproduction, and nonreproductive individuals, which prioritize structural growth (Andrews 1982).

**Model Implementation**

First, we describe a deterministic PSPM (Det) that follows cohorts of identical individuals over their life span. Cohorts may differ because of differences in the environmental conditions they encounter, but individuals within a cohort remain identical. We also present four individual-based stochastic versions of Det in which individuals, instead of cohorts, are followed. The first stochastic version (referred to as the Dis model) is a simple discretization of the Det model: each individual is represented explicitly, and its death and reproduction are modeled as discrete, stochastic events. Unless otherwise stated, all model assumptions and functions for Dis (and the other stochastic model versions) are as described in appendix A. For the Dis model, the time of death is determined through the expected-survival curve, computed for each individual with its time-varying total mortality rate ($\mu_0 + \mu_1 + \mu_3$; see app. A). The expected number of offspring depends on reserves mass, according to equation (A13) in the online edition of the American Naturalist. However, equation (A13) calculates real numbers that must be rounded into integers for use in the individual-based model versions. The simplest approach would be to round to the nearest integer, but this could introduce a bias. Therefore, in order to make sure that the expected fecundity exactly equals the fecundity based on the bioenergetic allocation rules (eq. [A13]; app. A), we round fecundity by using a simple probabilistic rule. If a female has, for example, an expected fecundity of 4.3, she produces either 5 offspring with probability 0.3 or 4 offspring with probability 0.7. The energetic investment of the mother is still defined as 4.3 in order to maintain a fully equivalent energy budget model, as in the Det model. As an artifact, this approach introduces limited stochasticity in the number of offspring per female. We have verified, however, that the overall distribution of offspring numbers per female predicted by the model generally captures the empirically observed distribution (app. C in the online edition of the American Naturalist). Note that in our models, the distribution of the number of offspring per female is a model prediction, not an a priori model assumption. Variability in the number of offspring, at a given time, results from the within-population variability in terms of age, size, and body condition of females and hence, indirectly, from their feeding history as well as from the (past) dynamics of the population through density-dependent feeding.

After discretization of the model, we introduce further modifications to explore two sources of within-cohort individual variation, first separately and then in combination. These additional stochastic model versions follow the same discretization approach as Dis. The Birth model modifies the Dis model to include a birthing period of 30 days instead of a single, population-wide birth pulse. The expected birth date for each female is drawn from a normal distribution with a mean equal to half the birthing-period length and a variance of half the mean, but truncated to span only the birthing period. The birthing day for each mother is assigned randomly each year to reflect variation in time of parturition of natural populations (J.-F. Le Galliard, unpublished data). Mature individuals with insufficient reserves to breed on their assigned date are given a second opportunity to reproduce on the last day of the breeding season. However, for the parameter values explored, more than 99.9% of the females that reproduce give birth on the originally assigned date. Results do not qualitatively change when different birthing-period lengths are considered (10–90 days).

The Food model modifies the Dis model by introducing temporal variation in prey availability for each lizard. For each individual separately, a stochastic food consumption factor is drawn daily from a normal distribution with a mean of 1 and a standard deviation reflecting observed variability in consumption rates of a lizard population (SD = 0.195; M. González-Suárez, unpublished data). Results do not generally change when the stochastic food consumption factor is drawn at longer intervals (2–15 days). A final model (F&B) combines both sources of individual variability.

We use a numerical integration method known as the Escalator Boxcar Train (EBT; de Roos et al. 1992), implemented in the EBTool software, to explore all these model versions. A general description of the EBT approach and the complete code files necessary to run the deterministic and stochastic model versions with the EBTool are available in appendix D in the online edition of the American Naturalist. We simulate population dynamics under varying initial conditions that have no effect on the stable dynamics. The code files in appendix D include the set of initial conditions used in most analyses. For each stochastic model version, we report results from 10 different simulations of population dynamics over a 300-year period. To discard transient dynamics, population statistics are calculated over the last 200 years. Additional details of the simulation methods are provided in appendix D.

Below, age is expressed in years such that 0+ represents the first year of life, 1+ the second year, and so on. For comparison with empirical estimates based on three age classes, we also present results grouped by ages 0+ (juveniles), 1+ (yearlings), and >1+ (adults). The term “mature individuals” refers to those with structural mass $x > x_{\text{mat}}$ (table 1), while “reproductive individuals” are those that have reproduced at least once.
Model Parameterization

Most parameters are defined on the basis of data for *Z. vivipara* from published sources (table 1) or a large unpublished data set (J.-F. Le Galliard, M. Massot, and J. Clobert, unpublished data) based on >1,200 *Z. vivipara* individuals from an experimental population at Centre de Recherche en Écologie Expérimentale et Prédicitive (CEREEP), France. We use the unpublished data set to estimate the length-to-mass relationship, fecundity rates, and body size at birth and at maturation and, in conjunction with published estimates of annual survival rates (Massot et al. 1992; Le Galliard et al. 2010), to define size-dependent daily mortality rates. The feeding rate is defined with data from a seminatural experiment at CEREEP based on >100 *Z. vivipara* (González-Suárez et al. 2011; M. González-Suárez, unpublished data). Standard conditions of sunshine duration at CEREEP during the lizard active season are used to define the climate-related parameters *sun* and *Msun*. When data from the common lizard are not available, we use information from related species (table 1). We use observations from the garter snake *Thamnophis sirtalis fitchi* to estimate the energetic costs of reserves- and structural-mass growth (Peterson et al. 1999). Data from the lizard *Uta stansburiana* is used to estimate condition thresholds (*q*~0*, q*~0*, and *q*~1*), defined as ratios of dry lipid and reproductive body mass over remaining dry mass (bones, organs, etc.) for different age classes and body conditions (Nagy 1983, pp. 32–33). These body condition thresholds are consistent with rough estimates calculated for *Z. vivipara* on the basis of dry lipid contents for adults (Avery 1970).

Data are not available to define some parameters (“free parameters” in table 1), and so we define their default values as follows. At equilibrium, the density-dependence parameters *B* and *δ* determine population size. Their default values are chosen so that size at equilibrium is approximately 100–150 individuals. Growth allocation rule parameters *k*~P~ and *k*~S~ are defined to broadly fit empirical patterns of individual growth, fecundity, and age of maturation from CEREEP (J.-F. Le Galliard, unpublished data). We explore the sensitivity of model predictions to changes in the default values of these free parameters. In addition, because observation error is possible, we also explore the sensitivity of the deterministic model predictions to changes in empirically determined parameter values.

Model Validation

There are difficulties in validating stochastic-model predictions (Waller et al. 2003; Grimm and Railsback 2005), and Grimm and Railsback (2005) suggest that simple comparisons are often best. We obtain empirical estimates independent of those used for model parameterization to calculate fecundity, survival, and body size. Empirical estimates (*Emp*) are calculated from a long-term data set (>11 years) of detailed individual measures of body size, clutch size, and a recapture series from a natural population in the Cévennes, France (Le Galliard et al. 2010; M. Massot and J.-F. Le Galliard, unpublished data). We estimate fecundity (number of female offspring per female) from total clutch size, assuming a 1:1 sex ratio. Survival estimates are calculated from mark-recapture histories, allowing for interannual variation and heterogeneous capture probabilities. Body size at fixed age is estimated from individuals of known age. These empirical estimates are compared with model predictions to address the question of whether observed data appear consistent with the model. In particular, we assess differences in central tendencies by determining whether empirical mean estimates fall within a narrow confidence interval (mean ± 1 SD) of model predictions. We explore data dispersion, comparing empirical and predicted variances with tests of homogeneity in variances. All unpublished data sets used to parameterize and validate the model are deposited at the Dryad Digital Repository (http://dx.doi.org/10.5061/dryad.jh87h).

Results

Deterministic PSPM

Model dynamics are insensitive to moderate changes in the empirically estimated parameters, and we find no evidence of alternative stable states. Bifurcation analyses reveal that long-term dynamics of our model remain relatively unaffected by changes in most free parameters or those defined by standard conditions (fig. 1B–1D). However, changes in *k*~P~ affect population dynamics noticeably (fig. 1A). The parameter *k*~P~ defines energy allocation to growth before first reproduction and thus determines the age at maturation. Stable fixed-point dynamics occur when all cohorts mature at the same time, while cycles or irregular dynamics are observed when cohorts mature at different ages (figs. 1A, 2). For example, for *k*~P~ = 0.42 (the default value), all individuals mature during their second year of life (fig. 2A), but for *k*~P~ = 0.32, some cohorts mature at age 1+ and others at age 2+, generating cyclic dynamics with years of higher and lower population sizes (fig. 2B). These 3-year cycles are akin to juvenile-driven cohort cycles (Persson et al. 1998), in that every three years a dense year class of newborns is born that reduces growth and reproduction of the older year classes. For *k*~P~ > 0.79, individuals reach the maturation size quickly but body condition remains below the reproductive threshold because growth of reserves mass is very limited. As a result, no reproduction occurs and popula-
Figure 1: Bifurcation diagrams of the free parameters and sun in the deterministic model. Each panel illustrates the results for a different parameter: A, energy allocation to structural growth in nonreproducing individuals $k_A$; B, energy allocation to structural growth in adults $k_A$; C, strength of density dependence $\delta$; and D, daily sunshine duration $\text{sun}$. Other parameters are set to their default values (see table 1). In A, the arrows indicate stable fixed-point regions in which all cohorts mature at the age indicated. The free parameter $B_0$ scales the population size linearly at equilibrium, and its bifurcation diagram is not shown.

Figure 1: Bifurcation diagrams of the free parameters and sun in the deterministic model. Each panel illustrates the results for a different parameter: A, energy allocation to structural growth in nonreproducing individuals $k_A$; B, energy allocation to structural growth in adults $k_A$; C, strength of density dependence $\delta$; and D, daily sunshine duration $\text{sun}$. Other parameters are set to their default values (see table 1). In A, the arrows indicate stable fixed-point regions in which all cohorts mature at the age indicated. The free parameter $B_0$ scales the population size linearly at equilibrium, and its bifurcation diagram is not shown.

Stochastic Individual-Based PSPM

The dynamic behavior of the stochastic versions of our model does not differ qualitatively from that of the deterministic version. Model runs starting with as few as 10 individuals show similar long-term behavior over 300 years. Results from the discretized model (Dis) and the deterministic model (Det) are also quantitatively very similar, although population size and mean fecundity are slightly lower in Dis (figs. 3A, 4A). On the other hand, introducing variation in birth date and food intake influences quantitative predictions. Scenarios including individual variation in birth date predict slightly larger median population sizes, while scenarios considering food intake variability predict a median population size lower than that from the Det or Dis model (fig. 3A). Combining both sources of variation (F&B), we find a median population size slightly higher than that from the Food model but still lower than that from the Det or Dis model. The age structure of the population on the last day of the season, defined as the relative proportion of individuals in each of the three age classes (0+, 1+, and >1+), is, however, nearly identical in all versions.

Mean fecundity is slightly reduced in all the stochastic model versions, with the lowest mean fecundity for F&B (fig. 4A). Reduced fecundity is accompanied by slightly higher survival rates of individuals of age 0+ (fig. 3B). Because there is no starvation mortality, older individuals...
are subject only to background mortality, and their survival rates do not differ among model versions.

Growth curves (snout-vent length, SVL) have a similar pattern in all model versions, with rapid growth in early stages of life and decreasing rates for older animals (fig. 4B). However, the mean SVL at fixed ages varies among versions. Older individuals are larger in the Food and F&B models. Variability in SVL among individuals within each age class is small in all versions (SD < 0.54 mm), but differences are more noticeable in models with food variation (Food and F&B) and among older age classes. The increase in mean SVL in older individuals in the Food and F&B models is accompanied by a decrease in body condition, so that these adults are characterized by being longer and skinnier. Variation in birth date alone (Birth) results in differences in SVL between individuals born early in the birthing period (first 10 days) and those born late (last 10 days), with the former being significantly larger at all ages and reaching larger maximum sizes (Student t-tests: \( P < .031 \)). Although significant, the actual differences in size are generally small (<1 mm). Interestingly, variability in food consumption eliminates these differences. There are no differences in SVL between earlier- and later-born individuals in the F&B model (Student t-test: \( P > .10 \)).

Maturation always occurs during the second year of life, but individuals mature slightly earlier in the Food model because of faster growth rates (mean \( \pm \) SD: Dis, 324 ± 23.5 days; Food, 313 ± 29.3 days; Birth, 331 ± 24.1 days; and F&B, 321 ± 28.8 days; ANOVA: \( F = 136.4, P < .001 \)). All mature individuals reproduce every year in all versions of our model. The Food and F&B models assume that consumption varies stochastically for each individual independently. Interestingly, an alternative model version assuming that food intake varies stochastically but simultaneously for all individuals (i.e., shared environmental stochasticity) predicts the same population-dynamic and life-history patterns, including the long-and-skinny syndrome, but eliminates variability among individuals.

**Model Validation**

Independent empirical observations are generally consistent with model predictions. All model versions predict multiple coexisting cohorts (age classes), as observed in

---

**Figure 2:** Time series of long-term dynamics predicted by the deterministic model for fixed-point dynamics (A; \( k_c = 0.42 \)) and cyclic dynamics (B; \( k_c = 0.32 \)). The top panels present the values of the density-dependence factor \( D(k) \) (see app. A in the online edition of the American Naturalist for detailed definition) that can be interpreted as “resource abundance.” The middle panels illustrate cohort abundance per age class, including newborns (<1 day old). The bottom panels present cohort growth curves (solid lines represent cohorts maturing at age 1+ and dot-dashed lines cohorts maturing at age 2+). The reference line in the bottom panel indicates body size at maturation. Other parameters are set to their default values (see table 1).
natural populations (Massot et al. 1992). The predicted age structure (fig. 3A) agrees well with empirical estimates (Massot et al. 1992). Mean empirical estimates of growth curves, fecundity, and survival rates fall within the range (mean ± 1 SD) of predicted estimates, except for a few estimates of SVL (figs. 3, 4). However, interannual variability in survival observed in natural populations is higher than predicted (F-test for homogeneity of variances: \( P < .01 \); fig. 3B). Similarly, observed variation in SVL for younger age classes (newborns and 200-day-old individuals) is greater than that predicted by the model (F-test for homogeneity of variances: \( P < .001 \); fig. 4B). Interestingly, observed variation in SVL for the older adult class is also greater than that predicted for models without variation in food intake (Dis and Birth; F-test for homogeneity of variances: \( P < .02 \)) but not greater than that for models in which food varies stochastically (Food and F&B; F-test for homogeneity of variances: \( P > .12 \)). Empirical and predicted variances in fecundity rates are not significantly different (F-test for homogeneity of variances: \( P > .05 \)), although the extreme values are farther from the mean in the empirical data set (fig. 4A).

Discussion

We follow a largely mechanistic framework to construct a physiologically structured population model for the common lizard *Zootoca vivipara*. This model allows us to evaluate the demographic consequences of considering variation in birth date and variation in food availability as mechanisms responsible for generating within-cohort individual differences. Parameterized with realistic values and ranges for the stochastic processes, our model predicts differences in life history among individuals that are generally smaller than the observed levels of within-cohort variability. This result is not surprising, because mechanisms other than the ones considered here can generate individual variation in natural populations (Tuljapurkar et al. 2009). For example, heritable traits, maternal effects, and climate conditions are potentially important sources of variation among common lizards (Massot et al. 2002; Marquis et al. 2008; Le Galliard et al. 2010). Yet our results indicate that variation in food availability and birth date can generate individual differences in life history and influence population dynamics.

Population-Dynamic Consequences of Within-Cohort Variability

The median population abundance depends on the model version, but we find no qualitative effects on model dynamics of incorporating within-cohort individual variation. Qualitative effects have been reported by previous studies, although the direction of the effects seems to vary. For example, Fox (2005) introduced heritable individual variation and concluded that increased individual variation reduces the risk of extinction by reducing population-level variance. However, other authors have reported more complex, nonmonotonic patterns (Grimm and Uchmanski 2002; Vindenes et al. 2008) and have shown that there is no unique, simple effect of intraspecific variability on population dynamics, as reviewed in the “Introduction.”
results show that qualitative changes may not always occur in response to individual variation (see also Vindenes et al. 2008). Instead, population-dynamic effects depend on diverse factors, including the intrinsic population dynamics, the source of individual variation, and how this source is incorporated into the model. The fact that population-dynamic effects of individual variation are context and approach dependent highlights the importance of using a mechanistic framework. Although models will always include a degree of structural uncertainty, as our knowledge of dynamics and mechanisms is never complete, a mechanistic framework can provide greater insights because the processes responsible for generating observed patterns are explicitly investigated. Mechanistic models are key to understanding the importance of the diverse sources of intraspecific variability and to gaining insight into how particular factors and mechanisms translate into life-history and population-dynamic changes. An example is our prediction of long, skinny individuals in response to variability in food intake rate (see below).

**Life-History Consequences of Food Variability**

Although our stochastic models predict population dynamics qualitatively similar to those of the deterministic model, we report important consequences of introducing individual variation for the predicted life histories and population densities. In particular, stochastic variation in the food intake rate results in long and skinny individuals, even though the mean food intake remains constant. Skinny individuals are also less fecund, and thus the number of offspring per female decreases, which reduces competition among the newborn class and leads to higher survival of young individuals. The predicted change in individual morphology can be explained by Jensen’s inequality and the nonlinear relationship between body growth and daily food intake (fig. 5). Jensen’s inequality states that for a set of values \( x_i \) with mean \( E(x_i) \), the average result of the nonlinear function \( f(x_i) \) (denoted \( E(f(x_i)) \)) need not equal the function of the average \( f(E(x_i)) \) (Ruel and Ayres 1999). Here, the nonlinearity in body growth is caused by the transition that occurs when the assimilated energy is not sufficient to cover metabolic costs. This transition leads to a concave-up relationship between structural-mass growth and resource availability, because growth is halted when energy intake is below maintenance costs (fig. 5). As a result, when food intake varies daily, the structural-mass mean growth rate is higher than the growth rate predicted for the mean food intake. Conversely, reserves-mass growth has a concave-down relationship because reserves are converted back to energy used to cover maintenance costs when food intake is insufficient. As a result, the mean growth rate of reserves mass in a stochastic environment is lower.

Transitions in growth are expected whenever individuals are able to survive for some time by using energy reserves and body growth is reduced or stopped at the time when food intake is not sufficient to cover maintenance costs. These simple requirements are met by a wide variety of taxa (Kooijman 2000); thus, the nonlinear relationship

---

**Figure 4**: Empirical (Emp) and predicted estimates of female fecundity (A) and growth curves (B). Predictions are obtained from the models Det, Dis, Food, Birth, and F&B (see fig. 3 for definitions of the models). In A, circles represent mean fecundity (number of female offspring per adult female), longer error bars are for the SD among years, and shorter error bars are for the SD among 10 stochastic replicates. Triangles and stars represent extreme values, estimated as the mean of yearly maxima and minima. In B, circles represent mean snout-vent length (SVL) at fixed ages (0, 200, 400, 600, and 800 days), error bars are for the SD among years, and stars represent maximum SVL estimates as the means of yearly maxima.
between body growth and food availability should be very widespread. However, morphological changes may not be apparent if food availability always remains above or below the transition point. Changes in morphology will become apparent only when food intake falls below maintenance costs for some individuals at some point in time. This is likely to occur in food-regulated populations when population size is near carrying capacity or in habitats with high intrinsic stochasticity in food availability. Confirming our predictions, laboratory studies have shown that changing the temporal variance in food availability while keeping the mean constant results in morphological changes in sticklebacks and sea urchin larvae (Ali and Wootton 1999; Miner and Vonesh 2004). Whether the observed morphological changes have demographic consequences in natural populations remains to be clarified. However, our results suggest that population sizes may change, thereby affecting overall resource levels.

**Life-History Consequences of Birth Date Variability**

Variation in birth date has some permanent effects on individual body size (with earlier-born individuals staying larger), but even large variation in birth date has few consequences on the predicted life histories and population dynamics. This suggests that birth date variation may be relatively unimportant in generating demographic differences in *Z. vivipara*, in contrast with empirical results from other lizards (Olsson and Shine 1997; Warner and Shine 2007). On the other hand, one interesting effect of assuming an extended birth period is the attenuated pressure on the resource by the newborns, such that resource levels do not drop sharply, as compared to the case of simultaneous reproduction. Previous PSPMs considering a single reproduction event have shown how newborn cohorts can quickly overexploit resources and starve older cohorts, generating single-cohort cycles (Persson et al. 1998; Claessen et al. 2000). Our model suggests that resources are not as quickly reduced by the newborns when reproduction is extended over a period of time, which may facilitate the coexistence of multiple cohorts. To test this idea, it would be interesting to explore whether including realistic variation in birth date is sufficient to eliminate the single-cohort cycles predicted by some PSPMs (e.g., Persson et al. 1998). The study by van Kooten et al. (2007) hints at this mechanism. Although reproduction in their model remains pulsed on a single day, the within-cohort spreading of life histories over groups and over a spatial gradient indeed reduces the strength of the birth pulse. This also results in dynamics other than cohort cycles that are instead characterized as stochastic fluctuations with relatively small amplitude.

**Implications and Perspectives**

Models that link individual physiological processes, environmental conditions, and population dynamics are use-
ful tools that have become increasingly popular (Kearney and Porter 2009). This study shows that these models may be used to explore the relative importance of diverse factors capable of generating individual variation but that no single, unique response of population dynamics to all sources of individual variation can be expected. This is an important message, and it suggests that the goal of modeling individual variation should shift from general, non-mechanistic approaches to models that allow exploration of how individual differences are generated and the effects of particular sources of variation. In agreement with Vindenes et al. (2008), a first important finding is that qualitative changes in population dynamics may not always occur in response to increased variation among individuals. Although unspectacular, this result is also reassuring and lends support to the deterministic approach to modeling populations. A second important finding is the fact that individual variation may lead to morphological changes, which in turn can affect population processes. In particular, we find that stochastic variation in food availability can affect individual morphology because of a general, widespread nonlinearity in the response of growth to resource availability. Interestingly, whether a morphological response would be observed depends on environmental and population conditions, illustrating the intertwined relationship between individual processes and population dynamics and once again highlighting the importance of using models that account for this feedback in a mechanistic framework. Future work based on this general mechanistic approach is necessary to better understand the consequences of other potentially important sources of variation, such as heritability, maternal effects, and climate conditions.

Acknowledgments

We are indebted to A. de Roos for insightful suggestions and enlightening discussion. Comments from four anonymous reviewers helped improve this manuscript. M. Masot and M. Mugabo generously provided access to unpublished data. This study was funded by the Région Île-de-France R2DS program (grant 2007-06); the Agence Nationale de la Recherche (ANR grant 07-JCJC-0120); the Centre National de la Recherche Scientifique (CNRS); the European Community’s Seventh Framework Programme (FP7/2007–2013), under grant agreement 235897; and the Spanish Ministry of Science and Innovation (Project CGL2009-07301/BOS).

Literature Cited


Disentangling the effects of predator body size and prey density on prey consumption in a lizard

Manuela González-Suárez*†,1, Marianne Mugabo1, Beatriz Decencière2, Samuel Perret2, David Claessen1,3 and Jean-François Le Galliard1,2

1CNRS/UPMC/ENS 7625, Écologie & Évolution, Université Pierre et Marie Curie, 7 Quai St Bernard, 75005 Paris, France; 2CNRS/UPMC/ENS 3194, CEREEP–Ecotron IleDeFrance, École Normale Supérieure, 78 rue du Château, 77140 St-Pierre-lès-Nemours, France; and 3CERES-ERTI, École Normale Supérieure, 24 Rue Lhomond, 75005 Paris, France

Summary

1. Understanding proximate determinants of predation rates is a central question in ecology. Studies often use functional response (density dependent) or allometric (mass dependent) models but approaches that consider multiple factors are critical to capture the complexity in predator–prey interactions. We present a novel comprehensive approach to understand predation rates based on field data obtained from a vertebrate predator.

2. Estimates of food consumption and prey abundance were obtained from 21 semi-natural populations of the lizard Zootoca vivipara. We identified the most parsimonious feeding rate function exploring allometric, simple functional response and allometric functional response models. Each group included effects of sex and weather conditions.

3. Allometric models reveal the importance of predator mass and sex: larger females have the highest natural feeding rates. Functional response models show that the effect of prey density is best represented by a Holling type II response model with a mass, sex and weather dependent attack rate and a constant handling time. However, the best functional response model only received moderate support compared to simpler allometric models based only on predator mass and sex.

4. Despite this limited effect of prey densities on feeding rates, we detected a significant negative relationship between an index of preferred prey biomass and lizard density.

5. Functional response models that ignore individual variation are likely to misrepresent trophic interactions. However, simpler models based on individual traits may be best supported by some data than complex allometric functional responses. These results illustrate the importance of considering individual, population and environmental effects while also exploring simple models.

Key-words: field experiment, functional response, Lacertidae, parsimony, prey–predator interaction, size-effect

Introduction

The study of prey–predator interactions and foraging behaviour can provide critical insights into the structure of food webs, population dynamics and species interactions (e.g. Persson et al. 1998; Abrams 2000; de Roos, Persson & Mccauley 2003; Gilg, Hanski & Sittler 2003; Miller et al. 2006). The foraging ability of a predator determines its energy acquisition and ability to grow, survive and reproduce, but can also influence the fitness of conspecifics as predators may aid or interfere with each other (Arditi & Akcakaya 1990). Predation also has a large effect on prey populations influencing their dynamics, behaviour and spatial distribution (Reeve 1997; Gilg, Hanski & Sittler 2003). Predation rates are determined by several factors including prey and predator densities, body size, habitat structure and weather conditions (e.g. Avery 1971; Angilletta 2001; Pitt & Ritchie 2002; Miller et al. 2006). The field of foraging ecology has emphasized density-dependent effects defining functional responses that estimate prey consumption by an average predator as a function of prey, and in some cases

*Correspondence author. E-mail: manuela.gonzalez@ebd.csic.es
†Present address. Department of Conservation Biology, Estación Biológica de Doñana-CSIC, Calle Américo Vespucio s/n, 41092 Sevilla, Spain.

© 2010 The Authors. Functional Ecology © 2010 British Ecological Society
Density- and size-dependent predation

Fig. 1. Adult male of the species Zootoca vivipara (European common lizard) at an outdoor enclosure in the CEREEP, France (48°17′N, 2°41′E). Photo credit: M. González-Suárez.

gate the effect of varying lizard densities and thus, predation pressure, on the invertebrate community. The importance of our study hinges on exploring a novel comprehensive approach to define prey–predator interactions using field data from a complex vertebrate predator. Our results highlight the importance of exploring diverse approaches using field data to advance our understanding of foraging dynamics.

Materials and methods

LIZARD POPULATIONS: ENCLOSURES

During the summer of 2008, we established five experimental density treatments of Z. vivipara in 24 outdoor enclosures at the Centre de Recherche en Ecologie Expérimentale et Prédicitive (CEREEP, 48°17′N, 2°41′E). Initial densities were equivalent to 700–3500 adults + yearlings per ha and sex ratios were close to 1 : 1. Surviving lizards (n = 326) from all treatments were recaptured in May–June 2009 (93% of recaptures occurred on 4 days in May) and a final population density (P) per enclosure was calculated. After capture, we estimated body mass for each lizard and classified female reproductive status as pregnant or non-pregnant. We also obtained an estimate of sunshine duration (I, in h day⁻¹) from a Campbell Scientific (Courtabœuf, France) CSD3 solar radiation sensor located within 300 m of the enclosures. Sunshine duration is defined as total time with direct solar radiation exceeding 120 W m⁻² (W.M.O. 2008) and was calculated over 10-min intervals. Although both air temperature and solar radiation influence activity in reptiles, based on previous studies we expected sunshine duration would influence activity (and hence, feeding) more directly than temperature in this species (Avery 1971; House, Taylor & Spellerberg 1980). Additional details of the enclosures, the density manipulation and lizard captures are provided in Appendix S1 (Supporting Information).

PREY POPULATIONS

Invertebrate abundance was estimated by a combination of pitfall trapping and sweep-net capture techniques (Brennan, Majer & Moir
2005) after all lizards had been captured to avoid injuries to the lizards, that is, drowning in a pit trap. Invertebrates were classified into preferred prey biomass index (\(N_p\)), which includes the orders Araneae, Homoptera, Heteroptera and Orthoptera (Avery 1966), and a spider biomass index (\(N_s\), Araneae). Spiders were considered independently because they are the principal food of *Z. vivipara* (Avery 1966; Le Galliard, Ferrière & Clobert 2005a) and also were the most common invertebrate group in the enclosures (Fig. S1, Supporting Information). Additional details of the invertebrate captures are provided in Appendix S1 (Supporting Information).

**ESTIMATION OF FEEDING RATES**

We inferred natural feeding rates from measured faecal production using a standard relationship between faecal production and food consumption. This relationship was defined using data from a laboratory feeding experiment (see Appendix S1, Supporting Information). We measured faecal production for 107 lizards captured in the outdoor enclosures. After capture these lizards were housed in individual terraria and kept without food for 3 days. All faecal pellets produced since capture were dried and weighed. Using the experimental relationship between faecal production and food consumption we estimated natural feeding rates (in mg of live prey per day). Experimental procedures are described in more detail in Appendix S1 (Supporting Information).

**DATA ANALYSIS: FEEDING RATES**

Using the laboratory data, we explored several models aiming to predict faecal production from food intake (\(E\)). Some models included effects of \(S\) (sex as male or female) and/or \(M\) (lizard mass in g). The best fitting model was selected using an information-theoretic approach described below. The selected model was used to infer natural feeding rates from the measured faecal output of the animals captured in the outdoor enclosures.

We investigated the effects of predator body mass (\(M\)) and sex (\(S\)), predator (\(P\)) and prey (\(N\)) densities and sunshine duration (\(I\)) on the estimated natural feeding rates, applying allometric functions and functional responses. The simplest allometric function considered was \(f(M) = AM^B\), in which feeding rates only depend on \(M\). The allometric coefficient \(A\) and exponent \(B\) are estimated from the data. Alternative allometric functions were derived from this function by allowing the \(A\) and/or \(B\) parameter to vary between males and females, or introducing a linear effect of sunshine duration [e.g. \(f(M, I) = AIM^B\)].

In addition, we formulated a group of functional response models considering Holling type I and II response models (Holling 1959) and a ratio dependent model (Hassell & Varley 1969). The type I response model assumes a linear increase in predation with increasing prey density (\(N\)), \(f(N) = bN\), where \(b\) is the attack rate. The type II response model assumes an asymptotic relationship of feeding rate with prey density, \(f(N) = \frac{bN}{1 + bN/H}\), where \(b\) is the attack rate and \(T_h\) the handling time. The Hassell–Varley flexible ratio function is a modified Holling type II model in which predation rate is influenced by prey (\(N\)) and predator density (\(P\)), \(f(N, P) = \frac{b(N/P)}{1 + b(N/P)/(P/S)}\), where \(b\) is the attack rate, \(T_h\) the handling time and \(mn\) an exponent that determines the strength of the predator density effect. In all models, prey densities (\(N\)) were defined as either preferred prey biomass (\(N_p\)) or spider biomass (\(N_s\)).

These three basic functional response equations were modified to include more complex attack rates that incorporate effects of predator body mass and sex, as well as sunshine duration. Attack rates are expected to increase with predator size as larger lizards have greater sensory acuity and locomotor ability (Garland 1984). In *Z. vivipara* prey size has been observed to increase with lizard size (Avery 1966) and we used an allometric attack rate function \(b(M) = b_1M^{b_2}\) where \(b_1\) is an allometric coefficient and \(b_2\) an allometric exponent. In some models we also included an effect of predator sex [e.g. \(b(M, S) = b_1(S) \cdot M^{b_2(S)}\)] and sunshine duration [e.g. \(b(I, M) = b_1 I \cdot M^{b_2}\)]. Although handling time (\(T_h\)) may vary with body size (Persson et al. 1998), a recent study found these rates were relatively constant except for the smallest predators (Aljletlawi, Sparrevik & Leonardsson 2004). We therefore assumed that handling time is constant. In total, we explored 54 functional response models.

All models were fitted using the non-linear procedure NLS in R 2.10.0 (R Development Core Team 2009). The best fitting model(s) was selected using an information-theoretic approach (Burnham & Anderson 2002) considering Akaike’s information criterion corrected for small sample sizes (AICc), model support as the difference in AICc between each model and the model with the lowest AICc (\(\Delta\)), and weight (\(w_0\)). Total \(w_0\) was calculated as the cumulative weight of all models including a particular variable or type of functional response (e.g. Holling type I response), which is similar to the variable weight \(w_0\) proposed by Burnham & Anderson (2002). All models with \(\Delta < 2\) were considered to be supported. We also estimated the percentage deviance explained as (model deviance-null model deviance/null model deviance)*100, where the null model is an intercept only model.

**DATA ANALYSIS: IMPACT ON INVERTEBRATE COMMUNITIES**

First, we explored the relationship between invertebrate biomass index (total, \(N_p\) or \(N_s\)) and predator density (\(P\)) using linear regression. Secondly, we defined an invertebrate community similarity matrix among enclosures based on pairwise Bray–Curtis indices. Bray–Curtis indices were calculated as the absolute difference in invertebrate order biomass between two enclosures, summed over all orders and divided by the total biomass in all enclosures and orders (Bray & Curtis 1957). To detect changes in community structure (biomass and composition) due to lizard density we used a test analogous to a multivariate analysis of variance called ADONIS (Oksanen et al. 2009). ADONIS returns a statistic \(R\), which is a measure of separation among groups (0 indicates complete mixing and 1 represents full separation), and a \(p\)-value estimated by repeated permutations of the data. We used the ADONIS procedures in the VEGAN package in R 2.10.0 (R Development Core Team 2009) with 999 permutations.

Although enclosures had overall similar habitat and environmental conditions, we expected a gradient of soil humidity due to differences in proximity to a nearby creek. Therefore, we introduced creek proximity, a proxy for humidity, as an additional regression variable to explain invertebrate biomass and as a block in the ADONIS procedure. Enclosures were distributed in five rows running more or less parallel to the creek, thus creek proximity was ranked from 1 to 5 with 1 assigned to the row of enclosures closest to the creek (\(-30\) m) and 5 to those furthest (\(-90\) m).
Results

EXPERIMENTAL RELATIONSHIP BETWEEN FEEDING RATE AND FAECAL PRODUCTION

The best model to explain faecal production includes only food intake ($E$). However, there were two additional models supported by the data (Table 1) that include mass and sex. At the time of this experiment, six females were in an advanced stage of pregnancy and ate considerably less than expected based on their body mass because their abdominal cavity was largely occupied by developing eggs. We repeated our analysis excluding these individuals and found that a single model, including only $E$, was supported (Table 1). Therefore, we used the function $\text{faecal output} = bE$ ($b = 0.072$, SE = 0.006, $p < 0.0001$, deviance explained = 29.7%), where $E$ is in mg of live prey day$^{-1}$ and faecal output is a daily mean estimated over 3 days (dry mg day$^{-1}$).

NATURAL FEEDING RATES

All individuals captured in the field produced faeces, which indicate that all had eaten prior to capture. There was no effect of capture date on food intake (ANOVA $F_{3,105} = 2.15$, $p = 0.10$). Two females had a faecal output much larger than expected based on their body mass (see Fig. 2) and were identified as outliers during the analysis of the data. Reported results do not include these outliers because parameter estimates were different (particularly the allometric exponent) even though selected models were similar in both data sets. In the allometric function group, two models were supported while only three had $w_i > 10\%$ (Table 2). Both supported models include the same predictors: sex ($S$) and body mass ($M$); however, they differ in how the sex effect was introduced (either modifying the allometric coefficient $A$ or the exponent $B$; Table 3). Supported models were nearly identical in their AIC$_c$ value and the resulting curves largely overlapped, predicting the highest feeding rates for larger female lizards (Table 3 and Fig. 2, deviance explained = 31.4%). Mean food intake was 182.68 ± 15.605 mg day$^{-1}$ for males and 251.51 ± 15.135 mg day$^{-1}$ for females. We found no differences between natural feeding rates of pregnant and non-pregnant females controlling for body mass (residuals of a mass model, Student $t = 0.75$, d.f. = 56, $p = 0.457$). Models including an effect of sunshine duration ($I$) received low support (total $w_i = 0.07$).

In the functional response group, two models were supported and only three models had $w_i > 10\%$ (Table 2). The top model was based on a Holling type II function for preferred prey biomass ($N_p$) with a $M$-, $S$- and $I$-dependent attack rate (Table 3). The second supported model also included a $M$-, $S$- and $I$-dependent attack rate but was based on a Hassell–Varley function (Table 3). However, the exponent $m$, which describes the effect of predator density, was not significantly different from zero (Table 3). It is important to note that we did not have cross-treatments in which both prey and predator densities were controlled and varied. As a result prey and predator densities were correlated.

Table 1. Selection results of models exploring faecal output in Zootoca vivipara kept in the laboratory for the complete data set ($n = 41$) and for a data set excluding pregnant females ($n = 35$).

<table>
<thead>
<tr>
<th>Fecal output model*</th>
<th>$k$</th>
<th>AIC$_c$</th>
<th>$\Delta$</th>
<th>$w_i$</th>
<th>% deviance explained</th>
</tr>
</thead>
<tbody>
<tr>
<td>All data</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\beta E$</td>
<td>2</td>
<td>213,469</td>
<td>0.000</td>
<td>0.4741</td>
<td>29.705</td>
</tr>
<tr>
<td>$\beta M^E$</td>
<td>3</td>
<td>214,788</td>
<td>1.318</td>
<td>0.2453</td>
<td>31.423</td>
</tr>
<tr>
<td>$\beta S^E$</td>
<td>3</td>
<td>215,347</td>
<td>1.877</td>
<td>0.1854</td>
<td>30.482</td>
</tr>
<tr>
<td>$\beta (S) M^E$</td>
<td>4</td>
<td>216,681</td>
<td>3.212</td>
<td>0.0952</td>
<td>32.369</td>
</tr>
<tr>
<td>Excluding pregnant females</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\beta E$</td>
<td>2</td>
<td>182,276</td>
<td>0.000</td>
<td>0.5505</td>
<td>26.805</td>
</tr>
<tr>
<td>$\beta S^E$</td>
<td>3</td>
<td>184,488</td>
<td>2.212</td>
<td>0.1822</td>
<td>27.999</td>
</tr>
<tr>
<td>$\beta M^E$</td>
<td>3</td>
<td>184,293</td>
<td>2.017</td>
<td>0.2008</td>
<td>27.195</td>
</tr>
<tr>
<td>$\beta (S) M^E$</td>
<td>4</td>
<td>186,504</td>
<td>4.228</td>
<td>0.0665</td>
<td>28.317</td>
</tr>
</tbody>
</table>

We report number of parameters ($k$), Akaike’s information criteria (AIC$_c$), model support ($\Delta$), model weights ($w_i$) and percentage deviance explained. Supported models are in bold.

*In the models: $E =$ experimental food intake, $M =$ lizard body mass, $S =$ lizard sex. $\beta$ is the allometric coefficient and $R$ is the allometric exponent.
Table 2. Selection results of simple allometric functions and functional responses describing feeding rates under semi-natural conditions in Zootoca vivipara

<table>
<thead>
<tr>
<th>Feeding rates model*</th>
<th>k</th>
<th>AICc</th>
<th>Δ</th>
<th>wi</th>
<th>% deviance explained</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Simple allometric functions</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( A(Np) )</td>
<td>4</td>
<td>1261.258</td>
<td>0.000</td>
<td>0.371</td>
<td>31.382</td>
</tr>
<tr>
<td>( A(Ns) )</td>
<td>5</td>
<td>1260.286</td>
<td>1.027</td>
<td>0.366</td>
<td>31.400</td>
</tr>
<tr>
<td>( A(Np) )</td>
<td>5</td>
<td>1265.38</td>
<td>2.130</td>
<td>0.128</td>
<td>31.450</td>
</tr>
<tr>
<td>( A(Ns) )</td>
<td>3</td>
<td>1267.814</td>
<td>3.556</td>
<td>0.063</td>
<td>27.560</td>
</tr>
<tr>
<td>( A(Np) )</td>
<td>4</td>
<td>1269.132</td>
<td>4.874</td>
<td>0.032</td>
<td>28.141</td>
</tr>
<tr>
<td><strong>Functional responses</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( b )</td>
<td>6</td>
<td>1267.204</td>
<td>0.000</td>
<td>0.325</td>
<td>32.380</td>
</tr>
<tr>
<td>( s )</td>
<td>6</td>
<td>1268.749</td>
<td>1.545</td>
<td>0.150</td>
<td>31.378</td>
</tr>
<tr>
<td>( i )</td>
<td>7</td>
<td>1269.354</td>
<td>2.150</td>
<td>0.111</td>
<td>32.475</td>
</tr>
<tr>
<td>( m )</td>
<td>5</td>
<td>1269.581</td>
<td>2.376</td>
<td>0.099</td>
<td>29.333</td>
</tr>
<tr>
<td>( w )</td>
<td>5</td>
<td>1270.141</td>
<td>2.936</td>
<td>0.075</td>
<td>28.956</td>
</tr>
<tr>
<td>( p )</td>
<td>6</td>
<td>1270.812</td>
<td>3.608</td>
<td>0.053</td>
<td>30.016</td>
</tr>
<tr>
<td>( q )</td>
<td>5</td>
<td>1271.698</td>
<td>4.493</td>
<td>0.034</td>
<td>27.894</td>
</tr>
</tbody>
</table>

We present the top models with the number of parameters (k), Akaike’s information criteria (AICc), model support (Δ), model weights (wi) and percentage deviance explained. Supported models are in bold.

*\( I \) = sunshine duration, \( N_p \) = preferred prey biomass, \( N_s \) = spider biomass, \( P \) = lizard abundance, \( M \) = lizard body mass, \( S \) = lizard sex, \( A \), \( B \), \( b \) and \( s \) are allometric coefficients and exponents. The exponent \( m \) describes the strength of a predator density effect.

Table 3. Parameter estimates for the best models defining natural feeding rates in Zootoca vivipara considering simple allometric functions and functional responses

<table>
<thead>
<tr>
<th>Feeding rates model*</th>
<th>Parameter ± SE(^{1})</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Simple allometric functions</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( A(Np) )</td>
<td>( A = 100.47 ± 18.050 )</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>( B )</td>
<td>( B = 0.070 ± 0.119 )</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>( b_0 )</td>
<td>( b_0 = 0.05 ± 0.146 )</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>( A )</td>
<td>( A = 107.85 ± 20.580 )</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>( B )</td>
<td>( B = 0.063 ± 0.124 )</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td><strong>Functional responses</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( b )</td>
<td>( b = 0.003 ± 0.0004 )</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>( b_0 )</td>
<td>( b_0 = 0.017 ± 0.100 )</td>
<td>0.098</td>
</tr>
<tr>
<td>( b_1 )</td>
<td>( b_1 = 3.58 ± 1.51 )</td>
<td>0.020</td>
</tr>
<tr>
<td>( b_2 )</td>
<td>( b_2 = 2.44 ± 0.825 )</td>
<td>0.004</td>
</tr>
<tr>
<td>( m )</td>
<td>( m = 0.63 ± 0.290 )</td>
<td>0.009</td>
</tr>
</tbody>
</table>

See Table 2 for model selection.

\( b(Np) \) = sunshine duration, \( N_p \) = preferred prey biomass, \( N_s \) = spider biomass, \( P \) = lizard abundance, \( M \) = lizard body mass, \( S \) = lizard sex.

\(^{1}\)Subscripts indicate fem: parameter value for females, male: parameter value for males.

\( (P-N_p) \) Pearson r = -0.69, \( P-N_s \) r = -0.56 and parameters in the Hassell–Varley models may not have been estimated adequately.

Overall, models based on a Holling type II response had more support (total \( wi = 0.65 \)) than those based on the Hassell–Varley function (total \( wi = 0.34 \), while Holling type I response models received no support (total \( wi < 0.0001 \)). Models based on \( N_p \) had higher weights (total \( wi = 0.89 \)) than models based on spider biomass only (\( N_s \); total \( wi = 0.11 \)). Models including \( M \)- and \( S \)-dependent attack rates were strongly supported (total \( wi > 0.99 \) and total \( wi = 0.98 \), respectively). In contrast to the results from the allometric group, functional response models including an effect of \( I \) were supported (total \( wi = 0.79 \)). Density-dependent models not including individual traits \((M \text{ and } S)\) received no support (\( \Delta > 34.34, wi < 0.0001 \)).

Because both groups of models used the same data base, we could also compare them to determine the most parsimonious model overall. The top two allometric functions including \( S \) and \( M \) (Tables 2 and 3) were identified as the only supported models when combining both groups of functions. The best functional response model had \( \Delta = 4.5 \) (Table 2), suggesting limited support for a density effect on natural feeding rates.

IMPACTS OF LIZARD DENSITY ON INVERTEBRATE POPULATIONS

We captured and identified invertebrates from 17 different orders. The most abundant group was Araneae (Fig. S1, Supporting Information), while Ephemeroptera, Neuroptera and Odonata were rare (found in less than six enclosures and representing <0.5% of the total biomass). There was no
relationship between the total invertebrate biomass index and lizard density or creek proximity ($F_{5,15} = 2.00, p = 0.14$; adjusted $R^2 = 0.20$; Fig. 3a). However, we found a significant negative effect of lizard density on biomass of preferred lizard prey $N_p$ ($\beta = -3.29$, SE = 1.305; Fig. 3b) with no effect of creek proximity (overall regression $F_{5,15} = 4.84$, $p = 0.008$, adjusted $R^2 = 0.49$). There was also an effect of lizard density on spider biomass $N_s$ ($\beta = -1.69$, SE = 1.393; Fig. 3b) with no effect of creek proximity (overall regression $F_{5,15} = 4.49$, $p = 0.011$, adjusted $R^2 = 0.47$). Despite these differences for particular invertebrate groups, we did not detect differences in invertebrate community structure with lizard density, although there was a weak but significant effect of creek proximity (density: $R = 0.05$, $p = 0.38$; creek proximity: $R = 0.14$, $p = 0.05$). Results did not change if the rare orders: Ephemeroptera, Neuroptera and Odonata, were excluded.

**Discussion**

**INTRINSIC FACTORS INFLUENCING FEEDING RATES**

Both lizard body mass and sex had important effects on individual feeding rates. Larger predators consume more prey than smaller individuals as previously reported for *Z. vivipara* (Avery 1971; Pilorge 1982) and many other species (Tripet & Perrin 1994; Aljetlawi, Sparrevik & Leonardsson 2004; Vuic-Pestic et al. 2010). A new finding is that females consume greater amounts of food than males, even after controlling for differences in body mass. Previous studies of *Z. vivipara* failed to detect (Avery 1971) or to consider (Van Damme, Bauwens & Verheyen 1991) these sex differences. However, our results suggest they may be important.

There are several possible non-exclusive explanations for these sex differences. Females could have increased feeding rates to compensate for their recent investment in reproduction (Avery 1974). However, we found that pregnant and non-pregnant females had similar feeding rates. Alternatively, males may spend less time foraging because they engage in reproductive behaviours during this time of the year. On the other hand, females and males may have different diets due to distinct energy requirements and investment strategies. For example, juvenile female *Z. vivipara* invest more in body length growth, while males invest in body condition (mass adjusted for length, Le Galliard, Ferrière & Clobert 2005b). Whether these differences in energy investments influence feeding rates remains to be determined.

**EXTRINSIC FACTORS INFLUENCING FEEDING RATES AND FUNCTIONAL RESPONSE FUNCTIONS**

Feeding rates generally increase with sunshine duration (see also Avery 1971; House, Taylor & Spellerberg 1980). However, the effect of sunshine was only noticeable within the functional response model group, suggesting attack rates (search and detection of prey) increase in sunnier days. Future experimental studies may be necessary to clarify the role of weather conditions in *Z. vivipara* feeding rates. Similarly, evidence of prey density effects in feeding rates was limited. Prey densities in semi-natural conditions cannot be easily manipulated. Instead prey biomass was expected to vary due to the experimental manipulation of lizard density. Although variability in prey was observed, there was only a weak effect of prey density in feeding rates. It is possible that prey densities remained sufficiently high to prevent strong food limitation even at the highest lizard densities.

Which functional response models better represent natural dynamics is a debated issue that this study directly addresses (Abrams & Ginzburg 2000; Schenk, Bersier & Bacher 2005). We found best support for a Holling type II functional response in *Z. vivipara* suggesting feeding rates reach a limit at high prey densities. However, unlike previous studies (Schenk, Bersier & Bacher 2005; Miller et al. 2006), we only found limited support for an effect of predator density. The common lizard is a non-territorial species with overlapping home ranges and conspecific interference during predation may be rare (Massot et al. 1992). Alternatively, the correlation between prey and predator densities may have limited our ability to correctly fit models including both density effects.

**MOS T PARSIMONIOUS FEEDING RATE FUNCTIONS**

A primary goal of this study was to identify the most parsimonious feeding rate function following a comprehensive approach that includes both allometric and functional responses. Many studies consider only one group of models and thus create a dichotomy that does not exist in reality.
Our results show that both prey density and body mass influence feeding rates, but that individual traits, mass and sex, are much better predictors. Recent studies have shown the importance of considering both density and body mass effects and have suggested the use of allometric functional response models in which attack rates and/or handling times are size dependent (Aljetlawi, Sparrevik & Leonardsson 2004; Vucic-Pestic et al. 2010). Allometric functional responses provide an important tool to represent individual and population level effects (Brose 2010). However, for empirical studies and generalist predators, simpler allometric functions may provide a more parsimonious description of the data than complex allometric functional response models.

Even though at some level prey density must affect feeding rates and functional response models are an important contribution to the field of ecology (Abrams 2000), variation in prey consumption may be better explained by individual differences than by natural variation in prey density. In light of these results, we urge researchers to explore diverse types of function, including allometric functional responses and simpler functions, while considering the effects of multiple individual traits such as mass and sex, prey and predator population densities, and whenever possible environmental variables.

Despite a weak effect of prey density on consumption rates, our study documents a negative effect of lizard density on preferred prey and in particular on spider biomass, presumably as a consequence of feeding pressure. A negative effect of lizard density on spider abundance has been previously reported (Spiller & Schoener 1998; Le Galliard, Ferrière & Clobert 2005a). This effect may be potentially important for community diversity and structure because spiders are themselves predators that can influence other invertebrates and habitat conditions (Greenstone 1999; Schmitz 2005a). Therefore, lizards could directly, via predation, and indirectly, via their effect on spiders, influence the composition and biomass of the invertebrate communities (Spiller & Schoener 1998). Although in our study we did not find a significant effect of lizard density on invertebrate community structure, which instead appears to respond to creek proximity (a proxy of humidity), effects may occur at different densities of lizards and/or spiders.

One caveat in our study is that prey and lizard densities could not be estimated at the same time. This time-lag may have limited our ability to identify existing effects of prey density on lizard feeding rates or of lizard density on prey communities. Regrettably, we could not estimate prey biomass until after all lizards were captured to avoid injuring lizards inside insect traps and thus, we could not estimate prey and lizard densities simultaneously. These difficulties are drawbacks of field studies. Nevertheless, we detected an expected effect of lizard density on prey abundance, suggesting measured prey densities did reflect, at least to some extent, enclosure conditions prior to lizard removal.

Finally, the observation that prey consumption depends only weakly on prey density is both interesting and puzzling. This result poses the question of how common lizard populations and their prey are regulated (Massot et al. 1992; Abrams 2000). If populations are regulated through prey depletion as typically assumed in population dynamic models, prey consumption should decrease with predator density due to a decrease in prey abundance. We indeed found a negative effect of lizard density on preferred prey and spider biomass. However, the associated reduction in predation rate was less evident. This might imply that other ecological interactions, which remain unexplored, are involved in the regulation of these lizard populations.

Acknowledgements

We would like to thank all personnel at the CEREEP field station for their help. Three anonymous reviewers and the associate editor provide helpful suggestions to improve an earlier version of this manuscript. This study was funded by the Région Île-de-France R2DS program (grant 2007/06), the Agence Nationale de la Recherche (ANR grant 07-SCC-0120), the Centre National de la Recherche Scientifique (CNRS), the Spanish Ministry of Science and Innovation (Project: CGL2009-07301/BOS) and the European Community’s Seventh Framework Programme (FP7/2007-2013) under grant agreement n° 235897. The protocol of this study was approved by the Ile-De-France Regional Ethics Committee for Animal Experimentation N°3 (file p/2008/008).

References


Received 12 March 2010; accepted 12 August 2010
Handling Editor: Duncan Iriscich

**Supporting Information**

Additional supporting information may be found in the online version of this article.

**Appendix S1.** Extended methods.

**Fig. S1.** Total invertebrate biomass per experimental enclosure.

As a service to our authors and readers, this journal provides supporting information supplied by the authors. Such materials may be re-organized for online delivery, but are not copy-edited or typeset. Technical support issues arising from supporting information (other than missing files) should be addressed to the authors.
A.2 Adaptive dynamics (section 3)


8. Aguilee, R., D. Claessen and A. Lambert. (manuscript). Landscape dynamics as a mechanism to explain adaptive radiations.
Ontogenetic niche shifts and evolutionary branching in size-structured populations

David Claessen¹* and Ulf Dieckmann²

¹Institute for Biodiversity and Ecosystem Dynamics, Section of Population Biology, University of Amsterdam, PO Box 94084, 1090 GB Amsterdam, The Netherlands and ²Adaptive Dynamics Network, International Institute for Applied Systems Analysis, A-2361 Laxenburg, Austria

ABSTRACT

There are many examples of size-structured populations where individuals sequentially exploit several niches in the course of their life history. Efficient exploitation of such ontogenetic niches generally requires specific morphological adaptations. Here, we study the evolutionary implications of the combination of an ontogenetic niche shift and environmental feedback. We present a mechanistic, size-structured model in which we assume that predators exploit one niche when they are small and a second niche when they are big. The niche shift is assumed to be irreversible and determined genetically. Environmental feedback arises from the impact that predation has on the density of the prey populations. Our results show that, initially, the environmental feedback drives evolution towards a generalist strategy that exploits both niches equally. Subsequently, it depends on the size-scaling of the foraging rates on the two prey types whether the generalist is a continuously stable strategy or an evolutionary branching point. In the latter case, divergent selection results in a resource dimorphism, with two specialist subpopulations. We formulate the conditions for evolutionary branching in terms of parameters of the size-dependent functional response. We discuss our results in the context of observed resource polymorphisms and adaptive speciation in freshwater fish species.

Keywords: Arctic char, bluegill, cichlids, evolution, feedback, ontogenetic niche shift, perch, population dynamics, resource polymorphism, roach, size structure.

INTRODUCTION

In size-structured populations, it is common for individuals to exploit several niches sequentially in the course of their life history (Werner and Gilliam, 1984). The change during life history from one niche to another is referred to as an ontogenetic niche shift. The shift can be abrupt, such as that associated with metamorphosis in animals like tadpoles and insects, or gradual, such as the switch from planktivory to benthivory in many freshwater fish species (Werner, 1988).

* Address all correspondence to David Claessen, IACR-Rothamsted, Biomathematics Unit, Harpenden, Herts AL5 2JQ, UK. e-mail: david.claessen@bbsrc.ac.uk

Consult the copyright statement on the inside front cover for non-commercial copying policies.
Ontogenetic niche shifts have been interpreted as adaptations to the different energetic requirements and physiological limitations of individuals of different sizes. The profitability of a given prey type generally changes with consumer body size because body functions such as capture rate, handling time, digestion capacity and metabolic rate depend on body size. For example, using optimal foraging theory, both the inclusion of larger prey types in the diet of larger Eurasian perch (*Perca fluviatilis*) individuals, and the ontogenetic switch from the pelagic to the benthic habitat, have been attributed to size-dependent capture rates and handling times (Persson and Greenberg, 1990). Determining the optimal size at which an individual is predicted to shift from one niche to the next, and how the optimum depends on the interactions between competing species, have been at the focus of ecological research during the last two decades (Mittelbach, 1981; Werner and Gilliam, 1984; Persson and Greenberg, 1990; Leonardsson, 1991). Research has concentrated on approaches based on optimization at the individual level, assuming a given state of the environment in terms of food availability and mortality risks. An important result of this research is Gilliam’s µ/g rule, which states that (for juveniles) the optimal strategy is to shift between niches in such a way that the ratio of mortality over individual growth rate is minimized at each size (Werner and Gilliam, 1984).

Individual-level optimization techniques do not take into account population-level consequences of the switch size. In particular, the size at which the niche shift occurs affects the harvesting pressures on the different prey types and hence their equilibrium densities. In an evolutionary context, this ecological feedback between the strategies of individuals and their environment has to be taken into account. On the one hand, the optimal strategy depends on the densities of the resources available in the different niches. On the other hand, these resource densities change with the ontogenetic strategies and resultant harvesting rates of individuals within the consumer population. A framework for the study of evolution in such an ecological context is the theory of adaptive dynamics (Metz et al., 1992, 1996a; Dieckmann and Law, 1996; Dieckmann and Doebeli, 1999; Doebeli and Dieckmann, 2000). In this framework, the course and outcome of evolution are analysed by deriving the fitness of mutants from a model of the ecological interactions between individuals and their environment. An important result from adaptive dynamics theory is that, if fitness is determined by frequency- and/or density-dependent ecological interactions, evolution by small mutational steps can easily give rise to evolutionary branching. However, although most species are size-structured (Werner and Gilliam, 1984; Persson, 1987), the adaptive dynamics of size-structured populations have received little attention so far. Although there have been several studies of adaptive dynamics in age- or stage-structured populations (e.g. Heino et al., 1997; Diekmann et al., 1999), only one of these explicitly accounted for the effects of the environment on individual growth and on population size structure (Ylikarjula et al., 1999). One motivation for the research reported here, therefore, is to determine similarities and differences between evolution in structured and unstructured populations subject to frequency- and density-dependent selection. We can even ask whether population size structure has the potential to drive processes of evolutionary branching that would be absent, and thus overlooked, in models lacking population structure.

In this paper, we examine a simple size-structured population model that includes a single ontogenetic niche shift. The ecological feedback is incorporated by explicitly taking resource dynamics into account. We assume that individuals exploit one prey type when they are small and another prey type when they are big. The ontogenetic niche shift is...
thought to represent a morphological trade-off: if efficient exploitation of either prey type requires specific adaptations, shifting to the second prey type results in a reduced efficiency on the first prey type. The size at which individuals shift from the first to the second niche is assumed to be determined genetically and is the evolutionary trait in our analysis. The shift is assumed to be gradual; we investigate how evolutionary outcomes are influenced by the width of the size interval with a mixed diet.

We focus on two specific questions. First, what is the effect of the ecological feedback loop through the environment on the evolution of the ontogenetic niche shift? The size at which individuals shift to the second niche affects the predation rate on both prey types and hence their abundances. The relation between strategy and prey abundance is likely to be important for the evolution of the ontogenetic niche shift. Second, what is the effect of the scaling with body size of search and handling rates for the two prey types? The profitability of prey types for an individual of a certain size depends on how these vital rates vary with body size. Data exist for several species on how capture rates and handling times depend on body size. Thus, if different evolutionary scenarios can be attributed to differences in these scaling relations, the results reported here may help to compare different species and to assess their evolutionary histories in terms of the ecological conditions they experience.

THE MODEL

As the basis for our analysis, we consider a physiologically structured population model of a continuously reproducing, size-structured population. We assume that the structured population feeds on two dynamic prey populations. Our model extends the Kooijman-Metz model (Kooijman and Metz, 1984; de Roos et al., 1992; de Roos, 1997) in two directions: first, by introducing a second prey population and, second, by the generalization of the allometric functions for search rate and handling time that determine the functional response.

Individuals are characterized by two so-called $i$-state variables (Metz and Diekmann, 1986): their current length, denoted by $x$, and the length around which they switch from the first to the second prey type, denoted by $u$ (Table 1). Individuals are assumed to be born with length $x_0$; subsequently, their length changes continuously over time as a function of food intake and metabolic costs. The switch size $u$ is constant throughout an individual’s life but, in our evolutionary analysis, may change from parent to offspring by mutation. In our analysis of the population dynamic equilibrium, we assume monomorphic populations, in which all individuals have the same trait value $u$. The per capita mortality rate, denoted $\mu$, is assumed to be constant and size-independent. Possible consequences of relaxing this assumption are addressed in the Discussion, under the heading ‘Assumptions revisited’.

Feeding

Individuals start their lives feeding on prey 1 but shift (gradually or stepwise) to prey 2 as they grow. We assume a complementary relation between foraging efficiencies on the two prey types, which is thought to be caused by a genetically determined morphological change during ontogeny. Figure 1 shows two sigmoidal curves as a simple model of such an ontogenetic niche shift. Immediately after birth, individuals have essentially full efficiency on prey 1 but are very inefficient on prey 2. At the switch size $x = u$, individuals have equal
efficiency on both prey types. Larger individuals become increasingly more specialized on prey type 2.

The ontogenetic niche shift is incorporated into the model by assuming that the attack rate on each prey type is the product of an allometric term that increases with body length, and a ‘shift’ term that is sigmoidal in body length and that depends on the switch size $u$. Using a logistic sigmoidal function for the shift term (Fig. 1), the two attack rate functions become:

$$A_1(x, u) = a_1 x^{q_1} \frac{1}{1 + e^{k(x - u)}}$$

(1)

$$A_2(x, u) = a_2 x^{q_2} \left(1 - \frac{1}{1 + e^{k(x - u)}}\right)$$

(2)

where $a_1$ and $a_2$ are allometric constants and $q_1$ and $q_2$ are allometric exponents. The parameter $k$ tunes the abruptness of the switch; $k = \infty$ corresponds to a discrete step from niche 1 to niche 2 at size $x = u$, whereas a small value of $k$ (e.g. $k = 20$) describes a more gradual shift. In the latter case, there is a considerable size interval over which individuals have a mixed diet.

### Table 1. Symbols used in model definition for state variables and constant parameters

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Value</th>
<th>Unit</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>$x_0$</td>
<td>0.5</td>
<td>cm</td>
<td>length at birth</td>
</tr>
<tr>
<td>$\lambda$</td>
<td>0.01</td>
<td>g cm$^{-3}$</td>
<td>length–weight constant</td>
</tr>
<tr>
<td>$a_1, a_2$</td>
<td>(1–10)</td>
<td>m$^{-3}$ day$^{-1}$ cm$^{-q}$</td>
<td>maximum attack rate scaling constants (prey types 1, 2)</td>
</tr>
<tr>
<td>$q_1, q_2$</td>
<td>(1–3)</td>
<td>—</td>
<td>maximum attack rate scaling exponent</td>
</tr>
<tr>
<td>$k$</td>
<td>(1–1000)</td>
<td>—</td>
<td>abruptness of ontogenetic niche shift</td>
</tr>
<tr>
<td>$h_1, h_2$</td>
<td>(10–100)</td>
<td>day$^{-1}$ g$^{-1}$ cm$^{-p}$</td>
<td>handling time constant, prey type 1</td>
</tr>
<tr>
<td>$p$</td>
<td>(1–3)</td>
<td>—</td>
<td>handling time scaling exponent</td>
</tr>
<tr>
<td>$\varepsilon$</td>
<td>0.65</td>
<td>—</td>
<td>intake coefficient</td>
</tr>
<tr>
<td>$\rho$</td>
<td>$2.5 \times 10^{-4}$</td>
<td>g day$^{-1}$ mm$^{-3}$</td>
<td>metabolic rate constant</td>
</tr>
<tr>
<td>$\kappa$</td>
<td>0.7</td>
<td>—</td>
<td>allocation coefficient</td>
</tr>
<tr>
<td>$\sigma$</td>
<td>$1.25 \times 10^{-3}$</td>
<td>—</td>
<td>energy for one offspring</td>
</tr>
<tr>
<td>$\mu$</td>
<td>0.1</td>
<td>day$^{-1}$</td>
<td>background mortality rate</td>
</tr>
<tr>
<td>$r_1, r_2$</td>
<td>(0.1)</td>
<td>day$^{-1}$</td>
<td>prey 1, 2 population growth rate</td>
</tr>
<tr>
<td>$K_1, K_2$</td>
<td>(0.1)</td>
<td>g m$^{-3}$</td>
<td>prey 1, 2 carrying capacity</td>
</tr>
</tbody>
</table>

*To avoid excessive notation, we dropped the time argument.

The dimension of $n$ is density (m$^{-3}$) after integration over $i$-state space; that is, $\int n(x, u) \, du \, dx$.

Note: For the parameters that are varied between runs of the model, the range of values or the default value is given in parentheses.
If we let the switch size $u$ increase to infinity, the attack rate on prey type 1 approaches the allometric term for all lengths. Similarly, if we let the switch size decrease to minus infinity, the attack rate on prey type 2 approaches the allometric term. In the rest of this article, we frequently make use of these two limits, denoted $\hat{A}_i(x)$:

$$\hat{A}_1(x) = \lim_{u \to +\infty} A_1(x, u) = a_1 x^{q_1}$$
$$\hat{A}_2(x) = \lim_{u \to -\infty} A_2(x, u) = a_2 x^{q_2}$$

Since the functions $\hat{A}_i(x)$ correspond to the highest possible attack rates on prey type $i$ at body length $x$, we refer to them as the possible attack rates. Accordingly, the functions $A_i(x, u)$ (equations 1 and 2) are referred to as the actual attack rates.

The digestive capacity is assumed to increase with body size, which results in handling times per unit of prey weight that decrease with body size, $H_i(x)$:

$$H_1(x) = h_1 x^{p}$$
$$H_2(x) = h_2 x^{p}$$

While we assume that the same allometric exponent $-p$ applies to both prey types, these types may differ in digestibility and the allometric constants $h_1$ and $h_2$ may therefore differ. We assume a Holling type II functional response for two prey species:

$$f(x, u, F_1, F_2) = \frac{A_1(x, u)F_1 + A_2(x, u)F_2}{1 + A_1(x, u)H_1(x)F_1 + A_2(x, u)H_2(x)F_2}$$

where $F_1$ and $F_2$ denote the densities of the two prey populations, respectively.

Extrapolating the terminology that we use for attack rates, we refer to the function $f(x, u, F_1, F_2)$ as the ‘actual’ intake rate. In the analysis below, we use the term ‘possible’ intake rate to refer to the intake rate of an individual that focuses entirely on one of the two niches. It is given by

$$\hat{f}_i(x, F_i) = \frac{\hat{A}_i(x)F_i}{1 + \hat{A}_i(x)H_i(x)F_i}$$

with $i = 1$ for the first niche and $i = 2$ for the second one, and where $\hat{A}_i(x)$ is the possible attack rate on prey type $i$. Note that $f_1(x, F_1)$ and $f_2(x, F_2)$ are obtained by taking the limit of $f(x, u, F_1, F_2)$ as $u$ approaches $\infty$ and $-\infty$, respectively.
Reproduction and growth

The energy intake rate is assumed to equal the functional response multiplied by a conversion efficiency $\varepsilon$. A fixed fraction $1 - \kappa$ of the energy intake rate is channelled to reproduction. Denoting the energy needed for a single offspring by $\sigma$, the per capita birth rate equals

$$b(x, u, F_1, F_2) = \frac{\varepsilon(1 - \kappa)}{\sigma} f(x, u, F_1, F_2)$$ (9)

To restrict the complexity of our model, we assume that individuals are born mature and that reproduction is clonal. The fraction $\kappa$ of the energy intake rate is used to cover metabolism first and the remainder is used for somatic growth. Assuming that the metabolic rate scales with body volume (proportional to $x^3$), the growth rate in body mass becomes:

$$G_m(x, u, F_1, F_2) = \varepsilon \kappa f(x, u, F_1, F_2) - \rho x^3$$

where $\rho$ is the metabolic cost per unit of volume. Assuming a weight–length relation of the form $W(x) = \lambda x^3$, and using $\frac{dx}{dt} = \frac{dw}{dt}(\frac{dx}{dw})$, we can write the rate of growth in length as:

$$g(x, u, F_1, F_2) = \frac{1}{3\lambda x} (\varepsilon \kappa f(x, u, F_1, F_2) - \rho x^3)$$ (10)

The length at which the growth rate becomes zero is referred to as $x_{\text{max}}$. Individuals with a size beyond $x_{\text{max}}$ have a negative growth rate (but a positive birth rate). Since in the analysis below we assume population dynamic equilibrium, we ensure that no individual grows beyond the maximum size. Note that in the special case with $p = q_1 = q_2 = 2$ the function $g$ becomes linear in $x$, yielding the classic von Bertalanffy growth model (von Bertalanffy, 1957).

Prey dynamics

The population size distribution is denoted by $n(x, u)$. For the analyses of the deterministic model below, we assume that the (resident) population is monomorphic in $u$. Therefore, we do not have to integrate over switch sizes $u$ but only over sizes $x$ to obtain the total population density,

$$N_{\text{tot}}(u) = \int_{x_{\text{min}}}^{x_{\text{max}}} n(x, u) \, dx$$ (11)

We assume that the two prey populations grow according to semi-chemostat dynamics and that they do not interact with each other directly. The dynamics of the prey populations can then be described by:

$$\frac{dF_1}{dt} = r_1(K_1 - F_1) - \int_{x_{\text{min}}}^{x_{\text{max}}} A_1(x, u) F_1 \frac{n(x, u)}{1 + A_1(x, u) H_1(x) F_1 + A_2(x, u) H_2(x) F_2} \, dx$$ (12)

$$\frac{dF_2}{dt} = r_2(K_2 - F_2) - \int_{x_{\text{min}}}^{x_{\text{max}}} A_2(x, u) F_2 \frac{n(x, u)}{1 + A_1(x, u) H_1(x) F_1 + A_2(x, u) H_2(x) F_2} \, dx$$ (13)
where \( r_1, \) \( r_2, \) \( K_1 \) and \( K_2 \) are the maximum growth rates and maximum densities of the two prey populations, respectively. The integral term in each equation represents the predation pressure imposed by the predator population.

The PDE formulation of the model is given in Table 2 and the individual level model is summarized in Table 3.

**Parameterization**

Since we intend to study the effect of the size-scaling of the functional response on the evolution of the ontogenetic niche shift, the parameters \( a_1, \) \( a_2, \) \( h_1, \) \( h_2, \) \( p, \) \( q_1 \) and \( q_2 \) are not fixed. Depending on whether handling time and search rate are determined by processes related to body length, surface or volume, the allometric exponents \( p, \) \( q_1 \) and \( q_2 \) are close to 1, 2 or 3, respectively. The remaining, fixed parameters are based on the parameterization of a more detailed model of perch (Claessen et al., 2000).

**ECOLOGICAL DYNAMICS**

Before we can study evolution of the ontogenetic niche shift, we have to assess the effect of the ontogenetic niche shift on the ecological dynamics. Our model (Table 2) is not analytically solvable. Instead, we study its dynamics through a numerical method for the integration of physiologically structured population models, called the Escalator Boxcar Train (de Roos et al., 1992; de Roos, 1997). When restricting attention to a single prey type (which is equivalent to assuming \( u \gg x_{\text{max}} \)) and to the special case \( p = q_1 = 2, \) our model reduces to the Kooijman-Metz model, of which the population dynamics are well documented in the literature (e.g. de Roos et al., 1992; de Roos, 1997). Numerical studies of the equilibrium behaviour of this simplified model show that the population dynamics always converge to a stable equilibrium, which can be attributed to the absence of a juvenile delay and to the semi-chemostat (rather than, for example, logistic) prey dynamics (cf. de Roos, 1988; de Roos et al., 1990). Simulations show that, also for the general functional

| Table 2. The model: specification of dynamics
| --- |
| PDE | \( \frac{\partial n}{\partial t} + \frac{\partial g n}{\partial x} = -\mu n(x, u) \)
| Boundary condition | \( g(x, u, F_1, F_2)n(x, u) = \int_{x_0}^{x_{\text{max}}} b(x, u, F_1) n(x, u) \, dx \)
| Prey dynamics | \( \frac{dF_1}{dt} = r_1(K_1 - F_1) - \int_{x_0}^{x_{\text{max}}} \frac{A_1(x, u) F_1}{1 + A_1(x, u) H_1(x) F_1 + A_2(x, u) H_2(x) F_2} \, n(x, u) \, dx \)
| | \( \frac{dF_2}{dt} = r_2(K_2 - F_2) - \int_{x_0}^{x_{\text{max}}} \frac{A_2(x, u) F_2}{1 + A_1(x, u) H_1(x) F_1 + A_2(x, u) H_2(x) F_2} \, n(x, u) \, dx \)

*The time argument has been left out from all variables and functions.
*Note: The functions defining the birth rate \( b \), growth rate \( g \), attack rates \( A_1, A_2 \) and handling times \( H_1, H_2 \) are listed in Table 3, parameters in Table 1. PDE = partial differential equation.
response (with values of \( p, q_1 \) and \( q_2 \) between 1 and 3), the equilibrium is stable for all investigated parameter combinations.

It is possible to choose parameter values (e.g. small \( K_i \) or high \( h_i \)) for which the predator population cannot persist on either prey 1 or prey 2 alone. In the results presented below, we use parameter values that allow for persistence on either prey type separately.

### Ontogenetic niche shift and prey densities

We now examine the ecological effect of the size at the ontogenetic niche shift on the equilibrium state of a monomorphic size-structured population and the two prey populations. Each specific choice of \( u \) and the parameters results in a stable size distribution \( n(x,u) \) and equilibrium prey densities \( F_1 \) and \( F_2 \). The effect of the switch size \( u \) on the prey densities \( F_1 \) and \( F_2 \), on the total predator population density \( N_{\text{tot}}(u) \) and on the the maximum length in the predator population \( x_{\text{max}} \) is shown in Fig. 2 for two different parameter combinations.

Three conclusions can readily be drawn from Fig. 2. First, prey density \( F_1 \) or \( F_2 \) is low if most of the predator population consumes prey 1 or prey 2, respectively. Second, the total number of predators, \( N_{\text{tot}}(u) \), reaches a maximum for an intermediate switch size \( u \) (i.e. when predators exploit both prey). Third, the maximum length in the predator population correlates strongly with the density of the second prey provided that individuals reach the size at which the ontogenetic niche occurs (i.e. \( x_{\text{max}} > u \)).

With very low or very high \( u \), the system reduces to a one-consumer, one-resource system. If the switch size is very large (\( u > x_{\text{max}} \); for example, \( u > 2.5 \) in Fig. 2), individuals never reach a size large enough to start exploiting the second prey. The second prey population is hence at the carrying capacity \( K_2 \), whereas the first prey is heavily exploited. Similarly, for a very small switch size (\( u < x_{\text{b}} \); for example, \( u = 0 \) in Fig. 2), even newborns have a low efficiency on prey type 1. In this case, prey 1 is near its carrying capacity \( K_1 \) and prey 1 is depleted. The two extreme strategies \( u > x_{\text{max}} \) and \( u < x_{\text{b}} \), therefore, characterize specialists

### Table 3. The model: individual level functions

<table>
<thead>
<tr>
<th>Function</th>
<th>Expression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Attack rate on prey 1</td>
<td>( A_1(x,u) = a_1 x^{q_1} \frac{1}{1 + e^{k_1 (x - u)}} )</td>
</tr>
<tr>
<td>Attack rate on prey 2</td>
<td>( A_2(x,u) = a_2 x^{q_2} \left( 1 - \frac{1}{1 + e^{k_2 (x - u)}} \right) )</td>
</tr>
<tr>
<td>Handling time, prey 1</td>
<td>( H_1(x) = h_1 x^{-p} )</td>
</tr>
<tr>
<td>Handling time, prey 2</td>
<td>( H_2(x) = h_2 x^{-p} )</td>
</tr>
<tr>
<td>Functional response</td>
<td>( f(x,u,F_1,F_2) = \frac{A_1(x,u)F_1 + A_2(x,u)F_2}{1 + A_1(x,u)H_1(x)F_1 + A_2(x,u)H_2(x)F_2} )</td>
</tr>
<tr>
<td>Maintenance requirements</td>
<td>( M(x) = \rho x^\theta )</td>
</tr>
<tr>
<td>Growth rate in length</td>
<td>( g(x,u,F_1,F_2) = \frac{1}{3} \lambda x \left( \kappa \sigma f(x,u,F_1,F_2) - \rho x^\theta \right) )</td>
</tr>
<tr>
<td>Birth rate</td>
<td>( b(x,u,F_1,F_2) = \frac{\epsilon (1 - \kappa)}{\sigma} f(x,u,F_1,F_2) )</td>
</tr>
</tbody>
</table>
on prey 1 and prey 2, respectively. Although at first sight a strategy $u < x_b$ appears to be biologically meaningless, it can be interpreted as a population that has lost the ability to exploit a primary resource which its ancestors used to exploit in early life stages. This evolutionary scenario turns up in the results (see pp. 208–210).

A striking result evident from Fig. 2 is the discontinuous change in maximum length at high values of $u$. For $u$ beyond the discontinuity, growth in the first niche is insufficient to reach the ontogenetic niche shift, such that the maximum length is determined only by the prey density in the first niche. As soon as the switch size is reachable in the first niche, the maximum size is determined by the prey density in the second niche. Just to the left of the discontinuity, only a few individuals live long enough to enter the second niche, and the impact of these individuals on the second prey is negligible ($F_2 = K_2$). These few survivors thrive well in the second niche and reach giant sizes (Fig. 2). This sudden change in asymptotic size corresponds to a fold bifurcation (see also Claessen et al., in press).

An important general conclusion from Fig. 2 is that there is a strong ecological feedback between the niche switch size $u$ and the environment ($F_1$ and $F_2$ equilibrium densities). Changing $u$ may drastically change prey densities, which, in turn, may change predator population density and individual growth rates. Comparison of Fig. 2a with Fig. 2b suggests that specific choices for the parameters of the size scaling of the functional response do not affect the general pattern. We have studied many different parameter combinations of $a_1$, $a_2$, $h_1$, $h_2$, $p$, $q_1$ and $q_2$ and all give the same overall pattern as illustrated in Fig. 2.
PAIRWISE INVASIBILITY PLOTS

This section briefly outlines the methodology and terminology that we use in our study of the evolution of the switch size $u$. Our evolutionary analysis of the deterministic model is based on the assumptions that (1) mutations occur rarely, (2) mutation steps are small and (3) successful invasion implies replacement of the resident type by the mutant type. The robustness of these assumptions will be evaluated later (see pp. 208–210). Under these assumptions, evolution boils down to a sequence of trait substitutions. To study this, we consider a monomorphic resident population with genotype $u$ and determine the invasion fitness of mutants, whose strategy we denote $u'$. With our model of the ecological interactions (see previous section on ‘Ecological dynamics’), we can determine the fitness of a mutant type from the food densities $F_1$ and $F_2$, as is shown on pp. 200–201. Since the food densities are set by the resident population, the fitness of mutants depends on the strategy of the resident. If the lifetime reproduction, $R_0$, of a mutant exceeds unity, it has a probability of invading and replacing the resident (Metz et al., 1992).

For all possible pairs of mutants and residents, the expected success of invasion by the mutant into the ecological equilibrium of the resident can be summarized in a so-called pairwise invasibility plot (van Tienderen and de Jong, 1986). For example, Fig. 3a is a pairwise invasibility plot for residents and mutants in the range of switch sizes from 0 to 2 cm, based on our model (Table 2). It shows that, if we choose a resident with a very small switch size, say $u = 0.1$, all mutants with a larger trait value ($u' > u$) have the possibility to invade the resident, whereas mutants with a smaller trait value ($u' < u$) have a negative invasion fitness and hence cannot establish themselves. Thus, the resident is predicted to be replaced by a mutant with a larger switch size. Upon establishment, this mutant becomes the new resident and the pairwise invasibility plot can be used to predict the next trait substitution. Figure 3a shows that, as long as the resident type is below $u^*$, only mutants with a larger trait value ($u' > u$) can invade. Thus, if we start with a resident type below $u^*$, the adaptive process results in a stepwise increase of the resident trait value towards $u^*$. A similar reasoning applies to the residents with a trait value above $u^*$. Here, only mutants with a smaller switch size can invade (Fig. 3a). Therefore, starting from any initial resident type near $u^*$, the adaptive process results in convergence of the resident to $u^*$. The strategy $u^*$ is hence an evolutionary attractor.

In a pairwise invasibility plot, the borders between areas with positive and negative invasion fitness correspond to zero fitness contour lines. The diagonal ($u' = u$) is necessarily a contour line because mutants with the same strategy as the resident have the same fitness as the resident. Intersections of other contour lines with the diagonal are referred to as evolutionarily singular points (e.g. $u^*$). Above, we used the pairwise invasibility plot to determine the convergence stability of $u^*$, but we can also use it to determine the evolutionary stability of singular points. For example, Fig. 3a shows that if the resident has strategy $u^*$, all mutant strategies $u' \neq u$ have negative invasion fitness. A resident with switch size $u^*$ is therefore immune to invasion by neighbouring mutant types and it is thus an evolutionarily stable strategy (ESS). A singular point that is both convergence stable and evolutionarily stable is referred to as a continuously stable strategy (CSS; Eshel, 1983). In general, the dynamic properties of evolutionarily singular points can be determined from the slope of the off-diagonal contour line near the singular point (Metz et al., 1996a; Dieckmann, 1997; Geritz et al., 1998). In our analysis below, we find four different types of singular points. As we showed above, $u^*$ in Fig. 3a corresponds to a CSS. In Fig. 3b, the
singular point $u^*$ is again an evolutionary attractor. However, once a resident population with strategy $u^*$ has established itself, mutants on either side of the resident (i.e. both $u' > u$ and $u' < u$) have positive fitness. Since mutants with the same strategy as the resident have zero invasion fitness, the singular point $u^*$ is located at a fitness minimum. It should be pointed out here that, under frequency-dependent selection, evolutionary stability and evolutionary convergence (or attainability) are completely independent (Eshel, 1983). In spite of being a fitness minimum, the strategy $u^*$ in Fig. 3b is nevertheless an evolutionary attractor. As will become clear below (see pp. 208–210), a singular point that is convergence stable but evolutionarily unstable (e.g. $u^*$ in Fig. 3b) is referred to as an evolutionary branching point (Metz et al., 1996a; Geritz et al., 1997).

In Fig. 3c, the singular point $u^*$ is also an evolutionary attractor, but it is evolutionarily neutral; if the resident is $u^*$, all mutants have zero invasion fitness. We consider it a degenerate case, because even the slightest perturbation results in the situation of Fig. 3a or Fig. 3b.

The last type of singular point that we will encounter is illustrated in Fig. 4. In these pairwise invasibility plots, there are two evolutionarily singular points, of which $u^*$ is an evolutionary branching point. From the sign of the invasion fitness function around the singular point $u^*$, we can see that if we start with a resident close to the singular point,
mutants with a strategy even closer to \( u_r \) cannot invade. Rather, successful invaders lie further away from \( u_r \). Trait substitutions are hence expected to result in evolution away from \( u_r \). Singular points such as \( u_r \) in Fig. 4 are convergence unstable and are referred to as evolutionary repellers (Metz et al., 1996a).

**EVOLUTIONARY DYNAMICS**

In this section, we study the evolution of the size at niche shift (\( u \)) within the ecological context established in the section on ‘Ecological dynamics’. First, we use the deterministic model to find evolutionarily singular points and their dynamic properties, using the method outlined in the previous section. Second, we interpret them in terms of ecological mechanisms. Third, we use numerical simulations of a stochastic individual-based version of the same model to check the robustness of the derived predictions.

**Invasion fitness of mutants**

We first have to determine the fitness of mutants as a function of their own switch size \( u' \) and of the the resident’s switch size \( u \). With our individual-level model (see section on ‘Ecological dynamics’), we can relate the lifetime reproduction, \( R_0 \), of a mutant to its strategy. We can use \( R_0 \) as a measure of invasion fitness, because a monomorphic resident population with strategy \( u \) can be invaded by mutants with strategy \( u' \) if the expected lifetime reproduction of the mutant in the environment set by the resident exceeds unity – that is, if \( R_0(u', u) > 1 \) (Mylíus and Dieckmann, 1995).

The environment that a mutant experiences consists of the two prey densities, which are in equilibrium with the resident population, so we write \( F_i(u) \) and \( F_j(u) \). The mutant’s
length–age relation can be obtained by integration of equation (10) after substitution of $F_1(u)$ and $F_2(u)$. Knowing the growth trajectory, the birth rate as a function of age can be calculated from equation (9). We denote this age-specific birth rate by $B(a, u', u)$, where $a$ denotes age. The mutant’s lifetime reproduction $R_0$ is then found by integration of this function, weighted by the probability of surviving to age $a$, over its entire life history:

$$R_0(u', u) = \int_0^\infty e^{-\mu u} B(a, u', u) \, da$$

Based on the assumption of size-independent mortality, $R_0(u', u)$ is a monotonically increasing function of the feeding rate at any size. The reason is straightforward: an increased feeding rate implies an increased instantaneous birth rate, as well as an increased growth rate. The size-specific birth rate $b$ (equation 9) is monotonically increasing in $x$. These three facts imply that an increase in the intake rate at any size increases the lifetime reproduction (in a constant environment).

For each value of the resident’s trait $u$ from the range between the two specialist trait values ($u = 0 \ldots 4$), we numerically determine the function $R_0(u', u)$ for values of $u'$ from the same range. The results of these calculations are summarized in pairwise invisibility plots (see pp. 198–200), which show the contour lines $R_0(u', u) = 1$ and the sign of $R_0(u', u) - 1$ (Figs 3 and 4).

The results for many different parameter combinations show that there are five qualitatively different pairwise invisibility plots, which are represented in Figs 3 and 4. All five pairwise invisibility plots have one important feature in common: there is an intermediate switch size that is an evolutionary attractor of the monomorphic adaptive dynamics. We denote this attractor by $u^*$ and refer to it as the generalist strategy. In Fig. 3, $u^*$ is a global attractor, whereas in Fig. 4 there is also an evolutionary repeller. Choosing a resident switch size beyond the repeller leads to evolution towards a single specialist population, leaving the other niche (the first niche in Fig. 4a; the second in Fig. 4b) unexploited. We first discuss the evolutionary attractor $u^*$ and return to the evolutionary repellers later in the section.

Evolutionary convergence to the generalist $u^*$

Here we relate the results presented in Fig. 3 to the underlying ecological mechanisms. We can explain the different evolutionary outcomes by considering the life history of individuals in terms of their size-dependent food intake rate (equation 7). To clarify the ecological mechanism, we compare the size-dependent food intake rate of a resident individual with the possible intake rates in each niche separately (equation 8; Fig. 5). Thus, we gain insight into whether the actual intake rate at a certain size is above or below the possible intake rate at that size.

The length at which the possible intake rates $\hat{f}_i(x, F_i)$ and $\hat{f}_j(x, F_j)$ (equation 8) intersect is denoted $x_c$. This particular body length is of special interest, because one niche is more ‘profitable’ to individuals smaller than $x_c$, whereas the other niche is more profitable to individuals larger than $x_c$. Here, ‘more profitable’ is defined as ‘providing a higher possible intake rate’. To an individual of length $x = x_c$, the two niches are hence equally profitable. Figure 5 illustrates that the evolutionary attractor $u^*$ is that particular strategy for which the switch size $u$ coincides with the intersection of the possible intake rates (i.e. $x_c = u$).

Depending on the size scaling of the two possible intake rates, two generic cases can be distinguished: (a) the first niche is more profitable than the second one to individuals
smaller than $x_e$, but less profitable to individuals larger than $x_e$, and (b) vice versa. The two cases are illustrated in Fig. 5a and b, respectively. Note that the switch size $u$ and the intersection of the two possible intake rates coincide. (a) Possible attack rate is proportional to body length in the first niche ($q_1 = 1$) and proportional to body surface area in the second ($q_2 = 2$); the resident ($u^* = 0.68$) is a continuously stable strategy (CSS). (b) Possible attack rate is proportional to body surface area in the first niche ($q_1 = 2$) and proportional to body length in the second ($q_2 = 1$); the resident ($u^* = 0.683$) is an evolutionary branching point (EBP). Other parameters: $k = 30$, $p = 2$, $a_1 = a_2 = 1$, $h_1 = h_2 = 10$ and as in Table 1.

Why $u^*$ is an evolutionary attractor can be understood by considering a perturbation in the switch size $u$; that is, by choosing a resident strategy $u$ slightly smaller or larger than $u^*$. In this case, the possible intake rates intersect at some body size $x \neq u$. In Fig. 6 (right-hand panels), the resident has a strategy slightly above the generalist strategy ($u > u^*$). Compared with Fig. 5, the curves of the two possible intake rates have shifted; $\hat{f}_1$ downward and $\hat{f}_2$ upward. The reason is that the prey densities $F_1$ and $F_2$ depend on the resident strategy $u$ (Fig. 2). As a consequence, to an individual with length equal to the switch length ($x = u$), the second niche seems underexploited. We define the ‘underexploited’ niche as the niche that gives an individual of length $x = u$ the highest possible intake rate (equation 8). The other niche is referred to as ‘overexploited’.

Now, consider a mutant with a strategy $u'$ in the environment set by a resident with $u > u^*$. If the mutant has a smaller switch size than the resident, it switches to the underexploited niche before the resident does. Its intake rate, therefore, is higher than the resident’s intake and, since fitness increases monotonically with the intake rate, the mutant can
invade. Mutants that switch later than the resident, however, spend more time in the over-exploited niche, have a lower intake rate and hence cannot invade. This shows how natural selection drives the system in the direction of the generalist $u^*$ when started from a resident with $u > u^*$.

For the case $u < u^*$, the opposite reasoning applies: a resident that switches between niches at a relatively small size underexploits the first niche and overexploits the second one. The curve describing the possible intake rate in the first niche ($f_1$) shifts upward, whereas the curve for the second niche ($f_2$) shifts downward (Fig. 6, left-hand panels). Only mutants that switch later ($u' > u$) profit more from the underexploited niche than the resident, and hence only these mutants can invade, such that evolution moves the system towards the generalist $u^*$ when started from a resident with $u < u^*$.

In summary, if one niche is underexploited, natural selection favours mutants that exploit this niche more. In consequence, only mutants that are closer to the generalist strategy $u^*$ than the resident can invade. This suggests that $u^*$ is an evolutionary attractor. Convergence to $u^*$, however, also depends on the effect of the environmental feedback on $x_c$. That is, once an invading strategy has replaced the old resident, it gives rise to a new ecological equilibrium. Because $x_c$ depends on the prey densities $F_1$ and $F_2$, we need to check the relation between resident switch size $u$ and the resultant $x_c$.

Again, we have to distinguish between cases (a) and (b) because the slopes of the possible intake rates at their intersection are crucial. Figure 6 shows that in case (a) the second niche is underexploited if $x_c < u$ and overexploited if $x_c > u$. This means that evolutionary
convergence to $u^*$ is guaranteed if all residents with $u > u^*$ have an intersection point $x_e < u$ and all residents with $u < u^*$ have an intersection point $x_e > u$. Figure 7a shows that this is indeed the case. In case (b), the second niche is underexploited if $x_e > u$ and overexploited if $x_e < u$. For convergence to $u^*$, the relation between $x_e$ and $u$ should hence be opposite to case (a); Fig. 7 confirms that this applies. The relations in Fig. 7, and hence convergence to $u^*$, hold as long as the following condition is fulfilled at $u = u^*$:

$$\left. \frac{\partial \hat{f}_1}{\partial u} \right|_{x = u} < \left. \frac{\partial \hat{f}_2}{\partial u} \right|_{x = u}$$

Although we cannot prove that this condition is met in general, intensive numerical investigations have found no exception for any parameter combinations. We conjecture that the inequality above can be taken for granted if the following, more elementary, condition is fulfilled at $u = u^*$:

$$\frac{\partial F_1}{\partial u} < \frac{\partial F_2}{\partial u}$$

**Evolutionary stability of the generalist $u^*$**

The pairwise invasibility plots (Fig. 3) suggest that the evolutionary attractor $u^*$ is either a continuously stable strategy (CSS), an evolutionary branching point (EBP) or neutral. Which of these cases applies depends on the size scaling of the possible intake rates in the two niches. We show this by considering the two generic possibilities in Fig. 5, starting with case (a). For a resident that is smaller than its switch size, the first niche is more profitable than the second – that is, $\hat{f}_1(x) > \hat{f}_2(x)$ for $x < u$ (Fig. 5a). Consequently, mutants that switch

![Fig. 7](image_url)

**Fig. 7.** The environmental feedback represented by the body length for which the two niches are equally profitable ($x_e$) as a function of the resident switch length ($u$). (a) and (b) as in Fig. 5 and Fig. 6. The switch size for which $x_e = u$ is referred to as the generalist strategy, denoted $u^*$. In (a) $u^* = 0.68$ and in (b) $u^* = 0.683$. 
earlier than the resident \((u' < u)\) switch to the second niche at a size at which the second niche is still less profitable to them than the first. They hence have lower fitness than the resident. For individuals larger than the resident switch size, the second niche is more profitable than the first – that is, \(f_2(x) > f_1(x)\) for \(x > u\). This implies that mutants that switch later than the resident \((u' > u)\) stay in the first niche, although this niche has become less profitable to them than the second one. These mutants, too, have lower fitness than the resident. Since mutants on both sides of the resident strategy cannot invade, the generalist \(u^*\) is an evolutionary attractor.

Case (b) is simply the opposite of the previous case. The first niche is less profitable to individuals smaller than the switch size, whereas the second niche is less profitable to individuals larger than the switch size. As a consequence, mutants that switch earlier \((u' < u)\) switch to the second niche while it still is more profitable to them. Mutants that switch later \((u' > u)\) stay in the first niche when it becomes more profitable to them. The evolutionary attractor \(u^*\) thus lies at a fitness minimum and, since it is nevertheless convergence stable, it is an evolutionary branching point.

Which biological conditions give rise to cases (a) and (b)? In the next two subsections, we derive conditions for theses cases in terms of our model parameters; this allows for a qualitative comparison between our results and empirical data on the size scaling of functional responses. To aid our biological interpretation of the results and because of the complexity of equation (7), we apply two alternative simplifying assumptions. In a first scenario, we assume that the handling times for the two prey types are equal \((h_1 = h_2)\). In a second scenario, we consider different handling times, but assume the same possible attack rates in both niches \((a_1 = a_2, q_1 = q_2)\).

**Scenario 1: different attack rates**

Here, we assume that the only difference between the two niches is the size scaling of the possible attack rates, whereas handling times are assumed to be the same. In this case, we can find an explicit expression for the length \(x_e\) at which the two possible intake rates intersect. The intersection \(x_e\) is obtained by substituting \(h_1 = h_2 = h\) in the possible intake rates (equation 8) and by solving for \(f_1(x) = f_2(x)\):

\[
x_e = \left(\frac{a_1}{a_2} \right)^{1/(q_2 - q_1)}
\]

(15)

To distinguish between cases (a) and (b), we define a function \(D(x)\) that is the difference between the possible intake rates in the two niches:

\[
D(x) = f_1(x) - f_2(x)
\]

(16)

In case (a), the first niche is more profitable before the switch, while the second one is more profitable after the switch; this requires that the slope of \(D(x)\) evaluated at \(x = x_e\) is negative. Case (b) results when the slope of \(D(x)\) at size \(x_e\) is positive.

The function \(D(x)\) can be written as

\[
D(x) = \frac{a_1 F_1 x^{q_1} - a_2 F_2 x^{q_2}}{1 + a_1 F_1 x^{q_1 - p} h + a_2 F_2 x^{q_2 - p} h + a_1 F_1 a_2 F_2 x^{q_1 - 2p + q_1 - q_2} h^2}
\]

(17)
By definition, \( D(x_e) = 0 \), so we only have to consider the sign of \( D(x) \) around \( x = x_e \). Since the denominator of equation (17) is always positive, we have to determine the sign of the numerator only. The numerator is positive for a length \( x \) between 0 and \( x_e \) if, and only if, \( q_1 < q_2 \). Thus we arrive at the conditions:

\[
\begin{align*}
q_1 < q_2 & \quad \text{CSS} \\
q_1 = q_2 & \quad \text{neutral} \\
q_1 > q_2 & \quad \text{EBP}
\end{align*}
\]

(18)

If the possible attack rate on the first prey type increases faster with body size than the possible attack rate on the second prey type (Fig. 5b), the evolutionary attractor \( u^* \) is predicted to be an evolutionary branching point (Fig. 3b). Otherwise, the generalist is predicted to be a CSS or to be neutral and the population to remain monomorphic. Note that Fig. 2, Fig. 5 and Fig. 7 illustrate this first scenario.

**Scenario 2: different handling times**

Here, we assume that the possible attack rates are the same (i.e. \( a_1 = a_2 = a \), \( q_1 = q_2 = q \)), but that the two prey types differ in digestibility (i.e. \( h_1 \neq h_2 \)). The reasoning is analogous to that applied in the first scenario. The length at which the niches are equally profitable is:

\[
x_e = \left( \frac{F_1 - F_2}{aF_1F_2(h_1 - h_2)} \right)^{1/(q-p)}
\]

(19)

The difference between the possible intake rates is:

\[
D(x) = \frac{aF_1F_2x^{q-p}(h_2 - h_1) + F_1 - F_2}{(1 + ax^{q-p}h_1F_1)(1 + ax^{q-p}h_2F_2)}
\]

(20)

Again, the denominator is always positive, so we consider the numerator only. Here it is crucial to recognize that \( D(x) \) is increasing if

\[
(h_2 - h_1)axF_1F_2x^{q-p}
\]

is increasing in \( x \). Since \( x^{q-p} \) is increasing in \( x \) if \( p < q \) and decreasing if \( p > q \), we arrive at the following conditions for the evolutionary stability of the generalist \( u^* \):

\[
\begin{align*}
p > q \text{ and } h_1 < h_2 & \quad \text{CSS} \\
p < q \text{ and } h_1 > h_2 & \quad \text{CSS} \\
p = q \text{ or } h_1 = h_2 & \quad \text{neutral} \\
p > q \text{ and } h_1 > h_2 & \quad \text{EBP} \\
p < q \text{ and } h_1 < h_2 & \quad \text{EBP}
\end{align*}
\]

(22)

Interpretation of these conditions is less obvious than for the first scenario and requires consideration of the size-dependent functional response (equation 7). If \( p > q \), the maximum intake rate on a pure diet of prey \( i \), \( H_i(x)^{-1} \), increases faster with body size than the search rate. This means that, with increasing body size, the feeding rate becomes less
limited by digestive constraints and more limited by prey abundance. This can be clarified by the case of a single prey population, assuming a constant prey density $F$. Dividing the functional response $f$ by the maximum intake rate, $H(x)^{-1}$, we obtain the level of saturation as a function of body size:

$$
\left( \frac{1}{ahF x^{q-p} + 1} \right)^{-1}
$$

which is a decreasing function of $x$ if $p > q$ and an increasing one if $p < q$. If the feeding rate is well below its maximum, the intake rate correlates strongly with the encounter rate between predator and prey, and the individual is ‘search limited’. If, on the other hand, the feeding rate is close to its maximum, the intake rate correlates weakly with prey abundance, and individuals are ‘handling limited’. For $p = q$, the level of saturation is independent of body size (like, for example, in the Kooijman-Metz model with $p = q = 2$).

Recall that, for a resident of size $x = u^*$, the two prey types are equally profitable (Fig. 5). If the feeding rate becomes more handling limited with body size ($p < q$), then for individuals larger than $u^*$, the prey that is more digestible (smaller $h_1$) is the more profitable one. If, on the other hand, the feeding rate becomes more search limited with body size, then for larger individuals, the more abundant prey (higher $F_i$) is more profitable. Rewriting equation (19) gives a relation between the prey densities at equilibrium of the resident population with switch size $u^*$:

$$
F_2 = \frac{F_1}{1 + (h_1 - h_2) a F_i x^{q-p}}
$$

This implies that the less digestible prey is the more abundant prey:

$$
h_1 > h_2 \Leftrightarrow F_1 > F_2 \quad \text{at } u = u^*
$$

We first investigate the case $h_1 > h_2$, $p < q$, and consider a resident population with the singular strategy $u = u^*$ and a mutant that switches at a larger size than the resident ($u' > u$). In the size interval between the resident’s switch size $u$ and its own switch size $u'$, the resident shifts its focus to prey 2 while the mutant is still focusing on prey 1. The mutant thus consumes the less digestible prey while it is relatively handling limited (relative to the size at which the two prey are equally profitable, $u^*$). Its intake rate is therefore smaller than that of the resident and hence also its lifetime reproduction. A mutant that switches at a smaller size than the resident ($u' < u$) consumes the less abundant prey 2 already at a size where it is relatively search limited. Also, this mutant has a smaller $R_0$ than the resident. Since mutants with $u' > u$ or $u' < u$ both cannot invade, the singular strategy $u^*$ is a CSS if $h_1 > h_2$ and $p < q$. For $h_1 < h_2$ and $p > q$, an analogous reasoning applies.

We now consider the case $h_1 > h_2$ and $p > q$. A mutant that switches at a larger size continues consuming the more abundant prey 1 while it is relatively search limited, yielding a higher feeding rate and hence a higher fitness than the resident. A mutant that switches at a smaller size starts consuming the more digestible prey 2 while it is relatively handling limited, also yielding a higher fitness than the resident. Thus, mutations in both directions yield a higher fitness than the resident, which implies that the singular strategy is a branching point. Again, a completely analogous reasoning applies for $h_1 < h_2$ and $p < q$. 

Ontogenetic niche shift and evolutionary branching 207
Evolutionary repellers

Under the assumptions that \( a_1 = a_2 = a \) and \( q_1 = q_2 = q \), we have identified parameter configurations leading to two singular points, where one is the generalist strategy \( u^* \) and the other is an evolutionary repeller (Fig. 4a,b). A repeller occurs at a small trait value if \( p > q \) and \( h_1 \) is sufficiently high (Fig. 4a). In contrast, a repeller occurs at a large trait value if \( p < q \) and \( h_2 \) is sufficiently high (Fig. 4b). In the latter case, if the population starts out with a trait value above the repeller, directional selection moves the population away from \( u^* \) and towards the strategy that is a specialist on prey 1. It is interesting to note – and, because of the asymptotic shape of the sigmoidal functions (Fig. 1), also biologically expected – that the fitness gradient goes asymptotically to zero as the resident switch size becomes larger. Similarly, starting below the repeller in the case with \( p > q \), the population evolves to a specialist on prey 2, leaving the first prey unexploited. The existence of the repellers relates to the fact that, for severely handling-limited individuals, the less digestible prey type can be less profitable than the more digestible prey type even if the former’s density is at its carrying capacity and the latter’s density is low.

After branching: dimorphism of switch sizes

What happens after the adaptive dynamics of switch sizes has reached an evolutionary branching point, such as \( u^* \) in Fig. 3b? Mutants on either side of \( u^* \) can invade the resident population, which may give rise to the establishment of two (slightly more specialized) branches and exclusion of the generalist \( u^* \) (Metz et al., 1996a; Geritz et al., 1997). Whether the branches can co-exist depends on whether they can invade into each other’s monomorphic equilibrium population. The set of \( u' \) and \( u \) strategies that can mutually invade is referred to as the set of protected dimorphisms. This set is found by flipping the pairwise invasibility plot (Fig. 3b) around the diagonal \( u' = u \) (corresponding to a role reversal of the two considered strategies) and superimposing it on the original (Geritz et al., 1998): combinations of strategies \((u, u')\) for which the sign of \( R_0(u', u) - 1 \) before and after the flip is positive are protected dimorphisms and can co-exist. The set of protected dimorphisms in the vicinity of the branching point \( u^* \) is referred to as the co-existence cone and its shape has implications for the adaptive dynamics after branching. Specifically, the width of the cone determines the likelihood that evolutionary branching occurs and that the two branches persist: branching is more likely if the cone is wide. The reason is that mutation-limited evolution can be seen as a sequence of trait substitutions, which behaves like a directed random walk (Metz et al., 1992; Dieckmann and Law, 1996). Due to the stochastic nature of this process, there is a probability of hitting the boundary of the co-existence cone, which results in the extinction of one of the two branches. The co-existence cone is wider the smaller the acute angle between the two contour lines at their intersection point \( u^* \). In our model, this angle depends on the abruptness of the ontogenetic switch. If the shift is more gradual (corresponding to a lower value of \( k \)), the angle is smaller and, consequently, the co-existence cone is wider. Hence, with a gradual niche shift, evolutionary branching is more likely to occur than with a more discrete switch.

To determine whether our results are robust against relaxing some of the simplifying assumptions inherent to the deterministic, monomorphic model considered in this article up to now, we investigate a stochastic, individual-based model (IBM) that corresponds to the deterministic model (Tables 2 and 3). In the IBM, the growth dynamic of individuals is
still deterministic, but birth and death are modelled as discrete events. An offspring receives the same trait value as its clonal parents unless a mutation occurs, which we assume to occur with a fixed probability of $P = 0.1$ per offspring. The offspring’s trait value is then drawn from a truncated normal distribution around the parental trait value. The standard deviation of the mutation distribution can be varied (we have considered values between 0.001 and 0.01). An essential feature of the IBM, and a major difference with the deterministic model studied above, is that it naturally allows for polymorphism to arise.

Convergence to the predicted singular point $u^*$ and the subsequent emergence of a switch-size dimorphism in simulations of the IBM (e.g. Fig. 8) confirm the robustness of the results derived from the deterministic model. In particular, this shows that the assumption in our deterministic model that the strategy of offspring is identical to their parents’ strategy is not critical to the results. The stochastic IBM has been studied for many different parameter combinations, and branching occurs only in runs with parameter settings for which this is predicted by the deterministic model (cf. conditions 18 and 22). Secondary branching, potentially giving rise to greater polymorphism, has not been observed.

The IBM allows us to study the evolution of the ontogenetic niche shift after branching. We will refer to the two emerging branches as A and B and denote the average switch sizes in the two branches as $u_A$ and $u_B$, respectively, such that $u_A > u_B$ (Fig. 8). Figure 8 illustrates that the branches in the dimorphic population evolve towards two specialist strategies. Switch size $u_A$ approaches the maximum size $x_{\text{max}}$, such that virtually all A-individuals consume prey 1 exclusively. Switch size $u_B$ approaches the length at birth ($x_b$), such that individuals in branch B consume prey 2 throughout their entire lives. Prey densities remain approximately constant after branching. With constant prey densities, the possible intake rates are also constant, and this observation enables us to use Fig. 5b to understand the mechanism of divergence. Individuals in branch A have a switch size $u_A > u^*$. Figure 5b shows that, for individuals with a length ($x$) larger than the switch size $u^*$, the possible

![Fig. 8. A realization of a stochastic, individual-based implementation of our model. The population started out as a monomorphic specialist in niche 2 with $u = 0.2$ and first evolves towards the generalist strategy $u^*$ ($u^* = 0.683$ predicted by the deterministic model; Fig. 5b). This singular point is a branching point. After branching, the two branches (denoted A and B) in the dimorphic population evolve towards the two specialist strategies, specializing on prey 1 (branch A) and prey 2 (branch B), respectively. Parameters as in Fig. 2b ($p = 2$, $q_1 = 2$, $p_1 = 1$, $a_1 = a_2 = 1$, $h_1 = h_2 = 10$, $k = 30$, Table 1). Mutation probability = 0.1, mutation distribution standard deviation = 0.003. Unit of time axis is $\mu^{-1} = 10$ time units.](image-url)
intake rate is higher in the first niche than in the second. Therefore, mutants with a strategy $u' > u_A$ profit more from the first niche than A-type residents and can hence invade. Mutants with a strategy $u^* < u' < u_A$ suffer from their earlier switch to the less profitable niche and thus do not invade. In branch B, the situation is similar. For small individuals ($x < u^*$), the second niche is more profitable than the first one. Hence, mutants that switch earlier than B-type residents can invade the system, whereas mutants with a strategy $u_B < u' < u^*$ suffer from a diminished intake rate. In summary, the whole range of mutant trait values in between the two resident types ($u' = u_B \ldots u_A$) have a lower fitness than both residents. Only mutants outside this interval can invade, resulting in the divergence of branches A and B.

The results from the polymorphic, stochastic model were complemented by an analysis of an extension of our deterministic model that allows for dimorphism in the switch size of the predator population. This model predicts that, after branching, the two branches continue to diverge from each other at a decelerating rate (results not shown). The analysis also confirms that the prey densities remain approximately constant after branching. Further branching is not predicted by this model; in general, in a two-dimensional environment (resulting from the density of the predator population being regulated through two prey types at equilibrium), more than two branches are not expected (Metz et al., 1996b; Meszéna and Metz, 1999). We can therefore conclude that Fig. 8 illustrates a typical scenario where a specialist first ‘invades’ the unexploited niche, then evolves towards the generalist strategy $u^*$, whereupon the population branches into two specialists.

DISCUSSION

Our results show that the presence of an ontogenetic niche shift in an organism’s life history may give rise to evolutionary branching. The size scaling of foraging capacity in the two niches determines whether the predicted outcome of evolution is a monomorphic, ontogenetic generalist or a resource polymorphism with two ‘morphs’ specializing on one of two niches. A generalist is expected if the possible intake rate increases slower with body size in the first niche than in the second one (case a in Fig. 3a and Fig. 5a). In contrast, the evolutionary emergence of two specialists is predicted if the possible intake rate increases faster with body size in the first niche than in the second one (case b in Fig. 3b and Fig. 5b).

Mechanisms of evolutionary branching

Previous studies of ontogenetic niche shifts have mainly focused on the question when to make the transition between niches, given certain environmental conditions in terms of growth rates and mortality risks in two habitats (Werner and Gilliam, 1984; Werner and Hall, 1988; Persson and Greenberg, 1990; Leonardsson, 1991). With such an approach, one is unlikely to predict disruptive selection, because the environmental conditions that result in disruptive selection are rather special. Previous studies did not include the ecological feedback loop in their analysis. They considered the effect of the environment on individual life histories but neglected the effect of the size-structured population on the environment. In this study, we have shown that, through the effect of the ontogenetic niche shift on prey densities, evolution of the size at ontogenetic niche shift converges towards a generalist strategy that exploits both niches equally ($u^*$). This result is important, because only the environmental conditions associated with $u^*$ have the potential to result in disruptive selection and, consequently, in evolutionary branching. Hence, despite the environmental
conditions for disruptive selection being rather special, it turns out that they are likely to arise because they correspond to an evolutionary attractor of the adaptive process.

Regarding the ecological mechanisms that drive evolution, our results show a clear dichotomy between two phases of evolution. As long as a monomorphic predator population consumes one prey type disproportionately, one niche is overexploited while the other remains underexploited. Mutants that utilize the unexploited prey more thoroughly can invade the system. As the predator’s strategy evolves towards the generalist strategy $u^*$, the two niches become more and more equally exploited, and the selection gradient becomes weaker. Hence, during the initial, monomorphic phase, it is the environmental feedback that drives evolution towards the generalist strategy $u^*$. This process does not depend qualitatively on the size scaling of the functional response in the two niches.

In the second phase, after the population has reached the generalist strategy $u^*$, the size scaling of foraging rates determines the evolutionary stability of $u^*$ (e.g. equation 18). If $u^*$ is a continuously stable strategy (CSS; Fig. 3a), the resident population remains a monomorphic generalist. In contrast, if $u^*$ is an evolutionary branching point (EBP; Fig. 3b), the resident population splits into two branches. In each branch, more specialized mutants can invade and replace the resident and hence the two branches diverge (Fig. 8). Why more specialized mutants can invade is explained by essentially the same mechanism as why $u^*$ is an evolutionary branching point (cf. Fig. 5b). Crucial to the mechanism is that, given the ambient prey densities, the first niche is less profitable than the second one to individuals with a size smaller than $u^*$, and the second niche is less profitable than the first one to individuals with a size larger than $u^*$. In other words, individuals with the strategy $u^*$ are in the least profitable niche at all sizes, whereas strategies that are different from $u^*$ spent at least part of their lives in the most profitable niche. It is important to note that the difference in profitability of the two niches results from the size scaling of the functional response. Hence, in the second phase, the driving force of evolution relates critically to size structure. However, the ecological feedback and the resultant frequency-dependent selection remain important. If, for example, branch A were removed from the lake, branch B would evolve back to $u^*$.

As summarized above, we have found an ecological mechanism for evolutionary branching that is inherently size dependent. One way to show that size structure is essential to evolutionary branching is to show that it cannot occur in an analogous, unstructured model. If we just consider the fraction of lifetime that individuals spend in each niche and ignore all other aspects of the population size structure, we can formulate an unstructured analogue of our model. Analysis of such a model indicates that the environmental feedback drives evolution to a generalist strategy, analogous to the strategy $u^*$ in the size-structured model (D. Claessen, unpublished results). With a linear functional response, this singular point is evolutionarily neutral (such as Fig. 3c). The reason is that, in the ecological equilibrium of this strategy, the two niches are equally profitable. By definition, if the niches are equally profitable, it does not matter which fraction of time individuals spend in each niche. With a Holling type II functional response, the evolutionary attractor can be either neutral or a CSS. Thus, in the simplest unstructured analogue of our model, evolutionary branching is not possible.

It should be noted, however, that evolutionary branching is possible in unstructured models of consumer–resource interactions with multiple resources. It can occur if there is a strong trade-off between foraging rates on different prey types (Egas, 2002). The essence of a strong trade-off is that, given prey densities, a generalist has a lower total intake rate
and hence lower fitness than more specialized strategies (Wilson and Yoshimura, 1994). With a weak trade-off, generalists have a higher intake rate than more specialized strategies and branching is not expected. In the unstructured analogue of our model, the time budget argument (i.e. defining the evolutionary trait as merely the fraction of lifetime spent in either niche) does not lead to a strong trade-off. For example, with a linear functional response, the trade-off is perfectly neutral because the actual food intake rate is merely a weighted average of the possible intake rates in the two niches. To obtain a strong trade-off, additional assumptions have to be made. It has been suggested that trade-offs may result from physiological or behavioural specialization (Schluter, 1995; Hjelm et al., 2000; Egas, 2002). An example that is particularly relevant to this article is the possibility that learning or phenotypic plasticity produces a positive correlation between the foraging efficiency in a niche and the total time spent in that niche (e.g. Schluter, 1995). If such a correlation exists, generalists are at a disadvantage because they have less time to learn or to adapt to a specific food type. With this additional mechanism, branching may be expected even without size structure (D. Claessen, unpublished results).

The comparison with unstructured population models suggests that, on a phenomenological level, a strong trade-off emerges from our assumptions about the ontogenetic niche shift: the generalist $u^*$ has a lower fitness than more specialized strategies. Our mechanistic modelling approach allows us to identify aspects of the underlying biology that are responsible for the strong trade-off. Critical to the mechanism of evolutionary branching in our model is the constraint of the order of niche use; individuals utilize the first niche before the ontogenetic niche shift and the second one after the niche shift. We assume that the order of niches is fixed by morphological development and physiological limitations. Evidence for such constraints includes, for example, that gape limitation prevents newborn perch to consume macroinvertebrates, whereas very large perch (longer than 20 cm) are not able to capture zooplankton prey, which has been attributed to insufficient visual acuity (Byström and Garcia-Berthou, 1999). Without the fixed order of niches, an individual would optimize its performance by always being in the niche that gives the highest possible intake rate, switching at the intersection point. As an example, consider a resident as depicted in Fig. 5b (i.e. an EBP) and a mutant that reverses the order of the ontogenetic niches, but still switches at length $u$. In this situation, the mutant can invade because its intake rate is higher than that of the resident at all sizes. When this mutant reaches fixation, we effectively obtain the situation as depicted in Fig. 5a. With this new order of ontogenetic niches, evolutionary branching is not expected. If the order of niches is also an evolutionary trait, as well as the switch size $u$, it is likely that the only possible evolutionary outcome is a monomorphic generalist (cf. the CSS in Fig. 5a). Thus, the constraint of the order of niches appears to be an essential element of our hypothesis that an ontogenetic niche shift can result in evolutionary branching.

**Assumptions revisited**

Several assumptions in our model are not very realistic and relaxing these may have important consequences for the predictions made. First, we assume that reproduction is clonal, which for all fish systems is unrealistic. In a randomly mating sexual population, the continual creation of hybrids may prevent evolutionary branching to occur. Yet, the study by Dieckmann and Doebeli (1999) shows that evolution itself may solve this problem, since once the population has evolved to the evolutionary branching point, natural selection
favours assortative mating (see also Geritz and Kisdi, 2000). Even if assortative mating is based on a character other than the ecological trait that has converged to the branching point, after a correlation between the ecological trait and the separate mating trait has been established, the population branches after all. Thus, we expect our results to be robust to the introduction of sexual reproduction in systems where assortative mating may arise. An example of the development of a correlation between ecological type and mating type (based on coloration) is the Midas cichlid (*Cichlasoma citrinellum*) (Meyer, 1990). Interestingly, the resource polymorphism in this species is associated with an ontogenetic niche shift. Wilson *et al.* (2000) argue that sexual selection through colour-based assortative mating is the primary reason for the polymorphism in this species. However, the appearance of colour-based assortative mating can also be a consequence of disruptive selection caused by ecological mechanisms such as described in this article. Such ecological differentiation might in fact be essential to ensure the sustained co-existence of colour morphs.

Second, we have assumed that individuals are born mature, which obviously is not the case in fish species. The presence of a juvenile period in size-structured populations can result in population cycles (Gurney and Nisbet, 1985; Persson *et al.*, 1998). The effect of non-equilibrium dynamics on evolution in our model remains to be investigated. A preliminary analysis shows that a sufficiently large maturation size threshold (> 10.5 cm) induces generation cycles. Less expected, however, is the result that for smaller values of the maturation size threshold, the juvenile delay introduces bistability through a cusp bifurcation (D. Claessen, unpublished results). Interestingly, the bistability gives rise to evolutionary cycling, in which the system never reaches the singular point \(u^*\). Thus, a juvenile delay may drastically change the evolutionary outcome. These issues provide interesting questions for future research. It is encouraging, however, that with a sufficiently small value of the maturation size threshold (< 2 cm), our results remain unaffected, which shows that they are robust to incorporating a juvenile delay, at least as long as this does not give rise to population cycles or bistability.

Third, a basic assumption in our analysis is a niche- and size-independent mortality rate. Previous work on ontogenetic niche shifts (e.g. Werner and Hall, 1988) has often considered habitat choice within a trade-off between habitat-specific growth rates and mortality risks. Moreover, there is good evidence that, in many fish populations, mortality is inherently size-dependent, even if we disregard the effect of habitat. Important causes of such size dependence are overwintering mortality and size-dependent vulnerability to predation (Sogard, 1997). It is easy to incorporate niche- or size-dependent mortality into our model, but adding such realism comes at the cost of a clear interpretation. Preliminary analysis of a model that includes niche-dependent mortality shows that the same types of predictions are possible regarding the evolutionary outcomes (results not shown). However, the conditions and mechanisms underlying these predictions (cf. equations 18 and 22) are much less transparent. Instead of comparing possible intake rates at the switch size, as we did in this article, one must then compare the contributions to fitness over entire size intervals. Thus, for systems in which differences in niche-dependent mortality are large, conditions (18) and (22) should be regarded as approximations.

For the issue of size-dependent mortality, it is useful to distinguish between two general scenarios; depending on whether mortality rate (i) decreases or (ii) increases with body size. We argue that size-dependent mortality is likely to lead to qualitatively different results in scenario (ii) only. Underlying the results reported here is that fitness increases with the food intake rate at any given size in our model. The validity of this assumption may
break down if mortality rate increases with body size, since an increased food intake rate eventually leads to a higher mortality rate. In scenario (i), this assumption is not violated, since a higher growth rate improves future survival. Although the risk of predation is not necessarily a monotonic function of prey body size (Lundvall et al., 1999), a general pattern in teleost fishes is that mortality decreases with body size (Sogard, 1997). We therefore argue that incorporating a realistic size-dependent mortality rate will not alter our results qualitatively.

The scope for empirical testing

With experimental data on size scaling of foraging rates, we can make predictions about whether or not evolutionary branching should be expected. For several reasons, freshwater fish populations are interesting test cases for the ideas developed in this article. The life history of freshwater fish species is often characterized by one or more ontogenetic niche shifts (Werner and Gilliam, 1984). Resource polymorphisms in several lake fish species have been suggested to represent early stages of speciation (Meyer, 1990; Smith and Skúlason, 1996). Most resource polymorphisms in lake-dwelling fish species involve a benthic morph and a pelagic morph (Robinson and Wilson, 1994). In Arctic char (Salvelinus alpinus; Snorrason et al., 1994; Smith and Skúlason, 1996) and sticklebacks (Gasterosteus aculeatus; Schluter, 1996; Rundle et al., 2000), empirical evidence suggests that evolutionary branching, giving rise to a benthic morph and a pelagic morph, has occurred several times independently.

Unfortunately, the number of species for which sufficient data on size scaling of foraging rates is available is still limited. Most detailed data exist for Eurasian perch (Byström and Garcia-Berthou, 1999; Wahlström et al., 2000), roach (Rutilus rutilus; Persson et al., 1998; Hjelm et al., 2000) and bluegill sunfish (Lepomis macrochirus; Mittelbach, 1981). For perch and roach, the handling times can be assumed to be independent of prey type (Claessen et al., 2000), such that scenario 1 (pp. 205–206) applies. Before the ontogenetic niche shift, perch and roach feed on zooplankton in the pelagic habitat; after the shift, they feed on macroinvertebrates in the littoral zone. For small individuals of both species, the attack rate on zooplankton scales approximately with body surface area (i.e. $q_1 = 2$). For larger individuals, the attack rate on macroinvertebrates scales roughly with length in perch (i.e. $q_2 = 1$; Persson and Greenberg, 1990) and is nearly constant in roach (i.e. $q_2 = 0.05$; J. Hjelm, personal communication). Bluegill sunfish switch from the littoral vegetation zone to the pelagic habitat at a length between 50 and 90 mm (Werner and Hall, 1988). In the former habitat they feed on macroinvertebrates and in the latter on zooplankton. Using data on the size scaling of encounter rates with prey from Mittelbach (1981), we arrive at estimates of $q_1 = 0.5$ and $q_2 = 2$ for bluegill.

This short inquiry of available data shows that there is at least the possibility of testing the results of our evolutionary analysis with empirical data. Although it is tempting to compare these data with conditions (18), we stress that, in spite of our model’s complexity, it is still rather strategic. Rather than being designed for a specific ecological system, it is designed to test the effect of a specific mechanism. To keep it tractable, we have based our model on several simplifying assumptions, such as the absence of sexual reproduction, the absence of a juvenile delay and population dynamic equilibrium. We believe that a thorough empirical test of our model predictions would require either (a) an extension of our model, tailored specifically for a particular experimental set-up, or (b) data on a larger number
of species than are presently available, which would permit the emergence of general patterns.

Concerning point (a), obvious extensions of our model include sexual reproduction, size-dependent mortality and a juvenile period (see pp. 212–214). For example, this will have to show whether it matters that individuals mature before or after the ontogenetic niche shift. With regard to point (b), it should be noted that comparison of our conditions (equations 18 and 22) with empirical data ideally requires estimates of the possible intake rates. The size scalings of actual attack rates as presented above must be interpreted as fairly crude approximations, since they are confounded by the effect of the ontogenetic niche shift of the species. In Eurasian perch, for example, the relation between the attack rate on zooplankton and perch body size is dome-shaped. In our interpretation (and in our model; see equation 1), the attack rate on zooplankton declines at large body size because of morphological adaptation to an ontogenetic niche shift to benthivory (Hjelm et al., 2000). One will have to make assumptions to filter out the effect of the ontogenetic niche shift on the actual attack rate function to arrive at an estimate of the possible attack rate function. Yet, in the case of a species pair which has diverged into specialists, the actual intake rate of a specialist in its preferred niche can be assumed to be a fair approximation of the possible intake rate. Candidate systems include Arctic char (Jonsson and Jonsson, 2001), sticklebacks (Schluter, 1996) and cichlids (Meyer, 1990; Schliewen et al., 1994). Measurements of the size scaling of foraging rates in such systems would provide material for a critical test of our hypothesis.

ACKNOWLEDGEMENTS

This research was inspired by many discussions with Jens Andersson, Jocke Hjelm, Lennart Persson and Rickard Svanbäck, in Umeå. Most of the research reported here was done during the Young Scientist Summer Program (YSSP) in the Adaptive Dynamics Network (ADN) at the International Institute for Applied Systems Analysis (IIASA), Laxenburg, for which D.C. received a grant from the Dutch Science Foundation (NWO). During the YSSP, discussions with the other ADN students (Sondre Aanes, Fabio Dercole, Juan Keymer, Rahel Luethy, Christian Magori and Kalle Parvinen) were very stimulating.

REFERENCES


Doctoral dissertation, University of Amsterdam.


Stabilization of Population Fluctuations due to Cannibalism Promotes Resource Polymorphism in Fish

Jens Andersson, Pär Byström, David Claessen, Lennart Persson, and Andre M. De Roos

1. Department of Ecology and Environmental Science, Umeå University, SE-901 87 Umeå, Sweden;
2. Department of Aquaculture, Swedish University of Agricultural Sciences, SE-901 87 Umeå, Sweden;
3. Institute for Biodiversity and Ecosystem Dynamics, University of Amsterdam, P.O. Box 94084, NL-1090 GB Amsterdam, The Netherlands;
4. Ecology Laboratory, University of Paris 6, 7 quai Saint Bernard, 75252 Paris Cedex 05, France

Submitted February 2, 2006; Accepted January 2, 2007; Electronically published April 10, 2007

Abstract: Resource polymorphism is a well-known phenomenon in many taxa, assumed to be a consequence of strong competition for resources and to be facilitated by stable environments and the presence of several profitable resources on which to specialize. In fish, resource polymorphism, in the form of planktivore-benthivore pairs, is found in a number of species. We gathered literature data on life-history characteristics and population dynamics for 15 fish species and investigated factors related to the presence of such resource polymorphism. This investigation indicated that early cannibalism and low overall population variability are typically associated with the presence of resource polymorphism. These findings match previously reported patterns of population dynamics for size-structured fish populations, whereby early cannibalism has been shown to decrease temporal variation in population dynamics and to equalize the profitability of the zooplankton and macroinvertebrate resources. Our study suggests that competition alone is not a sufficient condition for the development of resource polymorphism. These findings indicate that early cannibalism is a fundamental factor in dampening population oscillations and possibly by equalizing the profitability of different resources.

Keywords: resource polymorphism, population dynamics, size structure, fish, cannibalism.

Resource polymorphism, defined as the occurrence of different morphotypes within a single population using different resources, has been found in many different taxa (Collins and Cheek 1983; Smith 1987; Thompson 1992; McPhail 1993; Padilla 2001) and may constitute and provide an example of ongoing evolution (Schluter 2000), possibly associated with ecological selection (Skulason and Smith 1995). Resource polymorphism is assumed to develop in response to severe competition for a certain resource because adaptation to alternative resources under such circumstances increases the fitness for individuals (Schluter 2000). Morphological differences between groups using different resources have been shown to depend on phenotypic plasticity as well as genetic divergence (Nordeng 1983; Gíslason et al. 1999), and the two factors have also been suggested to act together (Adams et al. 2003). Although many studies of resource polymorphism exist, few discuss its development in a broader ecological context. It has been suggested that the development of resource polymorphism is favored in environments that are temporally stable with respect to both abiotic and biotic factors (Jo´ nasson et al. 1998; Egas et al. 2004; Snorrason and Skulason 2004) and by the occurrence of several niches of relatively high productivity capable of supporting different specialists (the habitat heterogeneity argument; Lavin and McPhail 1986; Walker 1997; Grant 2001). A further important factor is community composition, because new morphs are expected to be able to develop only in the presence of open (i.e., unoccupied) niches, as a result of the absence of one or several functional species (Schluter and McPhail 1992; Robinson et al. 1993; Bohn and Amundsen 2001). Finally, predation may also facilitate resource polymorphism, either by strengthening trade-offs between different strategies (Andersson 2003; Andersson...
and Persson 2005) or by decreasing the effects of resource population feedbacks (Rundle et al. 2003).

A large number of studies on resource polymorphism have been performed on fish (Svardson 1979; Ehlinger and Wilson 1988; Sandlund et al. 1992; Schluter and McPhail 1992; Robinson et al. 2000). These studies include many different types of specializations, but a frequently reported type of resource polymorphism concerns benthic-limnetic (benthivorous-planktivorous) pairs (Robinson and Wilson 1994; Smith and Skúlason 1996). However, studies have hitherto mainly restricted themselves to morphological differences between morphotypes per se; relationships among diet, morphology, and performance; and trade-offs as a result of morphology (Ehlinger 1990; Malmquist 1992; Malmquist et al. 1992; Schluter and McPhail 1992; Schluter 1995; Robinson et al. 1996; Svärdson 1998; Svanbäck and Eklöv 2003).

We investigated the hypothesis that stable environments promote the development of resource polymorphism and relate this to results from work on the effect of density-dependent life histories on population dynamics in size-structured populations. We propose that cannibalism is a candidate ecological factor that facilitates resource polymorphism. Previous work has shown that the population dynamics of consumer-resource systems are characterized by large-amplitude cycles if the consumer is size structured and lacking cannibalism (Hamrin and Persson 1986; Persson et al. 1998; Sanderson et al. 1999; Lammens et al. 2002). Large-amplitude cycles in resources imply that specialization on a certain resource is associated with long periods of very low resource levels, forcing specialists to change their diet in order to survive and hence promoting the existence of generalists. The effect of cannibalism on population stability depends on several aspects, but, in general, strong cannibalism that targets newborn individuals stabilizes both consumer and resource dynamics (Claessen et al. 2000, 2002). Another effect of cannibalism is that resources consumed by cannibalized size classes are released from predation and thereby increase in relative availability (Persson et al. 2004a). Hence, cannibalism can equalize or differentiate the relative profitability of different resources, allowing for specialization on resources previously exploited by smaller size classes.

To test our hypothesis that a decrease in temporal variability as a result of cannibalism promotes the development of resource polymorphism, we performed a literature survey that considered species-specific foraging traits and population dynamics. Our literature survey included ecological characteristics in fish species in which resource polymorphism is known as well as those for which it is not. On the basis of factors typically proposed to facilitate resource polymorphism and the ecological relationships previously described, we predicted that species displaying resource polymorphism should be characterized by cannibalism in early life stages, resulting in low temporal variation in population densities and possibly also equalization of resource levels.

**Material and Methods**

We used three criteria to select species for inclusion in our review of resource polymorphism in fish. First, we considered only resource polymorphism with regard to lake-dwelling planktivore-benthivore pairs, to be able to look at a set of parallel examples of resource polymorphism and to exploit the large body of information on resource use and foraging ability that already exists for such species. Second, species were included only for which there were extensive data on dietary shifts at different sizes. Finally, data on population size over time were required so that the presence/absence of resource polymorphism could be related to differences in population dynamics.

We gathered data for the following seven variables. (i) Maximum size for planktivory: the maximum body size when zooplankton can still be the main resource. (ii) Benthivory potential: confirmative data showing that the species uses benthic resources during ontogeny and hence that the species could potentially develop a benthivorous morph. (iii) Minimum size for piscivory: the smallest body size at which the species starts to include fish in its diet (inter- or intraspecific prey). (iv) Minimum victim-cannibal size ratio: the minimum body length ratio between victims and cannibals reported. In cases where the relationship could not be found, we used the lower size relation between the predator species and other prey fishes. If the species was cannibalizing on eggs or newly hatched larvae, the ratio was set to 0. (v) Maximum fish size: reported maximum body size in natural systems. (vi) Population abundance variation: the coefficient of variation in numbers of individuals 1 year old and older. We searched for data from relatively undisturbed environments and for surveys lasting at least 4 years. In some studies, we could not find actual numbers of individuals but only catch per unit effort (CPUE) data. In these cases, CPUE values for different years were used to compute the coefficient of variation. (vii) Resource polymorphism: to be classified as displaying resource polymorphism, we had to find at least five lakes from which different morphotypes have been reported to coexist.

We first performed a principal component analysis (PCA) on the data from the literature survey in which the following factors were included. (1) The ratio between maximum size and maximum size for planktivory: we use this ratio as an estimate of the importance of planktivory over the ontogeny (a low value close to 1 indicates that planktivory is important). (2) The ratio between maximum size and minimum size for piscivory: a high value...
indicates that piscivory is important. For nonpiscivorous species, this ratio was set to 1. (3) The maximum fish size. (4) The minimum victim-cannibal size ratio. For species with no cannibalism, this ratio was set to 1. (5) The coefficient of variation for population abundance. To homogenize the data, the ratios between (i) maximum size and maximum size for planktivory and (ii) maximum size and minimum size for piscivory and maximum size were \( \ln(x + 1) \) transformed. Data on the cannibalistic lower limit were arcsine transformed before conducting the PCA analysis. We could not find any data for the minimum size for piscivory in pumpkinseed (\textit{Lepomis gibbosus}); therefore, we used the same value as for bluegill (\textit{Lepomis macrochirus}) because of their close taxonomic relationship. In order to test our hypothesis that cannibalism, especially when it targets smaller individuals, stabilizes population dynamics, we performed a one-sided test using linear regression for the coefficient of variation in number of individuals on the minimum victim-cannibal size ratio. Thereafter, to further explore the effects of the different variables on the presence of resource polymorphism, and because of the small sample size, we performed a binary logistic regression with resource polymorphism as the dependent factor (present/not present) and the two principal components produced in the PCA analysis as independent. With respect to our hypothesis that variation in population size and a small minimum victim-cannibal size ratio promote resource polymorphism, the B coefficient (analogous to the slope in a linear regression) for the binary logistic regression should be negative when testing resource polymorphism and principal component (PC) 2.

Two possible caveats in our analysis of the literature data should be mentioned. First, no concern was given to phylogeny and past evolutionary history in our analyses because the aim of our article was not to unravel taxonomic relationships but to look for functional traits that may also co-vary within families. However, it is worth noting that for each of the species displaying resource polymorphism, there is almost always another species within the same family not displaying resource polymorphism, the only exception being threespine sticklebacks (\textit{Gasterosteus gasterosteus}). Another potential weakness in our analyses is that it is not always a simple task to conclude that a species does not display resource polymorphism. A species classified as nonpolymorphic may simply constitute a less thoroughly investigated taxon that may be classified as polymorphic in the future. However, this is unlikely to be a major problem in our data set because almost all species that we classified as polymorphic display quite dramatic differences, with their polymorphic nature first having been observed visually before undertaking morphometric analysis, with the exception of bluegill. Thus, we argue that even if polymorphism will be found in other species in the future, these species would already have been found if the morphological differences had been as large as for the species that we classified as polymorphic.

Results

Data used in the literature survey are summarized in table 1. Seventy-nine percent of total variation was accounted for by the first two PC axes (fig. 1; table 2). PCI was predominantly associated with the ratio of maximum fish size to maximum size for planktivory, the ratio of maximum size to minimum size for piscivory, and maximum size per se. PC2 reflected the minimum victim-cannibal size ratio and the coefficient of variation in population abundance (table 2). Four groups could be discerned in the PC1-PC2 plane (fig. 1). First, all species that lack evidence of cannibalism (i.e., bream [\textit{Abramis abramis}], vendace [\textit{Coregonus albula}], and roach [\textit{Rutilus rutilus}]) occur in the upper left corner of the PC plot (group A; fig. 1). These species have not been reported to display resource polymorphism but are characterized by high-amplitude population cycles, driven by intercohort competition dominated by recruiters (Hamrin and Persson 1986; Perrow et al. 1990; Lammens et al. 2002; table 1). Yellow perch (\textit{Perca flavescens}) was also associated with this group of species, reflecting common occurrence of intercohort competition-driven cycles and the lack of resource polymorphism (Sanderson et al. 1999; Persson et al. 2004b). Remaining species clusters are all characterized by cannibalism, but the extent of polymorphism differs substantially between the groups. The group B species (largemouth bass [\textit{Micopterus salmoides}], northern pike [\textit{Esox lucius}], and pikeperch [\textit{Sander lucioperca}]) are found to the far right in figure 1, reflecting a large maximum fish size, a low ratio of maximum fish size to minimum size for piscivory, and a low ratio of maximum fish size to maximum size for planktivory. Generally, these species have been classified as obligatory piscivores characterized by a short planktivorous life stage and an early shift to piscivory (Keast 1985; Mittelbach and Persson 1998), which is in accordance with the results of our PCA. Of the remaining species, those in group D (Arctic char, bluegill, pumpkinseed, threespine stickleback, and whitefish [\textit{Coregonus lavaretus}]) all display resource polymorphism (fig. 1). This group is characterized by relatively low population variability and a low value for the minimum victim-cannibal size ratio (all species in group D are reported to cannibalize on egg and larvae). Finally, group C (brown trout [\textit{Salmo trutta}], Eurasian perch [\textit{Perca fluviatilis}], and lake trout [\textit{Salvelinus namaycush}]) represents a quite heterogeneous group. Eurasian perch displays relatively high population variability and a relatively high minimum victim-cannibal size ratio, placing it closer to group A. In contrast, brown
<table>
<thead>
<tr>
<th>Species</th>
<th>Maximum size for planktivory (g)</th>
<th>Benthivory</th>
<th>Minimum size for piscivory (g)</th>
<th>Minimum victim-cannibal size ratio</th>
<th>Maximum size (g)</th>
<th>Coefficient of variation for population size</th>
<th>Resource polymorphism</th>
</tr>
</thead>
<tbody>
<tr>
<td>No cannibalism:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abramis brama</td>
<td>275^2</td>
<td>Yes^2</td>
<td>...</td>
<td>...</td>
<td>6,300</td>
<td>&gt;1^4</td>
<td>...</td>
</tr>
<tr>
<td>Coregonus albula</td>
<td>125^4</td>
<td>Yes^4</td>
<td>...</td>
<td>...</td>
<td>1,000</td>
<td>.86^6</td>
<td>...</td>
</tr>
<tr>
<td>Rutilus rutilus</td>
<td>40^7</td>
<td>Yes^7</td>
<td>...</td>
<td>...</td>
<td>2,500</td>
<td>.59^9</td>
<td>...</td>
</tr>
<tr>
<td>Cannibalism:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Piscivores:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Esox lucius</td>
<td>.49^9</td>
<td>Yes^9</td>
<td>1.0^9</td>
<td>.03^19</td>
<td>28,400</td>
<td>.40^11,a</td>
<td>...</td>
</tr>
<tr>
<td>Micropterus salmoides</td>
<td>.25^12</td>
<td>Yes^12</td>
<td>.5^12</td>
<td>.05^14,a</td>
<td>10,100</td>
<td>.44^13</td>
<td>...</td>
</tr>
<tr>
<td>Stizostedion lucioperca</td>
<td>1.0^17</td>
<td>Yes^17</td>
<td>2.0^17</td>
<td>.07^18,a</td>
<td>20,000</td>
<td>.94^19</td>
<td>...</td>
</tr>
<tr>
<td>Omnivores:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coregonus lavaretus</td>
<td>500^20</td>
<td>Yes^20</td>
<td>500^20</td>
<td>0^31</td>
<td>10,000</td>
<td>.21^21</td>
<td>Yes^28</td>
</tr>
<tr>
<td>Gasterosteus aculeatus</td>
<td>10^3</td>
<td>Yes^3</td>
<td>.8^3</td>
<td>0^15</td>
<td>10</td>
<td>.43^27</td>
<td>Yes^27</td>
</tr>
<tr>
<td>Lepomis gibbosus</td>
<td>35^4</td>
<td>Yes^4</td>
<td>...</td>
<td>0^3</td>
<td>6.80</td>
<td>.43^27</td>
<td>Yes^27</td>
</tr>
<tr>
<td>Lepomis macrochirus</td>
<td>50^5</td>
<td>Yes^5</td>
<td>40^5</td>
<td>0^6</td>
<td>2,150</td>
<td>.56^34</td>
<td>Yes^34</td>
</tr>
<tr>
<td>Perca fluviatilis</td>
<td>20^9</td>
<td>Yes^9</td>
<td>35^9</td>
<td>.08^13</td>
<td>1,910</td>
<td>.80^13</td>
<td>...</td>
</tr>
<tr>
<td>Perca flavescens</td>
<td>35^9</td>
<td>Yes^9</td>
<td>40^9</td>
<td>.05^13</td>
<td>4,750</td>
<td>.62^13</td>
<td>...</td>
</tr>
<tr>
<td>Salmo trutta</td>
<td>300^30</td>
<td>Yes^30</td>
<td>35^30</td>
<td>.07^17</td>
<td>20,000</td>
<td>.23^17</td>
<td>...</td>
</tr>
<tr>
<td>Salvelinus alpinus</td>
<td>200^20</td>
<td>Yes^20</td>
<td>40^20</td>
<td>0^15</td>
<td>15,000</td>
<td>.25^15</td>
<td>Yes^15</td>
</tr>
<tr>
<td>Salvelinus namaycush</td>
<td>245^31</td>
<td>Yes^31</td>
<td>5.4^31</td>
<td>.08^14,a</td>
<td>32,700</td>
<td>.15^14</td>
<td>...</td>
</tr>
</tbody>
</table>

Note: Maximum size for planktivory, minimum size for piscivory, and maximum size reported as weight. Victim-cannibal size ratio reported as the minimum size of the victim in percentage of the cannibal with respect to length. Coefficient of variation is for the density over time. The presence of benthivory and resource polymorphism reported only as confirmative data. A more detailed description of the different categories is given in "Material and Methods.”


Additional information given personally by the authors.
The American Naturalist

Figure 1: Two first principal components of the principal component analysis on the literature data. Filled circles show species displaying resource polymorphism, and open circles show species not displaying resource polymorphism. Four groups are recognized: A, high-amplitude dynamics and noncannibalistic/low-degree cannibalistic species; B, true piscivore species; C, cannibalistic but not egg/larvae cannibalistic species; D, egg/larvae cannibalistic species. The species are bream (br), vendace (ve), roach (ro), northern pike (Np), largemouth bass (lb), pikeperch (pp), whitefish (wh), threespine stickleback (ts), pumpkinseed (pu), bluegill (bg), yellow perch (yp), Eurasian perch (Ep), brown trout (bt), Arctic char (Ac), and lake trout (lt).

trout and lake trout display relatively stable dynamics but because of larger maximum fish size and minimum size for piscivory are more associated with group B (fig. 1). A higher minimum victim-cannibal size ratio was positively related to high population variability (linear regression, slope = 0.253, $F = 6.0, df = 1, 13, P = .015$). Finally, we found that PC1, mainly described by maximum size for planktivory, minimum size for piscivory, and maximum size, could not explain resource polymorphism (Wald coefficient = 2.3; $P > .1$). In contrast, PC2, mainly described by population variation and minimum victim-cannibal size ratio, relates the presence of resource polymorphism to small population variations and small minimum victim-cannibal size ratios ($B = -1.66$; Wald coefficient = 3.87; $P = .025$).

Discussion

Population Variability and Resource Polymorphism

Resource polymorphism did not occur in any noncannibalistic species in our analysis. Noncannibalistic fish populations and their resources have been shown previously to exhibit high-amplitude cycles driven by cohort competition (Hamrin and Persson 1986; Perrow et al. 1990; Lammens et al. 2002). This is also true for species with low cannibalistic voracity, such as yellow perch (Sanderson et al. 1999; Persson et al. 2004b). On the basis of these results, we propose that noncannibalistic species do not display resource polymorphism due to the large temporal variation in population densities leading to periods during which conditions are unfavorable for specialist strategies because of low resource availability. Our finding of the absence of resource polymorphism in noncannibalistic species, in association with large-amplitude population dynamics, is also in accordance with the general idea that a temporally unstable environment reduces the likelihood that resource polymorphism will develop (Egas et al. 2004; Snorrason and Skúlason 2004).

Although the remaining species were all cannibalistic, we found resource polymorphism only in those exhibiting early cannibalism (especially cannibalism on eggs and larvae). Theory predicts that early cannibalism in size-structured populations stabilizes population dynamics (Claessen et al. 2002), a prediction supported by the literature
data on temporal variation in numbers in species with early cannibalism relative to non- or weakly cannibalistic species. However, we also found cannibalistic species that do not display resource polymorphism despite their relatively stable population dynamics (e.g., pike, largemouth bass, and lake trout). This suggests that population stability is not a sufficient condition for the development of resource polymorphism in fish. Pike, largemouth bass, and lake trout can all grow to very large sizes. In order to do so, they become piscivores at a small size and use zooplankton resources for only a very short period early in development (Hudson et al. 1995; Mittelbach and Persson 1998; Skov and Koed 2004). We suggest that the lack of resource polymorphism involving zooplankton and benthic resources in the piscivore species in our study, despite their relatively stable population dynamics, reflects the importance of entering the piscivore niche as early as possible for an obligate piscivore’s future success. Consequently, an early shift to a fish diet is typically associated with special morphological adaptations to catch, handle, and ingest large prey (Webb 1984; Mittelbach and Persson 1998; Huskey and Turingan 2001), which reduces the scope for parallel adaptation for handling small prey, such as zooplankton.

### Relative Resource Profitability

Another effect of size-specific cannibalism is that the relative availability of resources used by the cannibalized size classes increases concomitantly with reduced consumption from these size classes (Claessen et al. 2002). Fish generally undergo one or two ontogenetic niche shifts: new hatchlings feed predominantly on zooplankton before shifting to benthic macroinvertebrates, with some species further becoming piscivorous when older (Persson and Brönmark 2002a; Persson et al. 2004a; Byström and Andersson 2005). The size-dependent nature of cannibalism has an indirect effect on the relative profitability of different resources. Cannibalism on the smallest size classes leads to an increase in the profitability of the zooplankton resource as a result of reduced consumption by small fish, whereas the benthic resource will be less affected, which results in a greater gain for larger fish using the zooplankton resource (Persson et al. 2004a). In contrast, cannibalism on intermediate size classes leads to an increase in the availability of the benthic resource, enhancing its relative profitability.

On the basis of the stronger positive effect of early cannibalism (i.e., on larvae and eggs) on zooplankton compared with benthic resources and the observed association between early cannibalism and resource polymorphism, we suggest that the availability of zooplankton is a crucial ingredient in the development of polymorphic planktivore-benthivore pairs. If this hypothesis is true, we anticipate patterns whereby exploitation of zooplankton is additional to a more fundamental dependence on benthic resources in species displaying resource polymorphism. In accordance with this hypothesis, several diet studies on benthivore-planktivore pairs indicate that complete specialization on zooplankton resources is not common, whereas benthic morphs normally are highly specialized for macroinvertebrate feeding and essentially exclude zooplankton from their diet (Malmquist et al. 1992; Schluter and McPhail 1992; Robinson et al. 1993).

### Competition and Population Stability

Hypotheses on resource polymorphism generally assume that strong competition is the crucial factor behind morphological divergence, with individuals able to escape competition by switching to an alternative resource (Schluter 2000). However, cannibalism, besides having the potential to stabilize dynamics, also has the potential to reduce competition by favoring higher overall resource densities (Claessen et al. 2002; Persson et al. 2004a).

Both empirical and modeling results clearly suggest that competition is the only regulating factor, this leads to high-amplitude dynamics (Hamrin and Persson 1986; Perrow et al. 1990; Persson et al. 1998, 2004b; Sanderson et al. 1999; Claessen et al. 2000; Lammens et al. 2002), which we suggest should also reduce the possibility for resource polymorphism to evolve. Although the stabilizing effect of early cannibalism has been shown to release competition for shared resources (Claessen et al. 2000; Persson et al. 2004a), stable (fixed-point) dynamics in competition-regulated systems without cannibalism, which would support both the stability and the competition arguments for resource polymorphism, is possible. But this is expected to occur only within a narrow range of attack rate values, a fundamentally important size-scaled parameter (Persson et al. 1998). In contrast, all existing experimental data on the size scaling of foraging rates in fish are well outside this range and point to exploitative cohort competition leading to high-amplitude cohort cycles (Mittelbach 1981; Byström and García-Berthou 1999; Hjelm and Persson

### Table 2: Factor loadings of the first two principal components (PCs) explaining 79% of the variation

<table>
<thead>
<tr>
<th>Factor</th>
<th>PC1</th>
<th>PC2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maximum size planktivory</td>
<td>.95</td>
<td>-.04</td>
</tr>
<tr>
<td>Minimum size piscivory</td>
<td>.80</td>
<td>-.45</td>
</tr>
<tr>
<td>Cannibalistic lower limit</td>
<td>-.14</td>
<td>.89</td>
</tr>
<tr>
<td>Population variation</td>
<td>-.047</td>
<td>.86</td>
</tr>
<tr>
<td>Maximum size</td>
<td>.80</td>
<td>-.007</td>
</tr>
</tbody>
</table>

H11002
Finally, interference competition favoring larger individuals could potentially stabilize dynamics at intermediate values (Persson et al., 1998), but this is an explanation not generally discussed or explored in the context of resource polymorphism, as far as we are aware.

Results for Eurasian perch provide particularly strong evidence for the idea that both predator population stability and the relative profitability of different potential prey influence the possibility for resource polymorphism to develop (Svanbäck and Persson 2004). According to both theory and empirical data, the value of the minimum victim–cannibal size ratio for perch is in a critical range that causes population dynamics to shift between periods of relatively stable cannibal-driven dynamics and periods characterized by large-amplitude population cycles driven by cohort-competition (Persson et al., 2000, 2003; Claessen et al. 2000, 2002). Diet analyses of perch over a 10-year period in relation to population dynamics show that individuals were more specialized on either resource during periods with cannibal-driven population dynamics than during periods with competition-driven dynamics (Svanbäck and Persson 2004). This fine-scaled behavioral pattern supports the hypothesis advanced above, that a control of recruitment through cannibalism has a positive effect on the likelihood of diversification into different morphotypes but that interruptions by periods with recruit dominance and severe competition impose a strong constraint on the development of a true planktivore-benthivore pair. The co-occurrence of higher resource densities (and weaker competition) and cannibalism together with the suggested relationship between cannibalism and resource polymorphism do contrast with most previous hypotheses on the evolution of resource polymorphism. However, a recent experimental study suggests that predation can facilitate diversification despite decreased competition (Rundel et al. 2003). Finally, Svanbäck and Persson (2004) also found that larger individuals consumed zooplankton to a greater extent during periods with cannibal-driven dynamics than periods with competition-driven dynamics (Svanbäck and Persson 2004). This fine-scaled behavioral pattern supports the hypothesis advanced above, that a control of recruitment through cannibalism has a positive effect on the likelihood of diversification into different morphotypes but that interruptions by periods with recruit dominance and severe competition impose a strong constraint on the development of a true planktivore-benthivore pair. The co-occurrence of higher resource densities (and weaker competition) and cannibalism together with the suggested relationship between cannibalism and resource polymorphism do contrast with most previous hypotheses on the evolution of resource polymorphism. However, a recent experimental study suggests that predation can facilitate diversification despite decreased competition (Rundel et al. 2003). Finally, Svanbäck and Persson (2004) also found that larger individuals consumed zooplankton to a greater extent during periods with cannibal-driven dynamics than periods with competition-driven dynamics, which supports the hypothesis that zooplankton is the limiting resource for the development of resource polymorphism.

**Extensions to Additional Species Configurations and Future Perspectives**

Our results have relevance for additional types of resource polymorphism. We have, for example, found studies showing that pike, lake trout, and brown trout can have benthi-vore-piscivore specialists in the same lake, which could be argued to be an effect of cannibalism and the consequent stabilization of resource dynamics (Ferguson 1986; Beaudoin et al. 1999; Henderson and Anderson 2002). In a broader context, resource polymorphism is expected to occur more frequently in populations in which recruitment is strongly regulated at an early stage (e.g., by cannibalism, interspecific predation, or limited breeding habitats) compared with other populations lacking such regulation. Our hypothesis could thus be tested by comparing populations of the same species found in different ecological settings with respect to early regulation. Overall, our study suggests that the evolution of resource polymorphism is best understood by considering population feedbacks and is hence in line with the rapidly growing theoretical framework of adaptive dynamics where evolution in ecologically dynamical systems can explicitly be explored (Dieckmann et al. 2004). In particular, a theoretical investigation into the effect of cannibalism on individual resource use and morphology in size-structured populations should yield new insights into the evolution of resource polymorphism.

**Acknowledgments**

We thank E. Lammens, C. Madenjian, and C. Pierce for sharing detailed and reanalyzed data from previously published articles and J. Fryxell, F. Johansson, B. McKie, and an anonymous reviewer for constructive comments on earlier versions of the manuscript. This study was supported by grants from the Mountain MISTRA program, the Swedish Research Council for Environment, Agricultural Science and Spatial Planning (FORMAS), and the Swedish Research Council (VR) to P.B. and L.P. and the Dutch Science Foundation (NWO) to D.C. and A.M.D.R.

**Literature Cited**


Buijse, A. D., and R. P. Houthuijzen. 1992. Piscivory, growth, and size-selective mortality of age-0 pikeperch (*Stizostedion-Lucio-
Cannibalism Promotes Resource Polymorphism


Skov, C., and A. Koed. 2004. Habitat use of 0+ year pike in exper-
Cannibalism Promotes Resource Polymorphism


Associate Editor: Claire de Mazancourt
Editor: Donald L. DeAngelis
Delayed evolutionary branching in small populations

David Claessen,1* Jens Andersson,2‡ Lennart Persson2 and André M. de Roos1

1Institute for Biodiversity and Ecosystem Dynamics, University of Amsterdam, PO Box 94084, 1090 GB Amsterdam, The Netherlands and 2Department of Ecology and Environmental Science, Umeå University, SE-90187 Umeå, Sweden

ABSTRACT

Question: How is the process of evolutionary branching influenced by demographic stochasticity?

Mathematical methods: Adaptive dynamics of (i) a simple consumer-resource model and (ii) an analogous but individual-based model with finite population size.

Key assumptions: Consumers have access to two habitats with dynamic resources. The fraction of time spent in each habitat is the evolving trait. System size influences absolute population size and hence demographic stochasticity but not the expected population densities. Reproduction is asexual.

Predictions: Absolute population size is an ecological factor that controls the outcome of evolutionary dynamics by modifying the level of demographic stochasticity. Small populations are predicted to remain monomorphic generalists while large populations are predicted to split evolutionarily into specialized sub-populations. Underlying the delayed or absent evolutionary branching in small populations are (i) random genetic drift and (ii) extinction of incipient branches due to near-neutral stability.

Keywords: adaptive dynamics, demographic stochasticity, evolutionary branching, extinction, finite population size, incipient species, random genetic drift.

INTRODUCTION

‘Adaptive dynamics’ is a theoretical framework for studying evolutionary dynamics in an ecological context. This theory asserts that evolution takes place in a dynamic fitness landscape in which fitness is the outcome of ecological interactions between individuals such as competition for food or for mates (Metz et al., 1992). Central to the theory are the classification of evolutionary scenarios based on the geometry of the invasion-fitness function (Metz et al., 1996; Geritz et al., 1998) and the so-called canonical equation of adaptive dynamics, which predicts the rate and direction of evolution (Dieckmann and Law, 1996; Champagnat et al., 2001).
A great deal of the theory, including the above-mentioned classification and canonical equation, is based on simplifying assumptions that allow for the derivation of analytical results (Metz et al., 1996). Two of these assumptions – i.e. that (i) mutations are rare and (ii) mutations have small phenotypic effect – have recently been discussed extensively in the literature (Van Dooren, 2005; Waxman and Gavrilets, 2005a, 2005b, and references therein). Here we focus on another common assumption, which is that (iii) the current population is large enough that demographic stochasticity and random genetic drift in the resident population can be ignored. Although demographic stochasticity in the mutant populations is implicitly incorporated in the canonical equation, it is ignored for the resident population. Demographic stochasticity results from discrete random events such as birth and death. In very large populations, these events have tiny effects and occur frequently enough to result in predictable and almost smooth changes of population density. The population dynamics are then well-approximated by a deterministic model. In small populations, however, the discrete events occur less frequently and moreover each event has a greater influence on the state of the population. In this case the population size and genetic composition may deviate significantly from the expectation based on a deterministic model. Natural populations are of finite size and hence subject to demographic stochasticity, and therefore the sensitivity of theoretical predictions to relaxing assumption (iii) is of great importance, especially in the context of confronting model predictions with empirical data on ecological systems. Note that natural populations are also subject to environmental stochasticity; this, however, is outside the scope of this paper.

Analytical predictions of adaptive dynamics have often been tested with individual-based simulation models in which population numbers are finite (and in which assumptions (i–iii) may be relaxed) (e.g. Dieckmann and Doebeli, 1999; van Doorn et al., 2004). The analytical predictions are usually found to hold in such simulations but a full understanding of the effect of absolute population size is still lacking.

One line of studies on evolutionary dynamics in finite populations is game-theoretic and investigates the consequences of the fact that a single mutant cannot play against itself (Riley, 1979; Schaffer, 1988). The evolutionarily stable strategy (ESS) then appears to depend on absolute population size: the smaller the population, the more spiteful the ESS (Schaffer, 1988). A second line of research addresses the effect of demographic stochasticity on evolutionary dynamics. Proulx and Day (2001) argue that the expected growth rate of a small mutant population [the standard definition of fitness in adaptive dynamics theory (Metz et al., 1992)] may not accurately predict the direction and endpoint of evolution in finite populations subject to environmental stochasticity. In the absence of demographic stochasticity, alleles with a negative expected growth rate have zero probability to reach fixation. Proulx and Day (2001) show that in a finite population they may yet have a fixation probability that is greater than that of a neutral allele. They argue that it is hence more correct to use the fixation probability of rare alleles to describe the evolutionary dynamics of small populations. Cadet et al. (2003) and Parvinen et al. (2003) study the evolution of the dispersal rate in a metapopulation model and demonstrate that accounting for finite population size in local patches alters the evolutionary prediction. They propose two explanations for the difference. First, when local populations are small, the relatedness of individuals is high, leading to kin competition. Second, demographic stochasticity results in variation in local population size such that a disperser from a non-empty patch always has a chance to find a patch with fewer competitors. Both explanations favour the evolution of a higher dispersal rate under the influence of demographic stochasticity. In conclusion, these studies show that
the direction of evolution in finite populations may differ from the expectation based on a deterministic model.

An important finding of adaptive dynamics theory is that ‘upward’ movement in a dynamic fitness landscape (i.e. resulting from directional selection) can take an evolving population towards a fitness minimum referred to as an ‘evolutionary branching point’ (Metz et al., 1992). At this point, selection turns disruptive and (depending on the mating system) the population may branch into two sympatrically diverging subpopulations (Metz et al., 1992; Dieckmann and Doebeli, 1999) or a genetic polymorphism (Kisdi and Geritz, 1999). In this paper, we focus specifically on the effect of finite population size on the dynamics of evolutionary branching. We ask the question: ‘How is the process of evolutionary branching influenced by demographic stochasticity?’ The answer to the question may be used to confront theoretical predictions with a comparative study of empirical data on a range of population sizes.

As the starting point for our study, we choose a very simple model for which evolutionary branching is predicted according to current adaptive dynamics theory. Several modelling studies have demonstrated that a consumer population exploiting two distinct food populations or habitats can evolve to an evolutionary branching point and hence potentially speciate or give rise to a genetic polymorphism (e.g. Kisdi and Geritz, 1999; Day, 2000; Claessen and Dieckmann, 2002; Schreiber and Tobiasen, 2003; Rueffler et al., 2004, 2006). Generally, the condition for evolutionary branching in such models is that the generalist strategy exploiting both resources has a lower fitness than mutant strategies with a slightly higher degree of specialization in either direction. In other words, the shape of the trade-off between the performance (contribution to fitness) in the two habitats determines the outcome of the evolutionary dynamics. When fitness is a linear combination of performance in two habitats (as is the case here), evolutionary branching is expected if the trade-off is ‘strong’ (convex), whereas an evolutionarily stable generalist is expected if the trade-off is ‘weak’ (concave) or ‘neutral’ (linear) (Rueffler et al., 2004, 2006).

We model a consumer population feeding on two resource populations that are assumed to occur in different habitats. The evolutionary trait is assumed to be the fraction of time spent in each habitat. If the functional response in each habitat does not depend directly on the trait value (but only indirectly through the effect on prey density), such time splitting amounts to a linear trade-off. The reason is that individuals cannot be in two habitats at the same time. However, if the functional response is a function of the trait value, the trade-off becomes non-linear. We assume that the habitat-specific foraging capacity increases with the time spent in the habitat. The foraging performance of individuals in a given habitat may improve with time by, for example, phenotypic plasticity or learning. Our deterministic model is very similar to that of Schreiber and Tobiasen (2003), who model the effects of different resource relations (essential, substitutable, antagonistic) and find that antagonistic resources may induce evolutionary branching. In our model, however, the resources are always substitutable and branching is caused by the trade-off in attack rates.

Our model is loosely based on the ecology of lake fish such as Arctic charr (Salvelinus alpinus), perch (Perca fluviatilis), and sticklebacks (Gasterosteus aculeatus). Such species often have access to two resources in different habitats: zooplankton in the pelagic habitat and macroinvertebrates in the benthic habitat. For a number of fish species, it has been demonstrated that diet influences individual development and morphology, resulting in increased habitat-specific foraging capacity (Robinson and Wilson, 1995; Day and McPhail, 1996; Andersson, 2003; Andersson et al., 2005). A strong trade-off results if the resources occur in different habitats.
and habitat-specific foraging ability is positively related to the amount of time spent in the habitat.

We use an individual-based model to show that absolute population size influences the probability of successful evolutionary branching. In our model, lake volume scales the total population size without affecting the ecological interactions. The only difference between small and large systems is thus the level of demographic stochasticity. By studying the evolutionary dynamics for different lake sizes, we gain insight into the effect of this stochasticity on evolutionary branching.

Since we are specifically interested in the effect of stochasticity on evolutionary branching, we choose to keep the model as simple as possible. The species that inspired this study (Arctic charr) is a sexual species whose populations are size structured (J. Andersson et al., submitted). However, in this paper we choose to ignore both these aspects in order to focus exclusively on the effect of absolute population size.

THE MODEL

Deterministic model

We model an unstructured, asexual consumer population whose density is denoted by \( N(t) \) and two resource populations whose densities are denoted by \( F_1(t) \) and \( F_2(t) \) and which are assumed to occur in two different habitats. We assume that the consumers have a heritable trait denoted by \( u \), which is the fraction of their lifetime they spend foraging on resource 1, while they spend the remaining fraction \( (1-u) \) foraging on the other resource. Note that \( u \) is hence restricted to the interval \((0, 1)\). For simplicity, we assume a Holling type 1 functional response, i.e. linear in prey density (but we have checked a model with a type 2 functional response that gave qualitatively the same results). We assume that the per capita birth rate is proportional to the consumption rate:

\[
\beta(u) = k_1 F_1(t) A_1(u) u + k_2 F_2(t) A_2(u) (1-u) \tag{1}
\]

where \( k_1 \) and \( k_2 \) are the efficiencies of converting food into offspring. \( A_1(u) \) and \( A_2(u) \) are the search rates in the two habitats (or ‘attack rates’: volume cleared of prey per unit of time). They are functions of \( u \) because we assume that the foraging ability on a resource depends on the time spent foraging on that resource. As a phenomenological model, we assume simple linear relations between \( u \) and the search rates:

\[
A_1(u) = a_1 + b_1 u \tag{2}
\]

\[
A_2(u) = a_2 + b_2 u \tag{3}
\]

The assumption that foraging capacity depends on the time spent foraging is based on experimental measurements in freshwater fish species (Andersson, 2003; Andersson et al., 2005). For Arctic charr, it has been demonstrated that exposure of juveniles during ontogeny to zooplankton prey, macroinvertebrate prey or a mixture of both influences the foraging performance at the end of the experiment [associated with a morphological effect (Andersson, 2003)]. A zooplankton diet increases the search rate on zooplankton, but diet has no effect on the search rate for macroinvertebrates. In terms of equations (2) and (3), this is modelled as \( b_1 > 0 \) and \( b_2 = 0 \) (assuming that habitat 1 is the pelagic habitat). We assume \( b_1 = 1, b_2 = 0, a_1 = 1, \) and \( a_2 = 1.5 \), resulting in an asymmetric, strong trade-off (Fig. 1e,f).
The total per capita search effort in a habitat equals the time that an individual spends in that habitat, multiplied with the search rate: $x_1(u) = uA_1(u)$ for habitat 1 and $x_2(u) = (1 - u)A_2(u)$ for habitat 2. A strong trade-off between search effort in the two

Fig. 1. (a–d) The symmetric trade-off $a_1 = 1$, $a_2 = 2$, $b_1 = 1$, $b_2 = -1$. (a) The search rate (equations 2–3) in habitat 1 (solid) and habitat 2 (dashed) vs. time spent in habitat 1. (b) The per-capita search effort in habitat 2 versus the one in habitat 1 reveals a strong (i.e. convex) trade-off. (c) The pairwise-invasibility plot (PIP). Shown is the invasion fitness (black: positive; white: negative) of a mutant with trait $u^\prime$ given the resident has trait $u$ and is at ecological equilibrium. The point $u^* = 0.5$ is an evolutionary branching point (EBP). (d) The trait evolution plot (TEP). The black area is the co-existence area defined as the set of pairs of traits which can mutually invade each other (set of protected polymorphisms). The arrow indicates the expected trajectory of divergence of $u_1$ and $u_2$. (e–h) The same as (a–d) but for Arctic charr parameters $a_1 = 1$, $a_2 = 1.5$, $b_1 = 1$, $b_2 = 0$. The point $u^* = 0.58$ is an EBP.

Other parameters: $K_1 = K_2 = 1$, $\delta_1 = \delta_2 = 1$, $\mu = 0.1$.

Delayed evolutionary branching

The total per capita search effort in a habitat equals the time that an individual spends in that habitat, multiplied with the search rate: $x_1(u) = uA_1(u)$ for habitat 1 and $x_2(u) = (1 - u)A_2(u)$ for habitat 2. A strong trade-off between search effort in the two
habitats is obtained if \( b_1/(a_1 + b_1) > b_2/a_2 \), and a weak trade-off with the opposite inequality. Plotting \( x_1(u) \) versus \( x_2(u) \) shows the shape of the trade-off. Figure 1a shows an example where the search rates in both habitats increase with time spent in that habitat \( (b_1 = 1, b_2 = -1, a_1 = 1, \text{ and } a_2 = 2) \). Figure 1b shows the resulting strong trade-off in the plot of \( x_1(u) \) versus \( x_2(u) \). Figure 1e shows a parameterization of the search rates based on the observations in Arctic charr, which also leads to a strong trade-off (Fig. 1f).

The resources are assumed to have no direct interaction with each other, and to follow semi-chemostat dynamics, which has been argued to appropriately describe resource dynamics in systems of size-selective fish foraging on zooplankton (Persson et al., 1998).

Assuming a consumer population monomorphic in trait \( u \), the deterministic dynamics of the three populations are described by the following set of ordinary differential equations (ODEs):

\[
\frac{dN}{dt} = (\beta(u) - \mu) N(t) \quad (4)
\]

\[
\frac{dF_1}{dt} = \delta_1(K_1 - F_1(t)) - F_1(t)N(t)A_1(u)u \quad (5)
\]

\[
\frac{dF_2}{dt} = \delta_2(K_2 - F_2(t)) - F_2(t)N(t)A_2(u)(1-u) \quad (6)
\]

where \( \mu \) is a constant mortality rate, \( \beta(u) \) is the per capita birth rate as defined in equation (1), and \( \delta_1 \) and \( \delta_2 \) are the renewal rates of the two resource populations. This deterministic formulation is appropriate for very large systems only; the model for populations in smaller lakes needs to incorporate demographic stochasticity and is described in the next sub-section.

Stochastic model

In finite populations, the number of consumer individuals is an integer number denoted by \( n(t) \). The consumer density \( N(t) \) is found by dividing the lake volume \( V \), thus \( N(t) = n(t)/V \). Very large systems (i.e. \( V \rightarrow \infty \)) have so many individuals that discrete events at the level of individuals (i.e. births and deaths) each have very small effects. The changes in the population density \( N(t) \) are then well-approximated by the deterministic model (equations 4–6). For small systems, however, these discrete events cannot be ignored. The deterministic (mean field) model can still be used to estimate the long-term average densities \( \bar{N} \), \( \bar{F}_1 \), and \( \bar{F}_2 \) by the equilibrium of equations (4–6), but the actual values will deviate from these values due to demographic stochasticity.

Each of the \( n(t) \) consumer individuals is characterized by its trait value \( u_i \) (where \( i = 1, \ldots, n(t) \)), which determines its use of the resources as described in the ODE model. The number of individuals changes through discrete birth and death events. The rate at which birth and death events occur depends on the number of individuals, their individual birth rates \( \beta(u_i) \), and the death rate \( \mu \). We describe the dynamics of a finite population using an individual-based, discrete event simulation model (i.e. a birth–death process in continuous time). Details of the simulation procedure are given in the Appendix.

In this paper, we assume clonal reproduction (sexual reproduction is discussed elsewhere). Offspring have the same genotype \( u \) as their parent unless, with probability \( P \), a mutation
occurs. In the case of a mutation, the newborn’s trait value is drawn from a truncated normal distribution with standard deviation $\sigma$ around the trait value of its clonal parent. If the drawn value is below 0 or above 1, it is replaced by 0 or 1, respectively.

Compared with the consumer population, the resources (zooplankton and macroinvertebrates) are much more numerous, with smaller body sizes and shorter generation times. Therefore, we choose to model their dynamics with ODEs analogously to equations (5–6) (see Appendix).

In the absence of mutations ($P = 0$), the individual-based model (IBM) defined like this is completely analogous to the deterministic model. In the limit of a very large lake volume ($V \to \infty$), the dynamics of $n(t)/V$ and the resource densities converge to equations (4–6).

RESULTS

Population dynamics

Deterministic

First, we scale away the conversion efficiencies by choosing scaled prey densities $F'_1 = F_1/k_1$ and $F'_2 = F_2/k_2$. Below, we assume parameter values that allow a monomorphic consumer population to have a positive population density in the entire interval of $u \in (0, 1)$. In terms of the model parameters, this requires that $\delta_1, \delta_2, K_1,$ and $K_2$ are positive and

$$0 < \mu < k_1K_1(a_1 + b_1u)u + k_2K_2(a_2 + b_2u)(1 - u)$$

for all $u$. We also require that $a_1$ and $a_2$ are positive and $b_1 > -a_1$ and $b_2 > -a_2$ such that the functions $A_1(u)$ and $A_2(u)$ have positive values for all $u$.

For the limiting cases $u = 0$ and $u = 1$, it can be shown analytically that the equilibrium of equations (4–6) is always stable (results not shown). An analytical result for the stability of the equilibrium for $0 < u < 1$ could not be obtained. Instead, the dynamics of equations (4–6) were studied using the software Content for numerical bifurcation analysis (Kuznetsov, 1995). We studied the stability of the internal equilibrium (i.e. positive densities of all three populations) for large ranges of all parameters. The dynamics were always found to converge to a stable equilibrium point ($\bar{N}, \bar{F}_1, \bar{F}_2$) (results not shown). Population cycles or alternative stable states were not found. Note that a linear functional response and semi-chemostat resource growth tend to produce more stable dynamics than a saturating functional response and/or logistic growth.

Stochastic

The dynamics of the individual-based model were studied with simulations. All else being equal, the variability around the expected steady state increases if the lake volume $V$ decreases: according to the scaling rule for demographic stochasticity (Desharnais et al., 2006), the coefficient of variation of population size is expected to scale like $CV(n) \propto n^{-0.5}$ (or, equivalently, $CV(n) \propto V^{-0.5}$). To establish a relation between the coefficient of variation (CV) of population abundance and lake volume, we used simulations without mutations ($P = 0$) and with a generalist consumer strategy ($u = 0.5$), for $10^4$ time units and for a range of lake volumes between $V = 1$ and $V = 1000$. For each lake volume, we computed the CV of abundance as $SD(n(t))/\bar{n}$, where $\bar{n}$ denotes the average abundance and $SD$ denotes the standard deviation. A power function $CV = c_1V^{c_2}$ was fitted to the measured
coefficient of variation of population abundance $n(t)$. Simulations of the IBM with $u = 0.5$ and $P = 0$ show that the coefficient of variation of abundance scales like

$$CV(n) = 0.3(V)^{-0.5} = 1.3(\bar{a})^{-0.5}$$

For the parameter values used in Fig. 2 and with $u = 0.5$, the expected number of consumers equals $\bar{N} = 18.7\ V$, while for $\bar{a} = 0$ it is $\bar{N} = 9.5\ V$. Demographic extinction occurs frequently when lake volume drops below $V = 2$, yet all runs with $V = 2$ persisted for more than $10^5$ generations and all runs with $V = 3$ for at least $10^7$ generations.

**Adaptive dynamics in large systems**

In large lakes, the adaptive dynamics of our stochastic model are straightforward (Fig. 2a). In the initial phase of the evolutionary dynamics, the resident population is monomorphic with $u = 0$. Habitat 1 is more or less unexploited and hence close to its carrying capacity, while habitat 2 is depleted. Directional selection then leads to the invasion of mutants that spend more time in habitat 1 and less time in habitat 2. This can be seen from the pairwise-invasibility plot (PIP) (van Tienderen and de Jong, 1986): if the resident has a low trait value,
then only mutants with a higher trait value ($u' > u$) have positive invasion fitness (Fig. 1c).

As the average trait value $\bar{u}$ gradually increases, the population feedback results in a balancing of the two resources: habitat 1 becomes more exploited while habitat 2 is released, reducing the selection gradient. The two habitats are ‘balanced’ when the consumption rate from the two habitats is equal: $F_1(t)A_1(\bar{u}) = F_2(t)A_2(\bar{u}) (1 - \bar{u})$, at which point the directional selection vanishes. The resident trait value that balances the resources is the evolutionary attractor of the monomorphic dynamics and is denoted by $u^*$. The value of $u^*$ depends on the parameter values: $u^* = 0.5$ with the symmetric trade-off (Fig. 1c) and $u^* = 0.58$ with the trade-off based on Arctic charr (Fig. 1g), assuming $K_1 = K_2 = 1$ and $\mu = 0.1$.

When the resources are balanced, evolution is no longer driven by resource densities but by the constraints imposed by the trade-off. In the case of a strong trade-off, the resident with trait $\bar{u} = u^*$ is located at a fitness minimum: mutations in both directions have positive invasion fitness (Fig. 1c). Consequently, selection becomes disruptive when $\bar{u}$ approaches $u^*$ and the asexual population can split into two sub-populations (Fig. 2a). A trait value that is both convergent stable (in the monomorphic dynamics) and evolutionarily unstable is referred to as an evolutionary branching point (EBP hereafter) (Geritz et al., 1998). By contrast, in the case of a weak trade-off, selection becomes stabilizing at this point and the population remains monomorphic with trait $\bar{u} = u^*$ (not shown).

After the split of the population into two incipient species, a rough demarcation of the course of the co-evolution of the two populations can be derived from a graph referred to as the ‘trait evolution plot’ or TEP (Geritz et al., 1998). This plot is constructed in three steps. The first step is to mark the areas for which a population with trait $u_2$ can invade the monomorphic equilibrium of a population with trait $u_1$ (i.e. the PIP). The second step is to mark the areas for which $u_1$ can invade $u_2$ (i.e. the PIP mirrored in the diagonal $y = x$). The areas that where marked twice (black areas in Fig. 1d) correspond to combinations of $u_1$ and $u_2$ which are mutually invasible and is referred to as the co-existence area or the set of protected polymorphisms (Metz et al., 1996). The third step is to mark, in the co-existence area, the points at which the fitness gradient is zero for one of the two branches, i.e. isoclines of the co-evolutionary dynamics. In the current model, the TEP has no isoclines; rather, the TEP predicts divergent co-evolutionary dynamics to $(\bar{u}_1, \bar{u}_2) = (0, 1)$ if $u^*$ is an EBP (Fig. 1d).

Co-existence of two emerging branches is likely only if their mean strategies, referred to as $\tilde{u}_1$ and $\tilde{u}_2$, are within the co-existence area. When a pair of traits of the incipient species $(u_1, u_2)$ moves out of the co-existence area (for some reason), one of the two branches is forced to extinction (i.e. its expected population size becomes zero), while the other one settles at its monomorphic equilibrium density (Metz et al., 1996). In Fig. 2a, branching is followed by symmetrical divergence of $\tilde{u}_1$ and $\tilde{u}_2$. Projected onto the TEP the trajectory of $(\tilde{u}_1, \tilde{u}_2)$ is expected to be well inside the co-existence area, close to a straight line from the evolutionary branching point $(u^*, u^*)$ to the final point $(0, 1)$ (Fig. 1d), but will deviate from it because mutations make random and finite steps.

The effect of lake size on evolutionary branching

The effect of lake volume on the adaptive dynamics in the IBM model is clearly illustrated with two examples in Fig. 2 showing the dynamics of the trait distribution in simulations of two lakes of different volume ($V = 1000$ and $V = 40$ units, respectively, corresponding to...
Both runs start with ten individuals with a trait value of \( u = 0 \). Note that the only ecological difference between the two lakes is their volumes. Hence given the same monomorphic trait value, the expected time-averaged densities \((\bar{N}, \bar{F}_1, \bar{F}_2)\) are the same in the two lakes. The two examples demonstrate three ways by which the evolutionary dynamics differ in the two lakes. First, in the small lake evolutionary change is slower; it takes longer both to approach the evolutionary attractor \( u^* \) and to diverge after branching. Second, in the small lake evolutionary branching is frequently followed by extinction of one of the incipient branches. Third, in the small lake the trait distribution fluctuates around \( u^* \), such that the mean trait in the population, \( \bar{u} \), spends little time at \( u^* \). The waiting time to eventual branching is much larger in the small lake, mainly for the two latter reasons.

These three observations are quantified more thoroughly in Fig. 3, which summarizes results for a range of lake volumes between \( V = 1 \) and \( V = 1000 \). For each lake volume ten simulations of the individual-based model were run, starting with ten individuals with a trait value of \( u = 0 \). Simulations ran for a maximum of \( 10^8 \) time units or shorter if branching occurred before that time.

Figure 3a shows the approach time, denoted by \( t_A \) and defined as the period until the mean trait \( \bar{u}(t) \) has approached the evolutionary attractor \( u^* \) to within 5%. The approach time \( t_A \) decreases gradually with increasing lake volume because mutants appear more frequently in large populations than in small ones. Figure 3b shows the ‘branching delay’, denoted by \( \Delta_B \), defined as the time elapsed between approaching the attractor and the moment of branching: \( \Delta_B = t_B - t_A \), where \( t_B \) is the time at branching. To detect branching, we subdivide the trait distribution into a lower, middle, and upper class. The middle class is defined as the central 10% of the current range of \( u \) in the population. Branching is defined as a moment when the middle class becomes empty. If during a single run multiple branching events occur (due to extinction of incipient branches), then \( t_B \) is defined as the last one. A striking result is that the branching delay \( \Delta_B \) increases dramatically when lake volumes drop below \( V = 100 \); in lakes with a volume below \( V = 20 \), branching is not observed within \( 10^8 \) time units (\( 10^7 \) generations). The cause of this result will be discussed below. Figure 3c shows the divergence delay, denoted by \( \Delta_D \) and defined as \( \Delta_D = t_B - t_P \), where \( t_P \) is the first moment that the population contains individuals with \( u = 0 \) and \( u = 1 \) at the same time. The divergence delay \( \Delta_D \) decreases slowly with lake volume, again because mutants appear more frequently in large populations than in small ones.

Sensitivity to mutation rate and step size

Quantitative aspects of the pattern in Fig. 3 depend on the width of the mutation distribution and hence on the mutation probability \( P \) and the standard deviation of the mutation steps \( \sigma \). Figure 4 shows results for the Arctic charr parameters and four different combinations of \( P \) and \( \sigma \). The figures show the same pattern of \( t_A \) and \( \Delta_B \) as discussed above (cf. Fig. 3a,b). Increasing \( \sigma \) or \( P \) has a large quantitative influence; it results in a reduction of both the smallest observed time to branching and the minimum lake volume in which branching can occur. The smallest observed branching delay in large lakes ranges from 100 generations (\( \sigma = 0.05, P = 0.01 \)) to 5000 generations (\( \sigma = 0.01, P = 10^{-4} \)). These patterns are confirmed by the results with a symmetric trade-off over the entire range of \( P \) and \( \sigma \) (data not shown). Despite these quantitative differences, for all values of \( P \) and \( \sigma \) a qualitative result holds: there is a critical lake volume \( V_{crit} \) below which evolutionary branching is not expected to occur within an ecologically realistic time scale (if ever).
Why is branching hard in small lakes?

Inspection of Fig. 2b suggests two processes that make branching hard in small lakes. First, the mean trait in the monomorphic resident drifts around $u^*$ such that during extended periods selection is directional rather than disruptive. Second, incipient branches may go extinct soon after branching.

Drift away from the evolutionary branching point

The first process, movement of the monomorphic resident away from $u^*$, may be explained by either a direct or an indirect effect of demographic stochasticity: (i) random genetic drift,
in which case \( \bar{u} \) changes randomly and hence possibly against the fitness gradient; or (ii) the consequence of variation in the fitness gradient caused by random variation in the consumer population (in terms of abundance and trait distribution). In the former case, demographic stochasticity affects the trait distribution directly (e.g. by chance individuals on one end of the distribution reproduce more frequently or die less frequently than on the other side). In the latter case, the stochastic effect is indirect: random effects in the consumer population result in an imbalance of the two resources, creating a fitness gradient pointing away from the evolutionary singular point \( u^* \). To distinguish between these two alternative explanations, we analyse a time series of the dynamics in a lake volume of \( V = 40 \) and a symmetric trade-off. During an interval of 20,000 generations, the consumer population fluctuates steadily around \( n \approx 748 \) (\( \bar{n} \) for \( u = 0.5 \)) while the two resources display complementary trends in their densities (Fig. 5a, b). The trends in the resources are strongly correlated to fluctuations in the mean trait value, \( \bar{u} \) (Fig. 5d). The instantaneous fitness gradient is found by differentiating the per capita growth rate \( W = \beta(u) - \mu \) (cf. equation 4) with respect to \( u \):

\[
\frac{dW}{du} = F_1(t)(a_1 + 2b_1u) - F_2(t)(a_2 - b_2 + 2b_2u)
\]  

(7)

Fig. 4. Approach time \( t_A \) (plus symbols) and branching delay \( \Delta_B \) (solid circles) for four different combinations of \( P \) and \( \sigma \) and trade-off parameters based on Arctic charr (Fig. 1e–h). Time is expressed in units of the average life span (1/\( \mu \)). All runs with \( V = 1 \) are extinct before approaching the attractor. Parameters: \( K_1 = K_2 = 1, \delta_1 = \delta_2 = 1, \mu = 0.1 \).
At regular intervals of 10 time units, the fitness gradient was plotted against the average trait in the population, $\bar{u}$. Figure 5c shows that only in a very narrow range around $u^*$ ($0.497 < \bar{u} < 0.503$) does the fitness gradient change sign due to random fluctuations. Yet Fig. 5d shows that $\bar{u}$ moves away from $u^*$ even outside this range (e.g. during the last 1500 generations). This implies that the mean trait moves against the fitness gradient during long periods, corresponding to the direct effect of demographic stochasticity (i.e. random genetic drift).

**Extinction of incipient branches**

The second process, extinction of incipient branches, may result from two causes: (i) drift-induced ‘forced’ extinction, or (ii) demographic ‘chance’ extinction. Random genetic drift could result in the pair of incipient species ($\bar{u}_1$, $\bar{u}_2$) moving out of the co-existence area (Fig. 1d), followed by the sure extinction of one of the two branches. To investigate this possible explanation, we analyse a number of extinction events in detail. Figure 6 shows the dynamics of two incipient branches in terms of their abundances, $n_1(t)$ and $n_2(t)$, and their mean traits, $\bar{u}_1(t)$ and $\bar{u}_2(t)$. According to our criterion for branching (see definition of $t_{gb}$, Fig. 5). A time series of the stochastic model in which no branching occurs, assuming a symmetric trade-off (Fig. 1a–d) and $V = 40$. (a) Consumer population abundance $n(t)$. (b) Food populations $F_1(t)$ and $F_2(t)$. (c) Instantaneous fitness gradient (equation 7) vs. the mean trait in the population $\bar{u}(t)$. (d) The mean trait in the population $\bar{u}(t)$. The horizontal dotted lines indicate $u = 0.497$ and $u = 0.503$, respectively (see text). Parameters: $K_1 = K_2 = 1$, $\delta_1 = \delta_2 = 1$, $\mu = 0.1$. Delayed evolutionary branching 63
the population branches at $t_B = 7800$ generations. At their origin, the two incipient branches have similar abundances ($n_1(t_B) = 393$, $n_2(t_B) = 349$). The numbers in the two branches, however, fluctuate considerably and these fluctuations are reflected in the resource densities (Fig. 6b, d). Figure 6c shows the trajectory of $(\hat{u}_1, \hat{u}_2)$ in the trait evolution plot (TEP) (cf. Fig. 1d). The expected path of $(\hat{u}_1, \hat{u}_2)$, based on the deterministic model, is along...
the sub-diagonal from the EBP (0.5, 0.5) towards complete divergence (0, 1) (see arrow in Fig. 1d). Branch \( n_1 \) goes extinct eventually; if this were a ‘forced’ extinction owing to random drift, then the actual trajectory of \((\bar{u}_1, \bar{u}_2)\) should drift towards the extinction boundary of \( n_1 \) (E1 in Fig. 6c). The plotted trajectory does indeed drift in this direction. The extinction of \( n_1 \) may thus be the result of random drift of \((\bar{u}_1, \bar{u}_2)\) reducing the expected abundance of this branch, although extinction occurs already before reaching the limit of the co-existence boundary.

To verify the generality of this observation, we analysed 33 simulations of 20,000 generations with parameters as in Fig. 6, in which 90 extinction events were recorded. We determined the trajectory of \((\bar{u}_1, \bar{u}_2)\) prior to extinction. In 79% of cases, we found that the extinct branch was closer to its extinction boundary than the other branch (as in Fig. 6). This result suggests that the extinction events can be partly attributed to random drift. The 21% of cases, however, suggest that ‘chance’ extinction occurs as well.

With respect to ‘chance’ extinction, we note that during the initial phase of divergence \( \bar{u}_1 \) and \( \bar{u}_2 \) are so similar that the dynamics of \( n_1 \) and \( n_2 \) are almost neutral (\( n_1 \) and \( n_2 \) are interchangeable for \( u_1 = u_2 \)). In mathematical terms, if \( u_1 = u_2 \) there is a stable equilibrium but its leading eigenvalue is almost zero such that fluctuations in \( n_1 \) and \( n_2 \) are not easily dampened. To check this idea we extended the deterministic model (equations 4–6) to two consumer populations \( N_1 \) and \( N_2 \), and computed the eigenvalues of the equilibrium of the ecological dynamics, assuming symmetric divergence (\( u_2 = 1 - u_1 \)). The first eigenvalue \( \lambda_1 = 0 \) for \( \bar{u}_1 = 0.5 \) and decreases slowly as \( \bar{u}_1 \) and \( \bar{u}_2 \) diverge. This deterministic model predicts that, following a perturbation, the total consumer density \( N_1 + N_2 \) converges quickly to its equilibrium value, while \( N_1 \) and \( N_2 \) converge very slowly to their respective steady states, mirrored by \( F_1 \) and \( F_2 \). Analogously, in the IBM the large fluctuations of \( n_1 \) and \( n_2 \) are complementary such that \( n_1 + n_2 \) remains more or less constant (Fig. 6b). The near-neutral stability (\( \lambda_1 = 0 \)) of the dimorphic equilibrium with small divergence (\( \bar{u}_1 = \bar{u}_2 \)) means that fluctuations of \( n_1 \) and \( n_2 \), caused by demographic stochasticity, are not readily dampened. This permits large, long-term fluctuations, which may lead to extinction of one of the branches.

We postulate that incipient branches are sensitive to ‘chance’ extinction owing to the near-neutral stability of the dimorphic equilibrium. Drift, however, results in a bias in the extinction probability: the branch that is closest to its extinction boundary is most likely to go extinct.

DISCUSSION

With a simple model we have shown that the absolute population size may influence the outcome of evolutionary dynamics. Only in large populations is evolutionary branching predicted to occur upon reaching the evolutionary branching point (EBP). In small populations, branching is predicted to be delayed. The delay increases quickly with decreasing absolute population size. Below a certain population size, the delay can be so long that on any relevant time scale (say, up to \( 10^6 \) generations) branching is not expected to occur at all. Based on this result, we expect that evolutionary branching has occurred more frequently in large than in small populations.

We identified two mechanisms that contribute to the delay in evolutionary branching. First, random genetic drift of the mean trait in the population causes the population to spend long periods (many consecutive generations) away from the EBP. During such time
intervals, selection is not disruptive but directional and branching is hence not expected to occur.

Second, soon after branching the incipient branches are prone to ‘chance’ extinction. The reason is that individuals of the two branches are almost substitutable because their trait values are very similar. The relative dynamics of the two branches are hence almost neutral: a perturbation in the ratio $n_1/n_2$ is restored very slowly. The contrast in time scales between (slow) relative dynamics of similar phenotypes and (fast) aggregate dynamics of the total consumer density was recently analysed by Meszéna et al. (2005), who studied the dynamics of a number of similar clones in a (unimodal) distribution. Our result suggests that their result is relevant even for the dimorphic dynamics soon after branching. We hypothesize that the contrast between relative and aggregate dynamics explains the frequent extinction of incipient branches in small systems with demographic stochasticity. However, owing to random drift, the two branches do not have equal probability to go extinct. The one that is closest to its extinction boundary is most likely to disappear.

We have shown that quantitatively, these effects depend on the mutation rate and step size. In addition, they depend on the strength of directional and disruptive selection. Increasing the curvature of the trade-off (Fig. 1b) reduces the branching delay (especially in small lakes) and allows for branching in smaller lakes. The qualitative pattern, however, of the relation between $V$ and $\Delta_B$ remains the same (data not shown).

The role of random genetic drift

Random genetic drift, resulting from ‘sampling error’ and finite population size, is one of the basic mechanisms of evolution, together with mutation and natural selection. Drift may decrease genetic variation or produce large shifts in allele frequencies (e.g. the founder effect) (Gavrilets, 2004). Allopatric speciation is usually seen as a by-product of divergence by random genetic drift (or by directional selection) in geographically isolated populations (Mayr, 1963; Provine, 2004). Yet in adaptive dynamics theory, the roles of drift and finite population size have received little attention (but see Prouls and Day, 2001; Cadet et al., 2003; Parvinen et al., 2003). In the recent polemic on adaptive dynamics and population genetics, it has been suggested that the effect of absolute population size on evolutionary dynamics can be scaled away by tuning the mutation rate (criticized by Waxman and Gavrilets, 2005b). Our results clearly falsify this assertion: the absolute population size per se may influence evolutionary dynamics through both drift and demographic extinction.

We found that random genetic drift can give rise to long delays of evolutionary banching. This means that random drift demotes speciation, a result that is in contrast with its role in classic allopatric speciation theory.

In small populations, random drift results in a weak coupling of changes of the genotype distribution and the fitness gradient. We found that the mean trait $\bar{u}$ may move against the fitness gradient during long periods, spanning many generations. This observation has implications for the evolution of small populations and of exploited fish populations in particular. It has been argued that exploitation of fisheries stocks has caused a fast evolutionary response (Olsen et al., 2004). However, most stocks are heavily depleted and hence at very low densities. This means that based on our results we do not expect genetic changes directed by the fitness gradient imposed by fisheries, but rather by random drift.
ACKNOWLEDGEMENTS

We thank S. Legendre, N. Champagnat, A. Lambert, and J.A.J. Metz for discussions on the evolutionary branching in stochastic populations. Eva Kisdi and two anonymous reviewers provided thoughtful comments that helped to improve the manuscript. D.C. and A.M.dR. acknowledge financial support by the Dutch Science Foundation (NWO).

REFERENCES

APPENDIX: DISCRETE EVENT SIMULATION

The simulation proceeds by stepping from one discrete event (birth or death) to the next. In between events, the model allows for resource dynamics. At time \( t \), the timing of the next event to take place (be it a death or birth event) is determined from the total event rate,

\[
E(t) = n(t)\mu + \sum_{i=1}^{n(t)} \beta(u_i)
\]

where \( \beta(u_i) \) is the rate at which an individual with trait \( u_i \) gives birth (equation 1). Assuming the events are exponentially distributed, the timing of the next event is chosen as

\[
t_{\text{next}}(t) = t + \frac{\ln(1 + z)}{E(t)}
\]

where \( z \) is a random number drawn from a uniform distribution. In between discrete events, the food populations change according to differential equations (equations 8–9) that are integrated with a simple Euler method (or assumed to be in quasi-steady state; see below). The integration step size \( \Delta t \) is either \( t_{\text{next}}(t) - t \) or \( \tau \), whichever is smaller (\( \tau = 0.1 \) by default). After each event and each integration step, the rates \( \beta(u_i) \) and \( E(t) \) and the event time \( t_{\text{next}} \) are updated to the current food densities. If the time of the next event is reached, an individual is chosen from the population randomly but weighed by the individuals’ event
rates $\mu + \beta(u_i)$. With probability $\beta(u_i)/(\beta(u_i) + \mu)$, the chosen individual reproduces one offspring, otherwise it dies.

The ordinary differential equations for the dynamics of the food populations in the individual-based model are:

\[ \frac{dF_1}{dt} = \delta_1(K_1 - F_1(t)) - F_1(t) \sum_{i=1}^{n(t)} \frac{A_1(u_i)u_i}{V} \quad \text{(A1)} \]

\[ \frac{dF_2}{dt} = \delta_2(K_2 - F_2(t)) - F_2(t) \sum_{i=1}^{n(t)} \frac{A_2(u_i)(1 - u_i)}{V} \quad \text{(A2)} \]

Since resource dynamics are fast relative to consumer dynamics ($\delta_i \gg \mu$), $F_1(t)$ and $F_2(t)$ can be assumed to be in steady state with the current consumer population. By setting $dF_1/dt = 0$ and $dF_2/dt = 0$, we find the quasi-steady state resource levels:

\[ \bar{F}_1(t) = \frac{K_1}{1 + \frac{1}{\delta_1 V} \sum_{i=1}^{n(t)} A_1(u_i)u_i} \quad \text{(A3)} \]

\[ \bar{F}_2(t) = \frac{K_2}{1 + \frac{1}{\delta_2 V} \sum_{i=1}^{n(t)} A_2(u_i)(1 - u_i)} \quad \text{(A4)} \]

We use either equations (A1–A2) or equations (A3–A4); the results are indistinguishable.
The Effect of Population Size and Recombination on Delayed Evolution of Polymorphism and Speciation in Sexual Populations

David Claessen,1,2,* Jens Andersson,3† Lennart Persson,3‡ and André M. de Roos2§

1. Centre de Enseignement et d’ Recherche sur l’Environnement et la Société–Environmental Research and Teaching Institute, Laboratory of Ecology and Evolution (Unité Mixte de Recherche 7625), École Normale Supérieure, 24 rue Lhomond, F-75230 Paris Cedex 05, France;
2. Institute for Biodiversity and Ecosystem Dynamics, University of Amsterdam, P.O. Box 94084, 1090 GB Amsterdam, The Netherlands;
3. Department of Ecology and Environmental Science, Umeå University, SE-90187 Umeå, Sweden

Submitted June 19, 2007; Accepted January 7, 2008; Electronically published May 15, 2008

Abstract: Recent theory suggests that absolute population size may qualitatively influence the outcome of evolution under disruptive selection in asexual populations. Large populations are predicted to undergo rapid evolutionary branching; however, in small populations, the waiting time to branching increases steeply with decreasing abundance, and below a critical size, the population remains monomorphic indefinitely. Here, we (1) extend the theory to sexual populations and (2) confront its predictions with empirical data, testing statistically whether lake size affects the level of resource polymorphism in arctic char (Salvelinus alpinus) in 22 lakes of different sizes.

For a given level of recombination, our model predicts qualitatively similar relations between population size and time to evolutionary branching (either speciation or evolution of genetic polymorphism) as the asexual model, while recombination further increases the delay to branching. The loss of polymorphism at certain loci, an inherent aspect of multilocus-trait evolution, may increase the delay to speciation, resulting in stable genetic polymorphism without speciation.

The empirical analysis demonstrates that the occurrence of resource polymorphism depends on both lake size and the number of coexisting fish species. For a given number of coexisting species, the level of polymorphism increases significantly with lake size, thus confirming our model prediction.

Keywords: adaptive dynamics, arctic char, demographic stochasticity, extinction, multilocus genetics, random genetic drift.

Populations with small absolute population size exhibit larger stochastic fluctuations caused by the randomness of demographic events than larger populations (Desharnais et al. 2006). In the hypothetical case of two populations in two lakes that differ only in their volume, the population in the smaller lake is therefore more likely to become extinct despite its average population density (number per m$^3$) being the same as the population in the bigger lake. Populations with small absolute population size are also subject to higher levels of genetic drift. Recent theoretical results have shown that absolute population size, by determining the level of these effects, may qualitatively influence the outcome of evolutionary dynamics. On the one hand, the direction of evolution in populations subject to demographic stochasticity may differ from the expectation based on a deterministic model (Proulx and Day 2001; Cadet et al. 2003; Parvinen et al. 2003). On the other hand, in asexual populations under disruptive selection, demographic stochasticity is predicted to interfere with the process of evolutionary branching (Claessen et al. 2007).

Large populations are predicted to undergo rapid evolutionary branching. However, in small populations, the waiting time to branching increases very steeply with decreasing population size, and below a critical size, the population is predicted to remain monomorphic indefinitely. The two processes that have been proposed to explain this delay in evolutionary splitting are genetic drift and the high risk of extinction of incipient branches (Claessen et al. 2007).

An open question is whether these results, and in particular delayed evolutionary branching, carry over to sex-
ual populations and hence to the dynamics of speciation (in the sense of reproductive isolation) or the evolution of genetic polymorphism. The process of evolutionary branching was originally described for purely phenotypic models (Geritz et al. 1997). The underlying genetics were implicitly or explicitly assumed to correspond to clonal reproduction, that is, the absence of diploidy, recombination, multilocus dynamics, and so forth. The strength of such asexual models is to elucidate the link between ecological dynamics and fitness and to discover which ecological processes can create conditions that favor evolutionary branching. Extensions of such models that include simple, sexual genetics have demonstrated that the role of genetics is nontrivial. First, sexual populations may respond to disruptive selection by finding other “solutions” than splitting into two daughter populations—for example, the evolution of genetic polymorphism (Kisdi and Geritz 1999) or of sexual dimorphism (Bolnick and Doebeli 2003). In the latter case, the ecological roles of males and females diverge to fill the ecological niches generating the disruptive selection. In both cases, the population remains panmictic. Second, sexual reproduction is expected to make evolutionary branching difficult through the continuous formation of intermediate hybrid genotypes that prevent the splitting of the population. Dieckmann and Doebeli (1999) have shown that evolutionary branching may yet occur in sexual models if the model allows for the evolution of assortative mating. Under disruptive selection, assortative mating is favored and may thus be expected to evolve once the population has reached an evolutionary branching point (EBP). Third, in the case of multilocus traits, interactions between loci may result in unexpected dynamical patterns, such as polymorphism formation and collapse, that influence the predicted evolutionary trajectory and the resulting level of polymorphism (Kopp and Hermisson 2006; van Doorn and Dieckmann 2006). These patterns emerge from the collective dynamics of multiple alleles at multiple loci under frequency-dependent disruptive selection. Due to the added complexities related to sexual reproduction and the associated possibility of multilocus genetics, it is not clear a priori that results from asexual population models carry over to sexual populations.

In this article, we extend the model of Claessen et al. (2007) by taking into account sexual reproduction. This allows us to assess whether the phenomenon of delayed evolutionary branching is robust to the occurrence of segregation, recombination, and multilocus evolution. In our model, evolutionary branching can result in either a protected polymorphism in a panmictic population (i.e., dimorphism at some or all the loci) or speciation (i.e., dimorphism at all loci followed by sorting of genotypes and reproductive isolation of purely homozygous genotypes). We can thus study the effect of small population size on the evolution of genetic polymorphism and on speciation.

The extended model will allow us to confront model predictions with data on sexual species. In particular, freshwater fish species offer interesting opportunities for testing the theory of adaptive dynamics due to the frequent but variable occurrence of resource polymorphism (Skúlason and Snorrason 2004). Such polymorphisms often include a pelagic morphotype feeding mainly on zooplankton and a benthic morphotype feeding mainly on macroinvertebrates (Smith and Skúlason 1996; Skúlason and Snorrason 2004). Examples are sticklebacks, whitefish pumpkineed, and bluegill sunfishes (Andersson et al. 2007). We focus on arctic char (Salvelinus alpinus), for which the degree of polymorphism varies between one and four morphotypes per lake (Alekseyev et al. 2002; Adams et al. 2003; Skúlason and Snorrason 2004). The explanation for the frequent but variable polymorphism in freshwater fish species is still a matter of debate and may differ for different lake systems. A current hypothesis is that resource polymorphism is an early stage of an ongoing speciation process (Lavin and McPhail 1986; Schluter 1996; Skúlason and Snorrason 2004; Knudsen et al. 2006). Different morphotypes may have allopatric origins, but once in sympathy through repeated invasion, they remain polymorphic due to disruptive selection that results from ecological interactions, such as competition for food (Svárdson 1979; Nymann et al. 1981; Schluter and McPhail 1993; Rundle and Schluter 2004). Alternatively, the morphotypes may originate sympatrically as a consequence of adaptive sympatric speciation. Evidence for sympatric (within-lake) origin of arctic char morphotypes stems from both genetic (Gislason et al. 1999) and morphological studies (Alekseyev et al. 2002). In both scenarios, disruptive selection is an essential force driving and maintaining the sympatric divergence and coexistence of morphotypes.

The goals of this article are (i) to extend the theory on delayed evolutionary branching to sexual populations and (ii) to confront the model prediction with empirical data. Part 1 of the article is theoretical and is a study of the implications of sexual reproduction for the phenomenon of delayed evolutionary branching. Part 2 is an empirical test of the main prediction based on this theory, using data on the level of resource polymorphism in arctic char (S. alpinus) populations in 22 lakes of different sizes. Under the hypothesis of a sympatric origin of the morphotypes, our theory predicts a positive relation between lake size and the occurrence and level of polymorphism, whether in the form of genetic polymorphism or reproductively isolated subpopulations.
Part 1: Theory

The theory is developed in the framework of adaptive dynamics. The basic assumption of this theory is that evolution takes place in a dynamic fitness landscape in which fitness is the outcome of density- and/or frequency-dependent ecological interactions between individuals (Metz et al. 1992). The theory shows that in such a dynamic fitness landscape, “upward” movement, resulting from directional selection, can take an evolving population to a fitness minimum (Eshel 1983), often referred to as an “evolutionary branching point” (Geritz et al. 1997). At the EBP, selection turns disruptive, and the population may branch into two sympatrically diverging subpopulations (Metz et al. 1992; Geritz et al. 1997; Dieckmann and Doebeli 1999), a genetic polymorphism (Kisdi and Geritz 1999), or a sexual dimorphism (Bolnick and Doebeli 2003).

We study evolutionary branching in a simple model of one consumer and two resource populations. The resources are assumed to occur in two distinct habitats, and so the consumers spend a certain fraction of their time in each habitat. We focus on the evolution of this time allocation, that is, of the relative utilization of either resource. The model is loosely based on the ecology of lake fish such as arctic char (Salvelinus alpinus), perch (Perca fluviatilis) and sticklebacks (Gasterosteus aculeatus). Such species often have access to two resources in different habitats: zooplankton in the pelagic habitat and macroinvertebrates in the benthic habitat. The evolutionary trait can be interpreted as either the fraction of the lifetime in each habitat (in the case of an ontogenetic niche shift) or the daily time fraction in each habitat.

Studies have shown that a consumer population exploiting two distinct resource populations can evolve to an EBP and hence potentially speciate into two specialists or give rise to a genetic polymorphism (e.g., Kisdi and Geritz 1999; Day 2000; Claessen and Dieckmann 2002; Rueffler et al. 2006). The condition for evolutionary branching in such models is that the generalist strategy exploiting both resources has a lower fitness than mutant strategies with a slightly higher degree of specialization in either direction. In other words, the shape of the trade-off between performance in the two habitats determines the outcome of the evolutionary dynamics. When fitness is a linear combination of performance in two habitats (as is the case here), evolutionary branching is expected if the trade-off is “strong” (convex). An evolutionarily stable generalist is expected if the trade-off is “weak” (concave) or “neutral” (linear; Rueffler et al. 2006).

Strong trade-offs may result from various biological mechanisms, including diet-induced changes in foraging capacity (Claessen et al. 2007). This mechanism can be understood intuitively as follows. Consider the energy gain obtained from two resources that occur in two different habitats. The total gain equals search rate \( \times \) resource density \( \times \) time spent in the habitat. Time splitting between the habitats amounts to a neutral trade-off because the gain from each habitat is a linear function of the time spent in the habitat. If, in addition, the search rate increases with the time spent in a habitat, the trade-off becomes strong (convex). A positive dependence of foraging ability on foraging time may result when foraging on a certain prey type (or habitat) induces physiological changes, for example, morphological adaptation. For a number of fish species, it has been demonstrated that diet influences individual development and morphology, resulting in increased habitat-specific foraging capacity (Robinson and Wilson 1995; Day and McPhail 1996; Andersson 2003; Andersson et al. 2005).

The Model: The Individual Level

Our deterministic model is the same as the one presented in Claessen et al. (2007), while our stochastic model is a multilocus, diploid, sexual version of the single-locus, asexual model presented in that article. Below, we outline the model with a focus on the details of sexual reproduction and inheritance; additional details of the deterministic model, parameter values, and the simulation procedure can be found in Claessen et al. (2007).

Phenotype and Genotype

We assume that consumers have a phenotype, denoted by \( u \), that is the fraction of time they spend foraging on resource 1, while they spend the remaining fraction \((1 - u)\) foraging on resource 2 (as discussed above). We assume that the value of \( u \) is determined additively (no dominance or epistasis) by \( L \) diploid loci. Each allele may have a value between 0 and 1, and the phenotype is determined as the mean of all alleles:

\[
u = \frac{1}{2L} \sum_{k=1}^{L} (x_k + y_k),
\]

where \( x_k \) and \( y_k \) are the values of the two alleles at locus \( k \).

Assortative Mating

For simplicity, we assume a fixed level of assortative mating. An alternative approach is to allow for the evolution of assortativeness as an independent evolutionary trait; studies have demonstrated that under conditions of disruptive selection, assortativeness is favored by natural or sexual selection (Dieckmann and Doebeli 1999; van
Doorn et al. 2004), but this is outside the scope of our study (but see app. B).

We model assortative mating as follows. For an individual $i$, a candidate partner $j$ is selected randomly from the population of the opposite sex. The phenotypic difference between the two individuals is computed as $d = \text{abs}(u_i - u_j)$. The candidate is rejected with probability

$$q = \begin{cases} 
0 & \text{if } d < 0.5 - (0.5/S), \\
1 & \text{if } d > 0.5 + (0.5/S), \\
S(d - 0.5) + 0.5 & \text{otherwise.}
\end{cases}$$  \hspace{1cm} (2)$$

Figure 1 shows the relation between the phenotypic difference between two potential partners and the probability of partner rejection. With $S = 0$, mating is random, while a value of $S > 0$ implies that the probability of rejecting a randomly selected partner increases with the phenotypic distance $d$ between the individuals. A value of $S > 1$ results in reproductive isolation between individuals at the extremes of the phenotype space and is hence required for speciation to be possible. Our standard value of $S = 2$ results in complete rejection of candidates at a large phenotypic distance ($q = 1$ if $d > 0.75$), thus allowing for reproductive isolation. In simulations, a mating event is canceled if after 100 attempts no suitable partner has been found (this amounts to a small cost of assortativeness but happens extremely rarely in our simulations).

**Inheritance**

We assume there are $L$ loci on a single pair of homologous, autosomal chromosomes. At reproduction, a gamete is defined by randomly drawing one of the two homologous chromosomes. We allow for recombination by assuming that between any two adjacent loci, crossing over occurs with a probability $p_c$. We thus assume the loci are equidistantly distributed on the chromosome. By tuning the parameter $p_c$, we can control the level of recombination, from no recombination at all ($p_c = 0$) to independent segregation ($p_c = 0.5$). In the case of $p_c = 0.5$, the loci behave as if they were each located on a separate chromosome. In the case of $p_c = 0$, all the loci together behave like a single locus. With $p_c = 0$ and $S = 0$, we obtain results analogous to Kisdi and Geritz (1999), that is, evolutionary branching of a single locus in a panmictic sexual population that results in a stable genetic polymorphism.

**Mutations**

At birth, mutations occur with frequency $p_m$ per allele. The allele’s trait value is then drawn from a truncated normal distribution with standard deviation $\sigma(2L)^{1/2}$ around the value of the parental allele. This mutation size at the allele level results in a variance of $\sigma^2$ at the trait level, independently of $L$ (van Doorn et al. 2004). If the drawn value is below 0 or above 1, it is replaced by 0 or 1, respectively.

Our assumption of several loci, each of them with a continuum of the possible alleles, differs from the perhaps more standard assumption that a quantitative character is determined by the cumulative effect of many biallelic, or few-allelic, loci (e.g., Dieckmann and Doebeli 1999). Our assumption is, however, the most straightforward extension of our previous model to multiple diploid loci and hence allows for a direct comparison.

**Vital rates.** We assume a constant death rate $\mu$. The per capita birth rate $\beta(u)$ is assumed to be proportional to the consumption rate, which in turn is assumed to be proportional to the resource densities $(F(t), F(t))$, the search rates in each habitat $(A_i(u), A_j(u))$, and the time spent in each habitat:

$$\beta(u) = F(t)A_i(u)u + F(t)A_j(u)(1 - u).$$  \hspace{1cm} (3)$$

The search rates are functions of the fraction of time spent in each habitat:

$$A_i(u) = a_i + b_iu,$$  \hspace{1cm} (4)$$

$$A_j(u) = a_j + b_ju.$$  \hspace{1cm} (5)$$

These functions represent our assumption of diet-induced changes in foraging capacity. For arctic char, it has been demonstrated that exposure of juveniles during ontogeny to either zooplankton prey, macroinvertebrate prey, or a...
mixture of both influences the foraging performance at the end of the experiment (associated with a morphological effect; Andersson 2003). Their results can be stated this way: the search rate on zooplankton increases with the time spent in the pelagic habitat, while the search rate on macroinvertebrates is unaffected. If the pelagic is habitat 1, this can be modeled as \( b_1 > 0 \) and \( b_2 = 0 \).

The Model: Population Level

**Deterministic Model**

For the deterministic model, we assume that all alleles are identical, in which case the dynamics of alleles and hence sexual reproduction can be ignored. Our deterministic model of the population dynamics is given by

\[
\frac{dN}{dt} = (\beta(u) - \mu)N(t), \tag{6}
\]

\[
\frac{dF_1}{dt} = \delta_1(K_1 - F_1(t)) - F_1(t)N(t)A_1(u)u, \tag{7}
\]

\[
\frac{dF_2}{dt} = \delta_2(K_2 - F_2(t)) - F_2(t)N(t)A_2(u)(1 - u), \tag{8}
\]

where \( N(t) \) is the consumer population density and \( F_1(t) \) and \( F_2(t) \) are the densities of its two resource populations. The resources are assumed to have no direct interaction with each other and to follow semichemostat dynamics, which has been argued to appropriately describe resource dynamics in systems of size-selective fish foraging on zooplankton (Persson et al. 1998). Parameters \( \delta_i \) and \( K_i \) are the renewal rate and maximum density of resource \( i \).

**Stochastic Model**

For small populations we need to incorporate demographic stochasticity. We denote the number of consumer individuals by the integer number \( n(t) \). The density of consumers \( N(t) \) is then obtained as \( N(t) = n(t)/V \), where \( V \) is the lake volume. Very large systems (i.e., \( V \to \infty \)) have so many individuals that discrete events at the level of individuals (i.e., births and deaths) each have very small effects. The changes in the population density \( N(t) \) are then well approximated by the deterministic model (eqq. [6]–[8]). For small systems, however, these discrete events cannot be ignored. The deterministic model can still be used to find the long-term average densities \( \bar{N} \), \( \bar{F}_1 \), and \( \bar{F}_2 \), which correspond to the equilibrium of equations (6)–(8) (unpublished data), but the actual values will deviate from these values due to demographic stochasticity. All else being equal, the variability around the expected steady state increases if \( V \) decreases; according to the scaling rule for demographic stochasticity (Desharnais et al. 2006), the coefficient of variation (CV) of population size \( n \) is expected to scale in this way: \( CV(n) \propto n^{-0.5} \) (or, equivalently, \( CV(n) \propto V^{-0.5} \)).

The consumer individuals are characterized by their trait value \( u_i \), with \( i = 1, \ldots, n(t) \). The number of individuals changes through discrete birth and death events. The rate at which birth and death events occur depends on the number of individuals, their individual birth rates \( \beta(u_i) \), and the death rate \( \mu \). We use an individual-based, discrete-event simulation model (i.e., a birth-death process in continuous time) to describe the population dynamics (Claessen et al. 2007). The stochastic model was coded in the C programming language, using algorithms for individual-based discrete-event simulation that were written by U. Dieckmann (Claessen and Dieckmann 2002).

Compared with the population of consumers, the resources (zooplankton and macroinvertebrates) are much more numerous, with smaller body sizes and shorter generation times. We therefore choose to model their dynamics with ordinary differential equations analogous to equations (7) and (8). In simulations, we assume that the resources are in quasi-steady state with the current consumer population (Claessen et al. 2007). In the absence of polymorphism, the individual-based model defined this way is fully analogous to the deterministic model. In the limit of \( V \to \infty \), the dynamics of \( n(t)/V \) and the resource densities converge to equations (6)–(8).

**Model Results**

First, we discuss two illustrative example runs of the stochastic model. Second, we present a systematic and quantitative analysis of the effect of lake volume and recombination on the predicted time to speciation. All the simulations presented below start from a monomorphic resident population with a trait \( u \) somewhere between 0 and 1. Generically, the adaptive dynamics then consist of two phases. In the first phase, the ecological equilibrium of the current resident population and its two resources is characterized by an imbalance: due to its biased trait value \( (u \neq u^*) \), the consumer overexploits one resource and leaves the other one relatively abundant (Claessen and Dieckmann 2002; Claessen et al. 2007). Mutants that spend more time in the underexploited niche can invade (directional selection). The average trait, denoted by \( \tilde{u} \), thus evolves gradually toward the singular point \( u^* \), at which the consumer’s intake rates in the two habitats are balanced and the directional selection vanishes. Evolution in the second phase depends on the trade-off between gain from the two habitats, that is, \( uA_1(\tilde{u})\tilde{F}_1 \) versus \((1 - u)A_2(\tilde{u})\tilde{F}_2 \), where \( \tilde{F}_1 \) and \( \tilde{F}_2 \) are the equilibrium resource densities. In the case of a “weak” trade-off (e.g., if the
Figure 2: Examples of adaptive dynamics with three loci (\(L = 3\)) in two lake volumes. Depicted is the phenotypic trait distribution \(n(u)\) at time \(t\) (note different scaling of X-axis in a and b). a, Lake volume \(V = 200\): all loci branch on reaching the evolutionary branching point (\(u^* = 0.5\)). Seven phenotypes appear once the alleles have evolved into discrete clusters. Assortative mating results in speciation (reproductive isolation) after \(\approx 8,800\) generations. b, Lake volume \(V = 30\): branching is delayed; note the average phenotype drifting around and unsuccessful branching events (arrows). Branching results in a polymorphism of three phenotypes caused by a stable dimorphism at a single locus. Parameters: \(S = 2\), \(d_c = 0.1\), \(b_{33} = 0.01\), \(\sigma = 0.01\), \(a_1 = 0\), \(a_2 = 2\), \(b_1 = 2\), \(b_2 = -2\).

In general, the relation between \(V\) and absolute population size \(n\) depends on \(u\), or more precisely, on the distribution of \(u\). Note, however, that we have chosen all parameter combinations used in this article so the EBP is at \(u = 0.5\) and the equilibrium population size of a monomorphic population with \(\hat{u} = 0.5\) equals \(n = 18V\).

Two Illustrative Examples

In the following, we use parameter values for which the singular point \(u^*\) is an evolutionary branching point (i.e., \(b_j/(a_j + b_j) > b_i/a_i\); Claessen et al. 2007). For simplicity, we use parameters that correspond to a symmetric trade-off (\(b_2 = -b_1\), \(a_1 = a_2 + b_2\)). Assuming symmetric resource parameter values (\(r_i = 1, K_i = K_j\)), the EBP is then at \(u^* = 0.5\). Qualitatively similar results have been obtained with asymmetric parameter combinations (data not shown). We assume \(S = 2\) (but see app. A).

Figure 2a demonstrates the two phases of adaptive dy-
amics for a diploid population in which trait \( u \) is determined by three loci in a relatively big lake \((V = 200)\). The initial condition of the simulation is 10 individuals, of which all (six) alleles equal 0.25. The population quickly grows to about 3,200 individuals, which is a stable equilibrium for these parameter and trait values. The population responds to the directional selection by converging to the predicted EBP \((u = 0.5)\). The average population size at \( \dot{u} = 0.5 \) equals \( 18V = 3,600 \) individuals. At the EBP, disruptive selection causes increasing genetic variability. During this period, all three loci undergo evolutionary branching. At each locus, two alleles appear that diverge toward the values \( \approx 0 \) and \( \approx 1 \), respectively (not shown). Initially, this phase is characterized by a continuous phenotypic distribution (fig. 2a) caused by variability in the degree of divergence between loci. Once the alleles approach their final values at all loci, discrete phenotypes appear. With \( k \) dimorphic loci, the number of phenotypes equals \( 2k + 1 \) (seven phenotypes in the example; fig. 2a). While extreme phenotypes have superior fitness due to the strong trade-off, the intermediate phenotypes are produced through segregation and crossing over. Speciation, defined as the emergence of two reproductively isolated subpopulations, can occur once the homogeneous chromosomes \([0, 0, 0]\) and \([1, 1, 1]\) appear in the population. The homozygotic genotypes \([0, 0, 0] + [0, 0, 0]\) and \([1, 1, 1] + [1, 1, 1]\), corresponding to phenotypes \( u = 0 \) and \( u = 1 \), respectively, have superior fitness to the intermediate ones, owing to the strong trade-off. In combination with assortative mating, this fitness advantage allows the homozygotes to outcompete the intermediate genotypes, while the production of new hybrids is prevented by assortative mating. In figure 2a, speciation occurs around \( t = 8,800 \) generations, resulting in two specialized and reproductively isolated populations.

Figure 2b shows the adaptive dynamics in a smaller lake \((V = 30)\). The population quickly converges to the EBP, but no successful branching occurs until \( t \approx 45,000 \). The average population size at \( \dot{u} = 0.5 \) equals \( 18V = 900 \) individuals. The subsequent coexistence of three phenotypes indicates a dimorphism at a single locus only. Despite considerable drift of the branches, the single-locus dimorphism appears stable and persists for more than \( t \approx 50,000 \) generations. Two effects of small population size can be recognized that have been identified as causes of delayed evolutionary branching. First, before branching, the average trait wobbles around the predicted EBP (random genetic drift), resulting in prolonged periods with directional rather than disruptive selection. Second, incipient branches frequently become extinct (arrows, fig. 2b). The figure thus illustrates, for a sexual population, that evolutionary branching is delayed in small populations, as shown by Claessen et al. [2007] for asexual populations.

The figure also illustrates aspects that are unique to sexual reproduction and multilocus evolution. First, speciation depends on the evolutionary branching of all loci, and thus, for multilocus traits, the delay is expected to be longer than for single-locus traits. This is illustrated in figure 2b, where successful branching of one locus is followed by a long delay (with considerable genetic drift) until the next branching event, which is followed by the extinction of an incipient branch (third arrow). Second, in sexual populations, phenotypic variation can evolve without speciation, in the form of a genetic polymorphism at one or more loci (fig. 2b). The population remains panmictic, while the polymorphism is maintained by sustained disruptive selection. The homozygotic types are specialists, while the heterozygotes are generalists. The latter have inferior fitness and are maintained in the population exclusively due to the Mendelian hybrid formation. Such a stable genetic polymorphism can occur even if a dimorphism has evolved at all loci. That is, the prespeciation phase (e.g., the seven coexisting phenotypes in fig. 2a) can last forever if the sorting of alleles into homogeneous chromosomes does not happen, which may be due to weak disruptive selection or to strong recombination (see “Trade-off curvature” in the following subsection).

Effect of Model Parameters on the Speciation Delay

Lake size. To investigate systematically the effect of lake size, we run 20 simulations of \( 10^5 \) generations, for a range of values of \( V \) and starting with 10 individuals of which all alleles have value 0.1, that is, \( x_i = y_i = 0.1 \) for all \( k \). For each run, the approach time \( t_s \) is noted (defined as the time elapsed until the mean of the phenotype distribution is close to the singular point, i.e., \( \hat{u} > 0.95u^* \)). Speciation is defined as the moment that two discrete, reproductively isolated clusters of individuals have formed (cf. fig. 2a). The time of speciation is denoted by \( t_c \). The algorithm used is analogous to the one used for evolutionary branching in Claessen et al. 2007. The delay to speciation, denoted by \( \Delta_s \), is defined as the time elapsed between approaching \( u^* \) and the moment of speciation: \( \Delta_s = t_s - t_c \).

For the case of three loci with two different settings of the mutation parameters, figure 3a, 3b shows that the delay to speciation is very sensitive to \( V \). There appears to be a minimum lake size below which speciation does not occur and above which \( \Delta_s \) decreases steeply with \( V \) until an asymptotic minimum delay has been reached. The approach time \( t_s \) decreases much less steeply with \( V \). We find the same pattern for different values of the mutation rate \( \mu_k \) and mutation width \( \sigma \) (fig. 3a, 3b). Qualitatively, this result does not depend on the chosen number of loci or the specific level of crossing over (see “Recombination and
Delayed Evolution of Polymorphism and Speciation

Figure 3: Delay to speciation depending on lake volume $V$ (a, b) or the crossing-over probability $p_C$ (c, d), with three loci ($L = 3$). For each simulation, the delay to approach $u^*$ ($t_a; \oplus$ symbols) and the delay to speciation ($\Delta_s; \bullet$ filled circles) are plotted. Parameters: $a_i = 0, a_2 = 2, b_i = -2, a, p_C = 0.1, p_M = 0.01, \sigma = 0.01$, $b, p_C = 0.1, p_M = 0.001, \sigma = 0.05$, $c, V = 50, p_M = 0.01, \sigma = 0.01$, $d, V = 50, p_M = 0.001, \sigma = 0.05$. Note the equilibrium population size of a monomorphic population with equals $\hat{u}$. These results show that our earlier results on delayed evolutionary branching in asexual populations (Claessen et al. 2007) extend to speciation in sexual populations.

Genetic polymorphisms may evolve long before eventual speciation (fig. 2b). To complete the analysis of the effect of lake size, we record in each simulation the first time, denoted by $t_P$, that the alleles $x_i = 0$ and $y_i = 1$ coexist in the population at any locus $i$. For example, $t_P = 42,871$ generations in figure 2b, just after the moment where three distinct phenotypes appear. The relation of $t_P$ with lake size is depicted in figure 4. The figure confirms that in general, genetic polymorphism evolves long before speciation. In intermediately sized systems (e.g., $V = 30$), the population may become polymorphic fairly quickly but remain polymorphic almost indefinitely without actually speciating into reproductively isolated populations.

Recombination and number of loci. For the case of three loci with two different settings of the mutation parameters, figure 3c, 3d shows that the delay to speciation increases at an accelerating rate with the probability of recombination. Increased crossing over tends to destroy the homogeneous chromosomes ([0, 0, 0] and [1, 1, 1]) and hence decreases the probability that these genotypes come to dominate the population. The approach time $t_a$, on the other hand, is insensitive to $p_C$. We find the same pattern for different values of the mutation rate $p_M$ and mutation width $\sigma$ (fig. 3c, 3d).

The effect of sexual reproduction on speciation is expected to depend on both the recombination probability $p_C$ and the number of loci $L$. First, the chance of crossing over increases with both $p_C$ and the number of loci. Second, in a polymorphic population, the frequency of extreme phenotypes decreases with $L$. Third, speciation requires the evolution of polymorphism at all loci, which may take longer with more loci. To quantify the combined effect of crossing over and the number of loci, 20 simu-
lutions of 20,000 generations were run for each combination of \( p_c \) and \( L \) and for four different lake volumes. For each parameter combination, the frequency of speciation was noted (fig. 5). The figure shows that increasing either one of these two parameters can impede speciation, even if the system size \( V \) is large. Only in simulations with up to three loci is speciation frequently observed. Figure 5 also shows that the qualitative effect of \( L \) and \( p_c \) is independent of lake volume, although the frequency of speciation depends on lake volume. By contrast, the evolution of polymorphism is not significantly affected by the crossing-over probability (fig. A2c).

Trade-off curvature. The strategic (simplified) nature of our model does not allow for a close parameterization to the ecology of a specific empirical case such as the arctic char. Yet certain qualitative consequences of parameterization can be assessed with the model. In particular, the parameterization of the trade-off between search rates in the two habitats may influence the results. Here, we concentrate on the effect of the curvature of the trade-off, which influences the curvature of the fitness function at the EBP (i.e., assuming a monomorphic resident population with trait \( u = u^* \)). A stronger, positive curvature of the trade-off function (and hence of the fitness function) corresponds to stronger disruptive selection and is expected to promote evolutionary branching. A flatter function is expected to result in longer delays.

In order to assess the effect of curvature, we use six different trade-offs by tuning the parameters of the search rate functions (eqq. [4], [5]). The different trade-offs are assumed to be symmetric (i.e., \( b_i = -b_i \) and \( a_i = a_i \)) and are defined such that \( A_i(u) \) and \( A_j(u) \) intersect at the point \( A_j(0.5) = A_j(0.5) = 1 \). At the EBP, the ecological equilibrium \((N^*, F^*, F^*)\) is then the same for all trade-offs, the only difference being the curvature of the fitness function. Defining fitness as the per capita growth rate, \( w(u) = \beta(u) - \mu \), the curvature of the fitness function is \( \partial^2 w(u)/\partial u^2 = 2b_iF_i^* - 2b_iF_i^* \). The trade-offs are specified by the parameter pair \((a_i, b_i)\), equal to \((0, 2), (0.2, 1.6), (0.4, 1.2), (0.6, 0.8), (0.8, 0.4), \) and \((1.0, 0)\), respectively. Given the other parameter values, the curvature at the EBP then equals \(0.8, 0.64, 0.48, 0.32, 0.16, \) and \(0\), respectively. Note the trade-off \((a_i, b_i)\), equal to \((0, 2)\), corresponds to the one used in the other figures.

We run 30 simulations of 20,000 generations for each trade-off, with \( L = 3 \) and \( p_c = 0.1 \). We find a strong effect of curvature on the delay to speciation because speciation is observed only with the highest curvature (0.8). A more subtle effect of curvature is revealed by observing the highest level of polymorphism (number of dimorphic loci; i.e., with alleles 0 and 1) attained during a simulation, which can be estimated from the number of discrete coexisting phenotypes, as explained above. Figure 6 shows that the level of polymorphism increases with the curvature of the fitness function. With a relatively strong curvature of 0.64, we find stable dimorphism in all three loci in all 30 simulations but no speciation. Apparently, the fitness differences are not sufficient for the fully homozygous to oust the hybrids. With intermediate curvature, we find dimorphism in either two or three loci (0.48) or in either one or two loci (0.32). With the linear trade-off (0 curvature), we find incipient branching resulting in a single dimorphic locus in two out of 30 runs. However, the dimorphisms last only briefly.

Close inspection of the dynamics at the allele level shows that with low curvature, branching occurs initially at all the loci but that polymorphism is subsequently lost at some or all loci (data not shown). This usually occurs during the initial period of phenotypic divergence, that is, before the appearance of the \( 2k + 1 \) discrete phenotypes. The subsequent reduced polymorphism is very stable, such as the single-locus dimorphisms observed in figure 2b.

Part 2: An Empirical Test

Material and Methods

Table 1 was modified from Alekseyev et al. (1998, 1999, 2002) and supplemented with new information about additional lakes (S. Alekseyev, personal communication). The table shows data on 22 lakes in Transbaikalia, Russia. Lake volume was estimated as the product of maximum length, maximum width, and maximum depth of the lake.
The number of char types was counted as the total number of morphotypes ever found. For the analysis, the independent factors were $\log(1 + x)$ transformed (where $x$ is the number of other fish species or lake volume).

We used ordinal logistic regression (SPSS v12.0.1) in order to test for relationships between lake volume, number of other species, and number of char morphs. First, we analyzed the relation between the number of char forms versus lake volume. Second, since the number of other species present can negatively affect the chance that new char morphs develop (as fewer niches remain unexploited when more species are present), we analyzed the relation between the number of char morphs versus the number of other species present in the lake. Third, we included both factors simultaneously in the analysis, with the number of char morphs as dependent and the number of other species present and lake volume as independent factors.

Results of Data Analysis

The number of char morphs does not show a significant relationship with lake volume only (pseudo-$R^2 = 0.13$, $P = .21$) or with only the number of other species present (pseudo-$R^2 = 0.03$, $P = .71$). Lake volume and the number of other species together, however, have a significant effect on the number of char morphs (pseudo-$R^2 = 0.66$, $P < .001$). The number of other species has a negative effect (Wald statistic $=-9.9$, $P = .001$), while lake volume has a positive effect (Wald statistic $=2.8$, $P = .009$) on the number of char morphs.

Discussion

With a simple model, we have shown that our previous results (Claessen et al. 2007) extend to sexually reproducing populations: evolutionary branching (resulting in ei-
Figure 6: Maximum number (out of $L = 3$) of dimorphic loci, that is, with alleles 0 and 1 present, observed in simulations of 20,000 generations, as a function of the curvature of the fitness function (evaluated at the evolutionary branching point). Dots = averages in 30 simulations. Error bars indicate the lowest and highest observations. Speciation occurred in all simulations with the highest curvature (0.8, the same as fig. 2a) but in none of the others. For search rate parameters, see text. Other parameters: $V = 200$, $S = 2$, $\rho_m = 0.01$, $\sigma = 0.01$.

Quantitatively, our results depend on model parameters, including the number of loci, the level of recombination, mutation probability, and the size of mutation steps (fig. 3a, 3b; see also Claessen et al. 2007). Without knowledge of these parameter values, it is not possible to pinpoint a lower limit of absolute population size below which branching is expected to be delayed or impossible. We can, however, make two qualitative predictions. First, in a given species, resource polymorphism (whether the result of speciation or a genetic polymorphism) is more likely to occur in large lakes than in small lakes, since absolute population size is expected to be proportional to lake size (Søndergaard et al. 2005). Second, given that the morphological trait underlying the resource polymorphism is most likely a multilocus trait (Skúlason and Snorrason 2004), our model further predicts that the extent of reproductive isolation in observed cases of resource polymorphisms depends on lake size, with complete reproductive isolation and hence speciation underlying the resource polymorphism in large lakes, while in intermediate lakes the resource polymorphism is more likely to be a genetic polymorphism in a panmictic population (fig. 4).

The first prediction is confirmed by our analysis of empirical data on arctic char polymorphism in 22 lakes in Transbaikalia that showed that, indeed, the number of
Table 1: Data on 22 lakes in Transbaikalia (Russia) with the number of arctic char morphotypes and the number of other fish species found in each lake

<table>
<thead>
<tr>
<th>Lake</th>
<th>Volume (km³)</th>
<th>No. other species</th>
<th>No. char morphs</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frolikha</td>
<td>3.6</td>
<td>10</td>
<td>1</td>
<td>Alekseyev et al. 1999 and references therein</td>
</tr>
<tr>
<td>Nomama</td>
<td>.1</td>
<td>5</td>
<td>2</td>
<td>Alekseyev et al. 1999 and references therein</td>
</tr>
<tr>
<td>Ogiendo-1*</td>
<td>.008</td>
<td>2</td>
<td>2</td>
<td>Alekseyev et al. 1998, 2002; and S. Alekseyev, pers. comm.</td>
</tr>
<tr>
<td>Ogiendo-3*</td>
<td>.003</td>
<td>3</td>
<td>1</td>
<td>Alekseyev et al. 1999 and references therein</td>
</tr>
<tr>
<td>Kudushkit*</td>
<td>.005</td>
<td>0</td>
<td>3</td>
<td>Alekseyev et al. 1999 and references therein</td>
</tr>
<tr>
<td>Soli</td>
<td>.27</td>
<td>3</td>
<td>3</td>
<td>Alekseyev et al. 1998, 2002; and S. Alekseyev, pers. comm.</td>
</tr>
<tr>
<td>Irbo</td>
<td>.13</td>
<td>4</td>
<td>2</td>
<td>Alekseyev et al. 1999 and references therein</td>
</tr>
<tr>
<td>Padorinskoe</td>
<td>.0024</td>
<td>1</td>
<td>2</td>
<td>Alekseyev et al. 1999 and references therein</td>
</tr>
<tr>
<td>Davatchanda*</td>
<td>.0055</td>
<td>2</td>
<td>2</td>
<td>Alekseyev et al. 1999 and references therein</td>
</tr>
<tr>
<td>Krestaki-1*</td>
<td>.013</td>
<td>0</td>
<td>3</td>
<td>Alekseyev et al. 1999 and references therein</td>
</tr>
<tr>
<td>Bol’shi Nararik</td>
<td>.98</td>
<td>4</td>
<td>3</td>
<td>Alekseyev et al. 1999 and references therein</td>
</tr>
<tr>
<td>Dzhelo</td>
<td>.011</td>
<td>2</td>
<td>2</td>
<td>Alekseyev et al. 1998, 2002; and S. Alekseyev, pers. comm.</td>
</tr>
<tr>
<td>Leprindokan</td>
<td>.42</td>
<td>8</td>
<td>2</td>
<td>Alekseyev et al. 1999 and references therein</td>
</tr>
<tr>
<td>Bol’shoe Leprindo</td>
<td>2.1</td>
<td>11</td>
<td>2</td>
<td>Alekseyev et al. 1999 and references therein</td>
</tr>
<tr>
<td>Gol’tsovo*</td>
<td>.004</td>
<td>0</td>
<td>3</td>
<td>Alekseyev et al. 1999 and references therein</td>
</tr>
<tr>
<td>Davatchan</td>
<td>.28</td>
<td>5</td>
<td>3</td>
<td>Alekseyev et al. 1999 and references therein</td>
</tr>
<tr>
<td>Severonichatskoe*</td>
<td>.01</td>
<td>3</td>
<td>2</td>
<td>Alekseyev et al. 1998, 2002; and S. Alekseyev, pers. comm.</td>
</tr>
<tr>
<td>Kiryalta-3*</td>
<td>.01</td>
<td>3</td>
<td>2</td>
<td>Alekseyev et al. 1998, 2002; and S. Alekseyev, pers. comm.</td>
</tr>
<tr>
<td>Kiryalta-4</td>
<td>.07</td>
<td>3</td>
<td>2</td>
<td>Alekseyev et al. 1999 and references therein</td>
</tr>
<tr>
<td>Kamkanda</td>
<td>.06</td>
<td>3</td>
<td>3</td>
<td>Alekseyev et al. 1998, 2002; and S. Alekseyev, pers. comm.</td>
</tr>
<tr>
<td>Lesha*</td>
<td>.0004</td>
<td>1</td>
<td>1</td>
<td>Alekseyev et al. 1999 and references therein</td>
</tr>
<tr>
<td>Tokko</td>
<td>.03</td>
<td>1</td>
<td>3</td>
<td>Alekseyev et al. 1999 and references therein</td>
</tr>
</tbody>
</table>

Note: Lakes with names not specified in topographic maps are marked with an asterisk. Numbers following the names of the lakes correspond to the number of the lake in a chain of lakes located in the upper reaches of the same river, starting from the upper one. See text for estimation of volume.

morphotypes is positively related to lake size. In addition, the data analysis revealed a negative relation with the number of other fish species present. We hypothesize that the presence of other species may reduce the likelihood of evolutionary branching in two ways. First, additional species reduce the availability of niches and hence limit the maximum level of diversification. The presence of another fish species in one of the available niches reduces the resource associated with that niche and hence changes the curvature of the fitness function, resulting in directional selection toward specialization in the other niche. This idea could be investigated theoretically by extending the model analysis to multiple species, but from general theory we already know that the number of resources is an upper limit to the level of diversity (including the other species) that can evolve (Tilman 1982; Meszéna and Metz 1999). Second, the presence of a predator species would not affect the relative abundance of the two resources but rather reduce the population size of the focal species. Our results suggest that a predator thus decreases the likelihood of evolutionary branching in its prey by reducing its absolute population size. This suggests that higher-dimensional environments (e.g., taking into account the interactions with competing and predatory fish) may modulate the relationship that the model has revealed; in both cases, evolutionary branching is inhibited by the presence of other species. A negative relation between the number of other species and the number of morphotypes is, however, consistent with our hypothesis of a sympatric origin of the morphotypes, depending on lake size.

It should be stressed, however, that alternative hypotheses may lead to the same prediction. First, if the probability of invasion of a lake increases with lake size, the hypothesis of multiple invasions of lakes by allopatrically diverged morphotypes would lead to the observed correlation between lake size and diversity. Second, it can be argued that larger lakes are likely to harbor more available niches or habitats and may therefore support higher levels of diversity. The data (table 1) therefore cannot differentiate between these hypotheses. However, analysis of meristic data from a subset of these lakes suggests that different sympatric morphotypes are more closely related than allopatric populations of the same morphotype, thus supporting a sympatric origin of morphotypes (Alekseyev et al. 2002). This observation seems to rule out the invasion hypothesis but leaves the second alternative hypothesis intact.
Adaptive Dynamics: Theory and Data

Mathematical models of ecological systems that can give rise to evolutionary branching, such as our model, abound in the literature and cover various ecological interactions such as resource competition, predation, interference competition, mutualism, parasitism, and competition for mates (Geritz et al. 2004 and references therein). Yet a largely unexplored aspect of adaptive dynamics theory is the confrontation of its predictions with empirical evidence. The recent volume edited by Dieckmann et al. (2004) makes an effort in this direction. It contains examples of systems in which important assumptions of the models of adaptive dynamics are fulfilled, such as mechanisms that give rise to frequency-dependent selection and mechanisms of assortative mating. Microbial systems have a large potential for rigorously testing theoretical predictions of adaptive dynamics (Travisano 2004), as demonstrated by Friesen et al. (2004). Indirect evidence for validation of the theory can be obtained from phylogeographic data, because certain patterns that are hard to explain assuming allopatric speciation are easily explained with sympatric speciation (e.g., monophyletic fish species in crater lakes; Schliewen et al. 1994; Tautz 2004). We view our study as a contribution in this direction. We identified an abiotic, ecological factor (lake size) that modifies the likelihood of evolutionary branching. The advantage of varying an abiotic factor such as lake size is that it does not coevolve with the life history under consideration. Our model predictions allow for straightforward testing with empirical data as demonstrated above, although not all alternative hypotheses can be ruled out. We argue that prediction and verification of such patterns of variation in evolutionary outcomes over ecological gradients constitutes a promising approach in exposing theory to empirical scrutiny. In this context, it is interesting to note that gradients in other factors that influence absolute population size, such as productivity, may equally influence evolutionary branching.

Acknowledgments
S. Alekseyev is gratefully acknowledged for providing us with data on Transbaikalian lakes. We thank N. Champagnat, U. Dieckmann, A. Lambert, S. Legendre, and J. A. J. Metz for discussions on the evolutionary branching in stochastic populations. D.C. and A.M.d.R. acknowledge financial support from the Dutch Science Foundation (NWO), and L.P. acknowledges support from the Swedish Research Council (VR). Two anonymous reviewers are kindly thanked for their constructive comments.

APPENDIX A
The Effect of Assortative Mating ($S$) on Polymorphism and Speciation

The strength of assortative mating is determined by the parameter $S$ (eq. [2]). To investigate the robustness of our results to the value of $S$, we have run a large number of additional simulations for constructing plots such as figure 5 for different values of $S$. Figure A1 thus shows the frequency of speciation in 20 simulations of 20,000 generations for each combination of $p_C$ and $L$ for a fixed lake size of $V = 50$. As expected, the figure shows that with random mating ($S = 0$) and weak assortative mating ($S = 1$), speciation does not occur (within 20,000 generations), while with strong assortative mating ($S = 2$ and $S = 4$) speciation is possible. Note that figure A1c corresponds to figure 5b.

By contrast, figure A2 shows that the evolution of polymorphism is unaffected by the level of assortative mating. As before, the time to polymorphism ($t_p$) is recorded in each simulation as the first time that the alleles $x_i = 0$ and $y_i = 1$ coexist in the population at any locus $i$. Based on figure A2, we can conclude that the evolution of polymorphism is not influenced by $S$ (see also app. B) nor by the crossing over probability $p_C$. Polymorphism evolves more quickly with a higher number of loci $L$, which is due to our model assumption that the size of mutations increases with $L$, in order to keep the phenotypic effect of mutations constant.
Figure A1: Effect of assortative mating (S) on the frequency of speciation in 20 runs of 20,000 generations. For each combination of the number of loci and the crossing-over probability, the color indicates the number of simulations (out of 20) in which speciation occurred. $V = 50$, $p_{ai} = 0.001$, $\sigma = 0.05$. 
Figure A2: Effect of assortative mating ($S$) on the evolution of polymorphism in 20 runs of 20,000 generations. For each combination of the number of loci and the crossing-over probability, the color indicates the number of simulations (out of 20) in which speciation occurred within 1,000 generations. $V = 50$, $p_u = 0.001$, $\sigma = 0.05$.

APPENDIX B

Evolution of Assortative Mating ($S$)

Figure B1 shows an example of the simultaneous evolution of the ecological trait $u$ and the mating trait $S$ for a lake volume of $V = 50$ and $L = 3$ loci for the ecological trait. The parameter $S$ is assumed to be determined by a single locus subject to the same mutation rate as the ecological trait. In this run, the allowed range of $S$ has been restricted to between $-2$ and $+2$. The figure shows that after a short delay, evolutionary branching occurs despite mating being random ($S \approx 0$, cf. eq. [2]). Evolution of dimorphism at both loci results in a (still diverging) genetic polymorphism. With two dimorphic loci, say (A, a) and (B, b), with unequal high and low values (i.e., $A \neq B$, $a \neq b$), the possible allele combinations result in a total of nine discrete phenotypes (i.e., aabb, Aabb, aaBb, etc.), which are visible in figure B1. However, the emerged polymorphism favors genotypes that mate assortatively, which results in directional selection at the $S$ locus. The trait $S$ converges to its allowed maximum of 2. In this particular run, speciation occurs at around 10,000 generations, when the ecological trait has diverged completely and assortative mating has evolved. Subsequently, drift at the $S$ locus to lower values results in a brief periods of interbreeding, marked by the appearance of intermediate hybrids. An open question is what is the effect of population size on the evolution of assortative mating. Small population size may favor the appearance of the linkage disequilibrium which is required for the evolution of reproductive isolation by assortative mating (Dieckmann and Doebeli 1999).
Figure B1: Simultaneous evolution of the polygenic ecological trait $u$ (top) and the assortative mating trait $S$ (bottom), in a single run of 20,000 generations. Parameters: $L = 2$, $V = 50$, $p_c = 0.1$, $p_m = 0.01$, $a_1 = 1$, $a_2 = 2$, $b_1 = 1$, $b_2 = -1$. Density of individuals: black = 0, blue = low, and red = high.

Literature Cited


Associate Editor: Troy Day
Editor: Michael C. Whitlock
Allele fixation in a dynamic metapopulation: Founder effects vs refuge effects

Robin Aguilée a,*, David Claessen a, Amaury Lambert b

a Laboratory of Ecology and Evolution (UMR 7625, University Paris 06, Ecole Normale Supérieure, AgroParisTech, CNRS), Unit of Eco-Evolutionary Mathematics, F-75005 Paris, France
b Laboratoire de Probabilités et Modèles Aléatoires, University Paris 06, F-75005 Paris, France

ARTICLE INFO

Article history:
Received 18 November 2008
Available online 21 May 2009

Keywords:
Allele fixation probability
Time to fixation
Subdivided population
Dynamic landscape
Bottleneck
Fragmentation
Fusion
Founder effect
Refuge effect
Variance effective size
Coalescent effective size

ABSTRACT

The fixation of mutant alleles has been studied with models assuming various spatial population structures. In these models, the structure of the metapopulation that we call the “landscape” (number, size and connectivity of subpopulations) is often static. However, natural populations are subject to repetitive population size variations, fragmentation and secondary contacts at different spatiotemporal scales due to geological, climatic and ecological processes. In this paper, we examine how such dynamic landscapes can alter mutant fixation probability and time to fixation. We consider three stochastic landscape dynamics: (i) the population is subject to repetitive bottlenecks, (ii) to the repeated alternation of fragmentation and fusion of demes with a constant population carrying capacity, (iii) idem with a variable carrying capacity. We show by deriving a variance, a coalescent and a harmonic mean population effective size, and with simulations that these landscape dynamics generate repetitive founder effects which counteract selection, thereby decreasing the fixation probability of an advantageous mutant but accelerate fixation when it occurs. For models (ii) and (iii), we also highlight an antagonistic “refuge effect” which can strongly delay mutant fixation. The predominance of either founder effects or refuge effects determines the time to fixation and mainly depends on the characteristic time scales of the landscape dynamics.

© 2009 Elsevier Inc. All rights reserved.

1. Introduction

The study of the fixation of novel alleles has known many developments since the beginning of population genetics (Fisher, 1922; Haldane, 1927; Wright, 1931). Fixation probabilities and times to fixation are indeed important factors influencing, among others, the rate of evolution, the genetic load (Whitlock, 2002; Theodorou and Couvet, 2006), and the level of genetic diversity (Vuilleumier et al., 2008). The importance of understanding and characterizing allele fixation is linked to its practical implications: for example, conservation generally tries to restore genetic diversity in small and/or fragmented populations which risk extinction (Gao and Zhang, 2005; Bohme et al., 2007); in public health, maintenance of resistance alleles to drugs is a major problem (Heinemann, 1999; McLean, 1995).

Most natural populations are subdivided into partially isolated demes (Hanski and Gaggiotti, 2004). Following Keymer et al. (2000) we call the spatial structure of a subdivided population the “landscape”; we define it as the number, the size, and the connectivity of subpopulations. The landscape strongly affects how drift and selection act (Barton and Whitlock, 1997; Colas et al., 2002; Roze and Rousset, 2003; Whitlock, 2004). It thus influences allele fixation probability and time. Understanding these influences is of great importance especially today because of intense landscape fragmentation due to human activities; many populations consist now of small demes poorly connected, leading to high local extinction risk (Wilcox and Murphy, 1985; Hanski and Gaggiotti, 2004).

There is an abundant literature about mutant fixation in subdivided populations (see e.g. the review of Charlesworth et al., 2003; Patwa and Wahl, 2008). Many spatial structures have been analyzed, in particular island, stepping-stone, spatially continuous, source–sink, and extinction–recolonization models. For populations of constant size such that migration does not change allele frequencies in the whole population, spatial structure does not affect allele fixation probability. Other spatial structures generally decrease the fixation probability of advantageous mutants. The landscape described by most of these models is static, or at most only one component of the landscape is varying. First, the number of patches is constant over time. Second, the size of demes is often considered as constant. Many authors analyzed population size variations (one size change, exponential/logistic growth or decline, size fluctuation), but only for one isolated population (see for example Ewens, 1967; Kimura and Ohta, 1974; Otto and Whitlock, 1997; Barton and Whitlock, 1997; Wahl and Gerrish, 2000).
2001; Iizuka, 2001; Iizuka et al., 2002; Heffernan and Wahl, 2002; Lambert, 2006). Note that extinction–recolonization models could be considered as models with population size variations since each deme can become extinct. Third, the connectivity of subpopulations via migration is assumed constant over time, except in Whitlock and Barton (1997) and Whitlock (2003).

However, all components of the landscape are dynamic simultaneously in natural populations. For example, external factors can cause variations of connections between demes, to the point where connectivity either falls to its minimum (unconnected demes, e.g., vicariance) or rises to its maximum (fusion of demes, e.g., postglacial scopic/contacts) (Young et al., 2002). Climatic variations as well as volcanic events can cause sea level changes resulting in separations and fusions of islands (Cook, 2008). Repeated changes of the water level causing fragmentation and fusion of lakes are known in the Great African Lakes (Owen et al., 1990; Delvaux, 1999; Galis and Metz, 1998; Stiaussyn and Meyer, 1999). At a different spatial-temporal scale, the number and size of populations can vary because of dispersal and recolonization events (establishment of new colonies and their later fusion) (DeHeer and Kamble, 2008; Vasquez and Silverman, 2008). All aspects of the spatial structure of a population can change because of new ecological interactions, e.g., the emergence or extinction of a predator or parasite (Batzli, 1992). Contemporary fragmentation of habitat due to human action is also always changing the landscape (Davies et al., 2006).

These spatial processes cause, repeatedly, bottlenecks and fragmentation of subpopulations. These two phenomena are well known, but have been studied separately and, most of the time, when occurring only once. Their association and their repetition have no simple outcome regarding allele fixation: bottlenecks and fragmentation are expected to decrease the fixation probability of a beneficial allele (Otto and Whitlock, 1997; Wahl and Gerrish, 2001; Whitlock, 2003), but they can increase or decrease the time to fixation, in particular depending on the effective size of the population (Whitlock, 2003). Moreover, to keep the number of demes of a fragmenting population constant, models generally assume repetitive extinctions. However, the spatial processes listed above do not necessarily lead to repetitive local extinctions. They can also lead, repeatedly, to the fusion of entire subpopulations. To our knowledge, such periodic fusions (repetitive secondary contacts) have not yet been studied regarding allele fixation, except in Jesus et al. (2006).

In this paper, we examine how such dynamic landscapes can alter fixation probability and time to fixation of a mutant allele, with or without selection. We consider three landscape dynamics: a population subject to repetitive bottlenecks (Model 1) and a population subject to the repeated alternation of fragmentation and fusion of demes (Model 2), that is, alternatively divided into two demes or undivided, with population size variations but a constant carrying capacity (Model 2a) and with a variable carrying capacity (Model 2b). Note that Wahl and Gerrish (2001) examined the effects of cyclic bottlenecks in experimental conditions, i.e., regular and severe bottlenecks. In contrast, we take into account the stochasticity of the occurrence of bottlenecks and any intensity of bottlenecks. We derive diffusion approximations based on the assumption of a large population. Depending on the characteristic time scales of the landscape dynamics, our models can mimic each of the spatial processes listed above. Our results constitute a first step in analyzing the rate of evolution, and then speciation, in dynamic landscapes.

2. The models

2.1. Within-deme population dynamics

We use a population genetics haploid model with two types, mutants and residents, representing individuals carrying two different alleles, respectively. This model, referred to as the Moran model or Moran process (Moran, 1962), is embedded into a model of landscape dynamics, specified below. The Moran process is similar to the Wright–Fisher model (Wright, 1931), but in continuous time (overlapping generations). It is a stochastic process which describes a finite population of constant size and based on the following mechanism: during an infinitesimal time interval, a birth or death event can occur or not; if it does, the population at time \( t + dt \) is updated from that of time \( t \) by randomly selecting an individual to reproduce and then, independently, randomly selecting an individual to be removed. Each individual with birth rate \( b \) has a probability \( b \ dt \) to reproduce during \( dt \).

Each resident reproduces at rate \( b = 1 \) and each mutant at rate \( b = 1 + s \) where \( s \) is its selective advantage (see Table 1 for a summary of the notation). For an undivided isolated population whose allele frequency fluctuates via a Moran process, classical results and approximations are known for the fixation probability and time to fixation and will be used as reference results of unstructured populations in a static landscape (Wright, 1931; Kimura, 1962; Kimura and Ohta, 1969; Ewens, 2004).

### Table 1

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Numerical values used:</th>
</tr>
</thead>
<tbody>
<tr>
<td>( s ) Selective advantage of mutants</td>
<td>From (-0.25) to (0.25)</td>
</tr>
<tr>
<td>( g ) Initial frequency of mutants</td>
<td>From (0.001) to (0.1)</td>
</tr>
<tr>
<td>( d ) Bottleneck rate</td>
<td>From (0.001) to (1)</td>
</tr>
<tr>
<td>( f ) Intensity of bottlenecks</td>
<td>From (0.001) to (0.99)</td>
</tr>
<tr>
<td>( p ) Time scale of the landscape dynamics</td>
<td>From (0.5) to (0.99) (symmetrical to (p))</td>
</tr>
<tr>
<td>( N ) Carrying capacity at state 1 (undivided)</td>
<td>From (50) to (1000)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Outputs</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>( U ) Fixation probability of a mutant allele</td>
<td></td>
</tr>
<tr>
<td>( T ) Time to fixation of a mutant allele, conditional on its fixation</td>
<td></td>
</tr>
</tbody>
</table>

**Fig. 1.** Model 1, repeated bottlenecks. Model 1 describes landscape dynamics which consist of repeated bottlenecks. Bottlenecks occur at rate \( g \). Each individual dies with probability \( d \) during a bottleneck. The size of the population is indicated at each step. After each bottleneck, the population reaches its carrying capacity \( N \) via a pure birth process. Between bottlenecks, mutant allele frequencies fluctuate via a Moran process.

2.2. Model 1: Repeated bottlenecks

Model 1 consists of a population which undergoes repeated decreases in population size (Fig. 1). We are mostly interested in bottlenecks, that is, severe reductions in population size. Bottlenecks occur stochastically at exponential rate \( g \). The higher \( g \) is, the more often a bottleneck is likely to occur. The intensity of bottlenecks is characterized by \( d \): during a bottleneck, each individual has a probability \( d \) to die; the number of surviving individuals is thus drawn from a binomial distribution. Note that we assume that the selective advantage of mutants does not convey resistance to bottlenecks: \( d \) is identical for residents and mutants.

Just after a bottleneck, we assume that the population reaches its carrying capacity (size \( N \)) instantaneously. Indeed, an initial
population of $N(1 - d)$ individuals will reach its carrying capacity $N$ in about $-\log(1 - d)$ time units, which is much smaller than the characteristic timescale of allele frequency change (about $N$ time units). We model this growth phase using a stochastic pure birth process. Between bottlenecks, the number of mutants, $X_t$, (and the mutant allele frequency $x_t$) fluctuates through a Moran process. We evaluate fixation probability $U$ and time to fixation $T$ of a mutant allele (conditional on its fixation) initially at frequency $x_0$ using simulations and diffusion approximations. Note that a bottleneck can lead to population extinction when the bottleneck intensity $d$ is high since the number of surviving individuals is stochastic. Therefore, we evaluate $U$ and $T$ conditional on the persistence of the population until the fixation of either the mutant allele or the resident allele.

Changing values of $g$ and $d$ allows us to model very different spatial (and ecological) processes. For example, a high value of $g$ ($g > 1$) with a small value of $d$ ($d < 0.2$) simulates frequent weak bottlenecks, which can correspond to periodic oscillations observed in consumer-resource systems (Turchin, 2003). In contrast, a small value of $g$ ($g < 0.01$) with a high value of $d$ ($d > 0.8$) simulates rare but strong bottlenecks: it can correspond to rare and violent climatic events such as severe fires (Malhi et al., 2008).

2.3. Model 2a: Alternations of fragmentation and fusion, constant carrying capacity

Landscape dynamics of Model 2a consist of an oscillation of the population between 1 and 2 demes (Fig. 2): (i) the population consists of one deme of $N$ individuals (state 1), (ii) the population splits into two demes (fragmentation), (iii) both demes reach their ecological equilibrium (size $N/2$), (iv) the population consists of two independent isolated demes (no migration between them) of $N/2$ individuals (state 2), (v) the two demes merge and then form one deme of $N$ individuals (fusion, return to state 1). The fragmentation–fusion dynamics can be interpreted in a second way: the number of demes is always 2, and they are either connected via enough migration to consider the two subpopulations as only one (state 1), or they are isolated from each other (no migration, state 2). Note that in both interpretations of the dynamics, no explicit spatial structure is assumed.

Fragmentations and fusions are modeled as stochastic events occurring respectively at exponential rates $f$ and $c$. The higher $f$ (respectively $c$), the more often a fragmentation (respectively fusion) is likely to occur. At a fragmentation event, we assume that each individual has a probability $p$ to be in the “left-hand” deme. The number of individuals in the “left-hand” deme is hence drawn from a binomial distribution. The closer $p$ is to 0.5, the more the two demes are likely to have the same population size just after fragmentation: $p$ thus characterizes the asymmetry of fragmentation and will be referred to as the “asymmetry parameter”.

As in Model 1, between landscape changes (here, fragmentation and fusion events), the number of mutants $X_t$ (and their frequency $x_t$) fluctuates through a Moran process. After a fragmentation, we assume that each deme reaches its ecological equilibrium instantaneously, which we model by using a stochastic pure birth process or pure death process, depending whether the population size is above or under its equilibrium. At state 2, the carrying capacity of each deme is assumed to be $N/2$: the overall carrying capacity is constant equal to $N$ because e.g. no more resources are available after a fragmentation and resources are equally divided between the two demes.

Values of $f$, $c$ and $p$ can be used to model various dynamic processes. For example, landscape changes due to human action frequently destroy and recreate suitable habitats, which can divide a population into subpopulations of very different sizes and merge other previously isolated demes. If the total area of suitable habitats stays constant, Model 2a using a high value of $f$ and $c$ ($>0.1$) associated to a value of $p$ very different from $0.5 ([p - 0.5] > 0.45)$ will be appropriate to model such fast landscape changes. In contrast, repetitive fusions and fissions of islands due to sea level changes are best simulated with a small value of $f$ and $c$ ($<0.01$) and a value of $p$ close to 0.5 ($[p - 0.5] < 0.05$). Note that this scenario is relevant when the amount of resources of each island is approximately identical because we assume that the two demes have equal carrying capacities.

2.4. Model 2b: Alternations of fragmentation and fusion, variable carrying capacity

The assumption of a constant overall carrying capacity is not relevant for all dynamic processes we aim to model, e.g. the fragmentation of a population due to the establishment of a new colony. Thus, we suggest a third landscape dynamics model, with carrying capacity variations. This Model 2b (Fig. 3) is analogous to Model 2a except that (i) a fragmentation doubles the population size since each subpopulation carrying capacity is $N$, and that (ii) the overall population size is regulated (divided by 2) just after the two subpopulations merge, using a pure death process. For this model, the population fusion followed by the reduction of its size can be for example interpreted as the movement of one subpopulation into the territory of the other because its own habitat has become unsuitable (e.g. climatic events, arrival of a new predator). The population size is then assumed to be reduced because of increased competition.

2.5. Reference model for a static landscape

Our goal is to understand how repetitive bottlenecks and cycles of alternating fragmentation and fusion affect the fixation probability $U$ and the time to fixation $T$ of a mutant allele, with or without selection. To this end, we compare results with fixation probability and time to fixation in static landscapes (i.e. one population of constant size). Let us recall some classical results in the latter case that we use as a reference case.
Using diffusion approximations for large populations, Kimura (1962) derived an expression for the fixation probability of a weakly advantageous allele in a Wright–Fisher undivided haploid population:

\[
U(x_0) \approx 1 - e^{-N x_0} - \frac{1}{1 - e^{-N x_0}}. \tag{1}
\]

Kimura and Ohta (1969) established approximations for the mean time to fixation, conditional on fixation, for a Wright–Fisher population. Fixation times in a Moran population can easily be deduced. In the advantageous case, the fixation time can be approximated by

\[
T(x_0) \approx J_1(x_0) + \frac{1 - U(x_0)}{U(x_0)} J_2(x_0) \tag{2}
\]

where \(U(x_0)\) is defined by Eq. (1).

\[
J_1(x_0) = \frac{1}{s(1 - e^{-N x_0})} \int_0^{x_0} \frac{e^{N x} - 1}{x(1 - x)} dx
\]

and

\[
J_2(x_0) = \frac{1}{s(1 - e^{-N x_0})} \int_0^{x_0} \frac{(e^{N x} - 1)(1 - e^{-N x})}{x(1 - x)} dx.
\]

The population effective sizes \(N_e\) given in the next sections are computed using a Moran population as the reference, that is, \(N\) terms should be replaced by \(N_e\) in Eq. (1) and (2) to obtain approximations of the fixation probability and time to fixation respectively.

2.6. Numerical methods

To simulate our models, we calculate the time until the next event changing allele frequencies as the expected value of this time which is the inverse of the total rate at which events occur. We then choose which event occurs according to the probability to occur at this time, which is the ratio of favorable rate over total rate. Possible events are those events modifying allele frequencies of the Moran process: the birth of a resident and the death of a mutant (occuring at rate \(N x_i^R Y_i^R / (N x_i^R + N x_j^M)\)) where \(x_i^R\) and \(x_i^M\) are the number of, respectively, mutants and residents in the deme \(i (i = 1, 2)\) at time \(t\), or the birth of a mutant and the death of a resident (at rate \((1 + s)X_i^M Y_i^M / (X_i^M + Y_i^M)\)). Other possible events are bottlenecks (at rate \(g\), only in Model 1), fragmentation of a population (at rate \(f\), only in Model 2 when the landscape is in state 1) and fusion of two subpopulations (at rate \(c\), only in Model 2 when the landscape is in state 2).

The stochastic pure birth process used in our three models is simulated similarly; possible events are the birth of a resident (at rate \(Y_i^R\)) or the birth of a mutant (at rate \((1 + s)X_i^M\)). The stochastic pure death process, used in Model 2, is analogous; residents die at rate \((1 + s)Y_i^M\) and mutants at rate \(X_i^M\). For the reasons explained above, the duration of pure birth and death processes is not taken into account for the computation of fixation times.

We estimate the fixation probability \(U\) and the time to fixation (conditional on its fixation) \(T\) of a mutant allele initially at frequency \(x_0\) by replicating each simulation 5,000,000 times for Model 1 and 1,000,000 times for Models 2a and 2b. In Model 1, \(U\) and \(T\) are evaluated conditional on the persistence of the population until fixation of one type (mutant or resident); simulations that lead to population extinction are thus ignored. In Models 2a and 2b, estimations of \(U\) and \(T\) depend on the initial state of the landscape (state 1 or 2). To compute an average value of \(U\) and \(T\), we started a proportion of the simulation replicates in state 2 equal to the ratio \(f / (f + c)\), which is the long-term proportion of time spent in state 2. Note that for Model 2b, because the total population size depends on the state of the landscape and because we start simulations with the same initial mutant frequency \(x_0\) regardless of the state of the landscape, the initial number of mutants depends on the state of the landscape. The Section 3.2.3 analyzes the results when there is initially one single mutant regardless of the state of the landscape.

We did simulations and numerical integrations using the GNU Scientific Library (The GSL Team, 2007). We plotted the results using R (R Development Core Team, 2008). Error bars give 95% confidence intervals of the estimations of \(U\) and \(T\). Table 1 gives a summary of notation, default values of parameters, and ranges of simulated values of parameters.

3. Results

3.1. Model 1: Repeated bottlenecks

3.1.1. Diffusion approximation and variance effective size

We use diffusion approximations (Kimura, 1962; Ewens, 2004) to compute the probability of fixation of an advantageous mutant allele in a large population of size \(N\) undergoing repeated bottlenecks. For large \(N\), the mutant allele frequency \(x\) makes infinitesimal changes that can be decomposed into \(d\delta x = \delta_1 x + \delta_2 x\), where \(\delta_1 x\) is the change due to genetic drift and selection, and \(\delta_2 x\) is the change due to bottleneck events. These changes occur at a rate which depends on the current state \(x\) of the mutant frequency. The resulting diffusion approximation is then characterized by its infinitesimal mean \(a(x) = a_1 x + a_2 x\) and its infinitesimal variance \(\sigma(x) = \sigma_1(x) + \sigma_2(x)\), which are the expectations of the infinitesimal changes and of their squares multiplied by their rate of occurrence. A heuristic justification of this fact can be given by the law of total probabilities applied to the different events that can occur at the first event changing allele frequencies (birth/death or bottleneck). We refer the reader to e.g. Karlin and Taylor (1981, Section 15.3) for details.

Let \(U(x_0)\) be the mutant fixation probability \((x_0 = 0)\) and \(T_0(x_0)\) the expected time to fixation on the event of mutant fixation, i.e. \(T_0(x_0) = 0\) when all mutants are lost. The expected time to fixation conditional on mutant fixation is then

\[
T(x_0) = T_0(x_0) / U(x_0).
\]

Diffusion theory then ensures that \(U\) solves the differential equation

\[
\frac{1}{2} \frac{\partial^2 U}{\partial x_0^2} + a \frac{\partial U}{\partial x_0} = 0, \tag{3}
\]

with boundary conditions \(U(x_0 = 0) = 0\) and \(U(x_0 = 1) = 1\), and that \(T_0\) solves the differential equation

\[
\frac{1}{2} \frac{\partial^2 T_0}{\partial x_0^2} + a \frac{\partial T_0}{\partial x_0} = -U(x_0), \tag{4}
\]

with boundary conditions \(T_0(x_0 = 0) = 0\) and \(T_0(x_0 = 1) = 0\). For a population of constant size \(N\) (static landscape), the solutions of these equations are respectively Eq. (1) and (2).

Let us first compute the infinitesimal mean and variance \((a_1, a_2)\) due to genetic drift and selection. Recall that the birth rate of a resident is 1 and that of a mutant is \(1 + s\). When the mutant allele frequency equals \(x\), birth–death events occur at total rate \((2 + s)Nx(1 - x)\), and with probability \((1 + s) / (2 + s)\), \(\delta_1 x = 1 / N\), whereas with probability \(1 / (2 + s)\), \(\delta_2 x = -1 / N\). Then

\[
E[\delta_1 x] = \frac{s}{N(2 + s)} \quad \text{and} \quad E[(\delta_1 x)^2] = \frac{1}{N^2}.
\]

Multiplying by the total rate, we get

\[
a_1(x) = sx(1 - x) \quad \text{and} \quad a_2(x) = \frac{(2 + s)x(1 - x)}{N}.
\]
The computation is more technical for bottlenecks. Recall that bottlenecks occur at rate \( g \), and that each individual independently survives the bottleneck with probability \( 1 - d \). Given the numbers \( N_x \) of mutants and \( N(1 - x) \) of residents, the numbers \( Z_m \) of mutant survivors and \( Z_r \) of resident survivors are independent binomial variables with probability \( 1 - d \) and respective parameters \( N_x \) and \( N(1 - x) \). By the central limit theorem, one can write

\[
Z_m \approx N_x(1 - d) + \sqrt{N_x}W_m \quad \text{and} \quad Z_r \approx N(1 - x)(1 - d) + \sqrt{N(1 - x)}W_r,
\]

where \( W_m \) and \( W_r \) are two independent centered normal variables with variance \( d \). Assuming that growth phases following a bottleneck event do not change allele frequencies significantly, the frequency change \( \delta_x \) due to the bottleneck event can then be written as

\[
\delta_x = \frac{Z_m}{Z_m + Z_r} - x \approx \frac{(1 - x)\sqrt{N_x}W_m - x\sqrt{N(1 - x)}W_r}{N(1 - d) + \sqrt{N_x}W_m + \sqrt{N(1 - x)}W_r}.
\]

Then

\[
E[\delta_x] \approx 0 \quad \text{and} \quad E[(\delta_x)^2] \approx \frac{x(1 - x)d}{N(1 - d)}.
\]

Multiplying by the total rate \( g \) yields \( (\delta_x) \) and \( \sigma_x \). Then recalling the values of \( a_1 \) and \( a_2 \), we get

\[
a(x) = sx(1 - x) \quad \text{and} \quad \sigma(x) = \frac{x(1 - x)N}{N} \left( 2 + s + \frac{gd}{1 - d} \right).
\]

Then solving Eqs. (3) and (4) assuming weak selection, i.e. \( s \) is negligible compared to 1, we get the same expressions for \( U \) and \( T \) as those in a static landscape, Eq. (1) and (2) respectively, but where all \( N \) terms are replaced by the variance effective population size defined by

\[
N_e^v = \frac{N}{1 + \frac{gd}{2(1 - d)}}. \tag{5}
\]

Another way of presenting our results is to consider the bottleneck phase as a (rescaled) time period during which the population undergoes pure drift and no selection. Indeed, the infinitesimal variance \( \sigma(x) \) was expressed as the sum of the infinitesimal variance \( \sigma_1(x) = 2x(1 - x)/N \) due to genetic drift (assuming weak selection), and of the infinitesimal variance \( \sigma_2(x) = gdx(1 - x)/(1 - d)N \) due to allele frequency change during bottlenecks. Then one can conclude that the effect of bottlenecks can be compared to that of genetic drift accelerated by a factor \( gd/2(1 - d) \). Put another way, the expected effects of bottlenecks in \( t = 1/g \) time units (i.e. on average one bottleneck) are equal to those of pure genetic drift in \( t' = d/(1 - d) \) time units during which selection would be relaxed.

### 3.1.2. Coalescent effective size

Let us now compute a coalescent effective population size. Relying on Sano et al. (2004), we define the coalescent effective size \( N_e^c \) as twice the expected coalescence time of a uniformly sampled pair of genes under neutrality (there is a factor 2 because we use a Moran population as the reference; see Section 2.6). In a population of constant size \( N \) where allele frequencies fluctuate via a Moran process with birth rate 1, the coalescence rate of two genes is \( 2/N \). Then the expectation of the coalescence time is \( N/2 \), thereby explaining the definition of \( N_e^c \).

In Model 1, two genes coalesce either during the Moran process, i.e. between bottlenecks, or during the growth phase following each population size reduction. Between bottlenecks, the population is of constant size \( N \), so that the coalescence rate of two genes is \( 2/N \). Let us now compute the probability of coalescence during the growth phase. For a population growing from size \( N_0 \) to size \( N_1 \) (\( N_1 \geq N_0 \)), let \( k_i \) be the number of descendants (in the population of size \( N_1 \)) of each individual \( i \) (in the population of size \( N_0 \)). Two genes sampled at random among \( N_1 \) have coalesced during this growth phase with probability

\[
\sum_{i=1}^{N_0} \frac{k_i(k_i - 1)}{N_1^2}.
\]

Because the variance of the number of descendants is small for a pure birth process, we can replace \( k_i \) with its expectation \( N_1/N_0 \). We can then write that two genes coalesce with probability

\[
\frac{1}{N_0} - \frac{1}{N_1}.
\]

In Model 1, the expected size of the population at a bottleneck event is \( N_0 = N(1 - d) \), and after the growth phase, the population size is \( N_1 = N \). As bottlenecks occur at rate \( g \), two genes coalesce during growth phases following bottlenecks at a rate of

\[
g \left( \frac{1}{N(1 - d)} - \frac{1}{N} \right) + \frac{2}{N}.
\]

Recalling that in Model 1, two genes coalesce either during Moran processes or during growth phases, the coalescence rate is then

\[
g \left( \frac{1}{N(1 - d)} - \frac{1}{N} \right) + \frac{2}{N}
\]

and therefore the coalescent effective population size is

\[
N_e^c = \frac{N}{1 + \frac{gd}{2(1 - d)}}. \tag{6}
\]

Using this coalescent effective size or the variance effective size Eq. (5) thus leads to the same approximation for \( U \) and \( T \).

#### 3.1.3. Comparison with harmonic mean and simulations

We can compare the previous variance/coalescent effective size to the harmonic mean effective size. Textbooks (see e.g. Wright, 1938; Kimura, 1970; Gillespie, 2004) indeed suggest that when a population is subject to rapid size variations, the harmonic mean effective size \( N_e^h \) is applicable. Let us compute it. Between bottlenecks, population size equals \( N \) for \( 1/g \) time units on average. At the time of a bottleneck, the population size shrinks instantaneously from size \( N \) to expected size \( N(1 - d) \). The population then exponentially grows to size \( N \). Assuming that each individual gives birth at rate 1, the growth phase lasts \( -\log(1 - d) \) time units. Then we get

\[
N_e^h \approx \frac{1/g - \log(1 - d)}{1/N(1 - d) + \log(1 - d)} \approx \frac{N(1 - g \log(1 - d))}{1 + \frac{gd}{2(1 - d)}}. \tag{7}
\]

Note that when \( g \) and/or \( d \) vanish (no bottleneck), all effective population sizes we have computed (Eqs. (5)–(7)) reduce to that in a static landscape, i.e. \( N_e^s = N_e^h = N_e^c = N \).

Fig. 4 shows that repeated bottlenecks decrease the fixation probability \( U \) of an advantageous mutant allele, compared to a static landscape: \( U \) decreases when the intensity of bottlenecks \( d \) increases and when bottlenecks are more frequent (higher bottleneck rate \( g \)). A bottleneck is indeed very similar to a founder event: it is a sampling event of a few individuals of the population. Thus, as we consider alleles that do not confer resistance to bottlenecks, bottlenecks generate additional genetic drift counteracting the effect of selection with an efficiency which increases with the intensity of bottlenecks \( d \) and with
their frequency $g$. Moreover, increasing $g$ decreases the expected duration between the emergence of a mutant and the first bottleneck event. Otto and Whitlock (1997) showed that the closer in time to a bottleneck event a mutant appears, the more its fixation probability is reduced. This effect is added to the accumulation of drift due to bottlenecks to reduce $U$.

Regarding the estimation of the fixation probability, Fig. 4 shows that neither the variance/coalescent effective size (Eq. (5)) nor the harmonic mean effective size (Eq. (7)) is better than the other for all bottleneck intensities and frequencies. For rare bottlenecks ($g = 0.001$), all approximations give almost identical results. The variance/coalescent effective size does better for weak bottlenecks ($d < 0.7$) than for strong bottlenecks. The derivation of the variance effective size $N_v$ (Eq. (5)) allows one to understand why: for strong bottlenecks, the assumption of small frequency variations which is necessary for a diffusion approximation to hold would not be satisfied. In particular, the expected number of survival individuals, $N(1 - d)$, can be very small, so that mutant frequency may jump from any frequency to 0 in one generation (if all mutants die during the bottleneck). This might explain why this approximation overestimates $U$ for strong bottlenecks. Nevertheless, the variance/coalescent effective size does better than the harmonic mean effective size for frequent and weak bottlenecks ($d < 0.7$ and $g = 0.1$). The harmonic mean effective size $N_h$ seems to be the most robust approximation in the sense that its fit with simulations is overall acceptable regardless of parameter values.

Fig. 5 shows that repeated bottlenecks decrease the time to fixation $T$ of an advantageous mutant allele compared to a static landscape (Eq. (2)) and that $T$ decreases with the intensity $d$ and/or the rate of bottlenecks $g$. This is due to the fact that each time a bottleneck occurs, it generates additional drift that counteracts selection, in disfavor of the mutant (regarding its fixation probability). In contrast, conditional upon fixation, drift acts in favor of the allele to fix, so that the cumulative action of drift generated by bottlenecks can strongly reduce $T$: for the range of parameters values explored in Fig. 5, $T$ can be divided by 10 compared to a population of constant size (static landscape).

Fig. 5 shows that both the variance/coalescent and harmonic mean effective sizes overestimate $T$. Sudden jumps from any frequency to 1 are possible in simulations and may explain why
thus occurs at rate $2a/N$. As fusion occurs at rate $c$, state 2 genes can also coalesce during the birth process following fragmentation at rate $cy$, where $y$ is the probability that two genes coalesce during the birth process following fragmentation. State 2 genes thus coalesce at rate $2a/N + cy$. State 2 genes can also switch to state 1 at rate $c(1 - y)$. Finally, consider a pair of state 2 genes. They cannot coalesce because they are not in the same deme. State 2' genes were thus previously necessarily state 1 genes. As fusion occurs at rate $c$, this transition occurs at rate $c$.

Knowing these coalescence rates and rates of transition between each state, we can write $\tau_i$ defined as the expected coalescence time of a pair of state $i$ genes ($i = 1, 2, 2'$). Each $\tau_i$ equals the sum of the expected time to the first event, plus, because this first event is not necessarily a coalescent event, the expected coalescence time $\tau_j$ of state $j \neq i$ genes weighted by the probability that the first event is a transition from state $i$ to state $j$ genes. Then

$$
\begin{align*}
\tau_1 &= \frac{1}{f + 2/N} + \frac{f/2}{f + 2/N} \tau_2 + \frac{f/2}{f + 2/N} \tau_1' \\
\tau_2 &= \frac{c + 2a/N}{c + 2a/N} + \frac{f/2}{c + 2a/N} \tau_1 \\
\tau_1' &= -\tau_1.
\end{align*}
$$

Let us now compute $y$, the probability that two genes coalesce during the birth process. Its expression differs between Model 2a and 2b. We have shown in Section 3.1.2 that the coalescence rate of two genes in a population growing from size $N_0$ to size $N_1$ is roughly $1/N_0 - 1/N_1$. In Model 2a, if the fragmentation is such that $p \leq 1 - p$, one deme grows from expected size $N_0 = Np$ to size $N_1 = N/2$, whereas the size of the other deme decreases (coalescence is impossible in this deme). A pair of state 2 genes is in the growing deme with probability $1/2$ and in the shrinking deme with probability $1/2$. Therefore, if $p \leq 1 - p$, $y = (1/2)[1/(Np) - 1/(N/2)].$ Similarly, if $p \geq 1 - p$, one deme grows from expected size $N(1 - p)$ to size $N/2$, the size of the other deme decreases, and state 2 genes are in each of these two demes with the same probability. In that case, $y = (1/2)[1/(N(1 - p)) - 1/(N/2)].$ Therefore, for Model 2a,

$$
y = \frac{|1 - 2p|}{2N\min(p, 1 - p)}.\quad(9)
$$

In Model 2b, after a fragmentation the sizes of both demes increase: one deme grows from size $Np$ to size $N$, the other from size $N(1 - p)$ to size $N$. As a pair of state 2 genes is in each of these demes with equal probability, the probability that they coalesce during the birth process is

$$
y' = \frac{1}{2} \left( \frac{1}{Np} - \frac{1}{N} \right) + \frac{1}{2} \left( \frac{1}{N(1 - p)} - \frac{1}{N} \right) = \frac{1 - 2p(1 - p)}{2Np(1 - p)}\quad(10)
$$

In a constantly undivided population of constant size $N_c$, two uniformly sampled genes would always be state 1 genes, with an expected coalescence time $\tau_1 = N_c/2$. Consequently, solving Eq. (8) gives the effective coalescent size for Models 2a and 2b

$$
N_c = \frac{2N(f + c) + 2af/c + 4\alpha}{2c + \alpha f + \alpha f y/2 + 4\alpha/N} \quad(11)
$$

where $\alpha = 2$ and $y$ is defined by Eq. (9) for Model 2a, and where $\alpha = 1$ and $y$ is defined by Eq. (10) for Model 2b. Replacing $N$ terms by this expression of $N_c$ into Eq. (1) and (2) should give an approximation of, respectively, the fixation probability $U$ and time to fixation $T$ of an advantageous allele. Let us first analyze the results obtained using simulations, and then compare the approximations of $U$ and $T$ obtained using $N_c$ to simulations.

3.2.2. Fixation probability of advantageous alleles

To provide some intuition on the allele dynamics in fragmentation–fusion models, Fig. 7 shows a typical time series from simulation runs with an extreme value of the asymmetry parameter ($p = 0.99$). Asymmetrical fragmentations can strongly affect allele frequencies: one newly formed deme is large (its size is close to that of the undivided population), so that just after fragmentation the mutant allele frequency in this deme is close to the overall mutant frequency just before fragmentation. The other deme is small, allowing a founder effect, as after a bottleneck in Model 1. As a result, the more asymmetrical the fragmentation, the stronger the founder effect, the more the mutant frequency in the whole population is likely to change strongly after a fragmentation, as illustrated in Fig. 7.

The fragmentation rate $f$ scales the expected time spent between fusion and fragmentation: increasing $f$ increases the frequency of fragmentation, which increases the cumulative number of possible founder events whose intensity is determined by $p$. Using our results of Model 1, we can formulate expectations based on the hypothesis that the founder effect is the principal factor in the allele dynamics. Since in Model 1 the bottleneck rate $g$ sets the cumulative number of founder events due to bottlenecks and if their intensity, we should observe the same effects of $f$ and $p$ on fixation probability in Models 2a and 2b as the effects of $g$.
and $d$ respectively in Model 1. Moreover, increasing the fusion rate $c$ increases the frequency of fusions, and thus increases the frequency of fragmentation (if a fusion occurs earlier, the following fragmentation occurs earlier). Consequently, we expect that $c$ alters fixation probabilities in the same way as $f$ does.

As this reasoning predicts, in the advantageous case, compared to a static landscape the mutant fixation probability $U$ decreases with the asymmetry of fragmentations $p$ (Fig. 8) and/or with the fragmentation rate $f$ (Fig. 9) and/or with the fusion rate $c$ (Fig. 10). The reference model is the same for Models 2a and 2b (static landscape, Eq. (1)). However, in Model 2b the overall population size fluctuates between $N$ (state 1) and $2N$ (state 2). The population size used in Eq. (1) for Model 2b is thus the harmonic mean of overall carrying capacities (Gillespie, 2004), $(f + c)/(c/N + f/(2N))$, which is greater than $N$ (i.e. the carrying capacity of the population in Model 2a at any time). As a result, $U$ is higher in Model 2b than in Model 2a (Figs. 8–10). However, the effect of the landscape dynamics relative to a static landscape is identical in both models: $U$ is reduced when the landscape dynamics are fast enough and fragmentations are asymmetrical enough.

We could use the coalescent effective size $N_c^f$ (Eq. (11)) to predict $U$. When $f$ tends to zero, i.e. when the landscape tends to be most of the time in state 1, $N_c^f$ tends to $N$. This is consistent with results of Fig. 9: for small values of $f$, the fixation probability of mutants tends to be identical to that of a static landscape. However, when $c$ tends to zero, i.e. when the landscape tends to be most of the time in state 2, $N_c^f$ tends to infinity. It means that fixation tends to become impossible when $c$ decreases. But as long as $c$ is not zero, fixation is nevertheless possible. $N_c^f$ should thus tend to the effective size of a population in a static landscape, as simulations of Fig. 10 show it. Therefore, for small values of $c$, using the coalescent effective size $N_c^f$ does not make sense to predict $U$.

### 3.2.3. State-dependent fixation probability of advantageous alleles

Figs. 8–10 give the fixation probabilities of mutants arising in state 1 or 2 averaged over the relative duration of state 1 and 2 (see Section 2.6). However, the fate of one particular mutant depends on the state of the landscape when it appears.

Fig. 11 shows that for Model 2b, a mutant is more likely to be fixed if it appears in state 1 ("state 1 mutant") than in state 2 ("state 2 mutant"). This is principally because we consider here only one initial mutant; the initial overall frequency of mutants arising in state 1 is thus twice that of those arising in state 2. Fig. 11 shows that for Model 2a without founder events ($p$ close to 0.5), the mutant fixation probability is independent of the state of the landscape when the mutant arises. In Model 2a, only fragmentations may alter the fixation probability of an advantageous mutant; fusion events have no effect on $U$. With symmetrical fragmentation, there is no population size variation, and state 1 and 2 mutant fixation probabilities are thus identical. With founder events ($p$ close to 1), a mutant is more likely to be fixed if it appears in state 2 than in state 1 (Fig. 11). Let us recall that founder events are analogous to bottlenecks and that Otto and Whitlock (1997) showed that the closer in time to a bottleneck event a mutant appears, the more its fixation probability is reduced. A state 1 mutant appears necessarily closer in time to the first fragmentation event than a state 2 mutant,
Asymmetry parameter $p$

Mutant fixation time $T$ increases:

- The difference between “fixation time $T$” and “without refuge effects” thus quantifies refuge effects. With symmetrical fragmentation ($p = 0.5$, circles), only refuge effects affect $T$. The higher the fusion rate $c$, the weaker the refuge effects.
- For a high fusion rate ($c > 0.05$), refuge effects are negligible; $T$ equals the fixation time without landscape dynamics. For a small fusion rate ($c < 0.05$), refuge effects are very strong, $T$ can be more than 10 times longer than without landscape dynamics. With asymmetrical fragmentation ($p = 0.95$, triangles) both refuge and founder effects affect $T$. For a high fusion rate ($c > 0.05$) refuge effects are negligible, founder effects determine $T$; $T$ decreases with $c$ (more frequent founder effects generating additional drift). For a small fusion rate ($c < 0.005$) founder effects are negligible, refuge effects determine $T$ which equals $T$ in the case of symmetrical fragmentation. Dashed and dotted lines correspond to the approximation of $T$ we obtained using the coalescent effective size $N_e$ derived (Eq. (11)). This approximation is accurate under weak founder effects or weak refuge effects. For high $c$ and high $p$, this approximation tends to underestimate founder effects; for small $c$, it predicts refuge effects, but underestimate them.

Parameter values: $N = 100$; $X_0 = 1$ where $t$ is when the mutant appears; $s = 0.1$; $f = c = 0.1$.

which explains why its fixation probability is smaller. Moreover, taking into account only the first fragmentation event is enough to understand this result because each fragmentation–fusion cycle reinforces the effect of the first cycle.

3.2.4. Time to fixation of advantageous alleles

We measure fixation time $T$ as the mean time to fixation, conditional on the fixation of mutants. Fixation times can either be strongly decreased or increased compared to the case of a static landscape, depending on the relative strength of founder effects and so-called “refuge effects”. We use the term “refuge” (Fig. 7) to refer to the situation in which the mutant is fixed in one deme while the resident is fixed in the other deme (and the landscape is necessarily in state 2). Allele frequencies then cannot change until the next fusion; neither mutants nor residents can invade or become extinct. Therefore, the effect of a refuge is to delay fixation. We quantify this “refuge effect” by the length of the time spent in refuges. The fusion rate $c$ affects the intensity of refuge effects since it scales the waiting time until the next fusion. Besides, the asymmetry parameter $p$ can alter refuge effects: the more asymmetrical a fragmentation, the more the small deme formed by fragmentation is likely to be monomorphic, so that “half” a refuge is created by fragmentation, as it happens in the simulation showed in Fig. 7.

For Model 2a, when fragmentations are symmetrical, fragmentations do not generate founder effects, so that only refuge effects affect the mutant fixation time $T$. Results of simulations in Fig. 12 (with $p = 0.5$) indeed show that in this case, the fusion rate $c$ strongly influences $T$. For a small fusion rate ($c < 0.05$), the time between fragmentation and fusion is long, so that if a refuge appears, the associated refuge effect can be strong. $T$ is then strongly increased: $T$ can be more than 10 times longer than in a static landscape. In contrast, with a high fusion rate ($c > 0.05$), refuge effects are negligible. In this case, even if refuges are formed, the time until the next fusion is short and does not significantly delay fixation. Fixation times then approximately equal those in a static landscape (Eq. (2)).
and $f/c \sim 1$ or $f/c \ll 1$) and that fragmentations are symmetrical ($p \approx 1/2$). Then, for Model 2a, $N^c_e$ approximately equals $N$. For Model 2b, $N^c_e$ is slightly less than the harmonic mean size of the population (i.e. the size of the reference population in a static landscape):

$$N^c_e \approx \frac{f + c}{c/N + f/(2N) + fc/(4N)}.$$  

The term $fc/(4N)$ corresponds to the effect of population size variations that occurs in Model 2b even when fragmentations are symmetrical (Fig. 3). It explains the discrepancy between the expected time to fixation in a static landscape and these for Model 2b with symmetrical fragmentation (Fig. 13).

Still assuming weak refuge effects, when fragmentations are not symmetrical, compared to a static landscape, we obtain an effective size decreased by a term $fcγ/4$ for Models 2a and 2b:

$$N^c_e \approx \frac{f + c}{c/N + af/(2N) + fc/(4N)}.$$  

In contrast, when refuges can take place ($c \ll 1/N$) and last a long time compared to duration of the state 1 of the landscape ($f/c \gg 1$), $N^c_e$ is strongly increased compared to a static landscape and is independent of the asymmetry parameter $p$:

$$N^c_e \approx \frac{2N}{1 + \alpha(Nc)} \left[1 + \alpha^2(1 + 4/(fN))\right].$$  

$N$ is the size of the population when the landscape is in state 2 (which is the predominating state when refuges last a long time) and the term multiplying it is higher than 1 under the assumption we have made here. This term corresponds to the intensity of refuge effects.

It is interesting to note that the coalescent effective size cannot satisfactorily describe the fixation probability of an advantageous mutant in Models 2a and 2b, but that it gives a faithful prediction of (at least) the variations of the time to fixation with the parameters describing the spatial structure of the population.

3.3. Fixation probability and time to fixation of neutral and deleterious alleles

In the neutral case, as expected, mutant fixation probability is not altered by landscape dynamics of our three models (data not shown). After some time, all individuals of the population have indeed the same ancestor from the initial population. Given the spatial structure we assume in our models, all individuals of the initial population — neutral mutants and residents — are interchangeable. In particular, in Model 2 when the population is subdivided into two demes, the two demes are interchangeable. As a result, all individuals of the initial population have the same probability to be the future common ancestor of the population. Thus, the fixation probability of neutral mutants equals their initial frequency. Moreover, additional genetic drift due to founder effects speeds up the fixation of a mutant allele, even neutral, and refuges defined in Model 2 can be created in the neutral case also, delaying fixation. Consequently, the time to fixation of a neutral allele behaves qualitatively like an advantageous allele (data not shown).

We have built our three models such that advantageous and deleterious cases should be symmetrical because only the relative difference $s$ in birth rates between mutants and residents matters. A Moran process with an advantageous mutant birth rate $1 + s$ and a resident birth rate $1$ is identical to a Moran process with a deleterious mutant birth rate $1 + s$: reversing mutant and resident roles allows one to look at the deleterious case instead of the advantageous case. However an asymmetry due to the mutant initial frequency remains: an advantageous mutant with initial frequency $x_0$ is symmetrical to a deleterious mutant with initial frequency $1 - x_0$. However, we are of course interested in results with an initial deleterious mutant frequency $x_0$. We thus checked with simulations that this asymmetry does not significantly alter the expected results (data not shown): for our three models in the parameters values where the advantageous mutant fixation probability is lower than in a static landscape, the deleterious mutant fixation probability is higher. Moreover (and as expected), times to fixation of deleterious alleles are qualitatively similar to those of advantageous and neutral cases (data not shown), for all landscape dynamics we examined.

3.4. Comparison between Moran and Wright–Fisher processes

We obtained all results presented above using a Moran process for population dynamics. We did the same analyses for the more classical Wright–Fisher model (Wright, 1931). For the landscape dynamics we studied, results are qualitatively identical, for both fixation probability and time to fixation (data not shown). Fixation probabilities of advantageous (respectively deleterious) alleles are higher (respectively smaller) since for a same census population size, the effective size of a Wright–Fisher population is twice as high as that of a Moran population. For the same reason, fixation times are longer.

4. Discussion

We have proposed three models for understanding how dynamic landscapes influence the fixation probability and the time to fixation of a mutant allele. We have shown that compared to a static landscape (undivided population of constant size), in the case of repeated bottlenecks (Model 1), a succession of founder events decreases the fixation probability of an advantageous mutation, but accelerates its fixation (conditional on fixation). These effects are stronger when the landscape dynamics are faster. Also in the case of the repeated alternation of fragmentation and fusion of demes (Models 2a and 2b) founder events decrease the fixation probability of an advantageous mutation and accelerate fixation. However, the coexistence of two temporarily disconnected demes (state 2) generates a “refuge effect” which can strongly delay fixation. If population fusions are rare, refuge effects are the principal factor determining fixation times which are longer than in a static landscape. In contrast, if fusions are frequent, founder effects are the principal factor and fixation times are then shorter than in a static landscape. Note that founder effects are only observed in the case of asymmetrical fragmentation.

We have derived for Model 1 a variance (Eq. (5)), a coalescent (Eq. (6)) and a harmonic mean (Eq. (7)) effective size. The variance and coalescent effective sizes lead to the same approximation.
None of the effective sizes can give an accurate prediction for both the fixation probability and the time to fixation of an advantageous mutant. We have also shown that none of them does better than the others for all bottleneck intensities and frequencies (Figs. 4 and 5). The harmonic mean effective size nevertheless appears to be the most robust. For Models 2a and 2b, we have derived a coalescent effective size (Eq. (11)) which fairly predicts time to fixation under weak founder effects or weak refuge effects (Figs. 12 and 13). Depending on the characteristics of the landscape dynamics, this effective size is decreased or increased compared to that of a static landscape. Because refuge effects strongly increase time to fixation but do not affect fixation probability, there cannot exist one single effective population size which can describe both fixation probability and time to fixation for spatially structured populations subject to landscape dynamics generating refuges.

Otto and Whitlock (1997) studied the effect of a single bottleneck on the fixation probability of a beneficial mutation. Later, Wahl and Gerrish (2001) and Heffernan and Wahl (2002) analyzed bottlenecks occurring cyclically with an extreme regularity. In spite of the differences with our Model 1, our results are qualitatively similar to theirs. The occurrence of bottlenecks at stochastic times appears to weakly alter fixation compared to regular bottlenecks. However, an additional feature of our work is that the variance effective size we derived (Eq. (5)) takes into account the variance of allele frequencies between bottlenecks (i.e. during Moran processes). Our approximation may thus be used also for weak reduction of population size, when an advantageous mutant is more likely to be lost between bottlenecks than because of a bottleneck.

Extinction–recolonization models (Slatkin, 1977; Barton, 1993; Whitlock, 2003) can be compared to Models 2a and 2b of repetitive fragmentations and fusions. They are island models with a large number of demes. In each generation, extinction of one colony occurs with probability \( e \), and a number \( k \) of individuals recolonize instantaneously the extinct colony (allowing a founder event if \( k \) is small). Both extinction–recolonization models and our Models 2a and 2b allow one to model fragmenting populations where the number of occupied patches varies and the overall population size is nevertheless limited. However, they assume different mechanisms that result in a fluctuation of the number of patches: extinctions for the first case, secondary contacts (with, in Model 2b, competition following fusion, modeled by the pure death process) for the second case. Therefore, geographical and biological processes that can be modeled with these models are quite different. In extinction–recolonization models, excluding the case of extinction of small demes due to stochastic demographic fluctuations, extinctions implicitly assume dramatic and sudden events (such as violent climatic events), with immediate recolonization. All alleles in a deme which goes extinct are necessarily lost. In contrast, in our models, population size reductions are less dramatic events: we assume a complete fusion of demes, so that all alleles compete with each other and can survive after the population size reduction. It thus corresponds rather to the movement or expansion of a subpopulation to the place where another is already established, thereby increasing competition. In spite of these differences, our conclusions on fixation probability are qualitatively similar to the results of extinction–recolonization models (reduction of the fixation probability compared to an unidivided population). In contrast, fixation times can be drastically higher in Models 2a and 2b (Figs. 12 and 13) than in extinction–recolonization models for which the diffusion approximation in a static landscape (Eq. (2)) gives good predictions (Whitlock, 2003). The reason is that the dynamics we assume allow the creation of refuges. Consequently, alleles can be retained for a long time without risking a disappearance, and then be reintroduced in the total population. Hence, geographical and biological processes that can be modeled with our models should probably allow one to keep a higher global diversity than those that are modeled with extinction–recolonization models.

Our three models of landscape dynamics assume (sub-)population size variations. Our results of Model 2a and 2b about the mean mutant fixation probability \( U \) (Figs. 8–10) show that the effect of these variations can be considered as analogue to repetitive founder events. However, when considering the state of the landscape when a mutant appears, a more accurate analysis is possible. Otto and Whitlock (1997) analyzed fixation probability in populations of changing size. They showed that the fate of a mutation strongly depends on the direction of variation of population size (increase or decrease) when the mutation occurs. Our results on state-dependent fixation probability for Model 2a and 2b with successive population increases and decreases confirm that the closer in time to a population size variation a mutant arises, the more its fixation probability is altered.

Our question was to understand how dynamic landscapes alter mutant fixation. One main modification is that founder effects and refuge effects are repetitive and cumulative. These effects can exist in static landscapes, but can occur only once. Landscape dynamics thus strengthen alterations of fixation probabilities and times to fixation. This is particularly true for times to fixation which can be increased by a factor of more than 10 (Figs. 12 and 13). This stresses the importance of considering the isolation of some population and their fusion after a possibly long time. Depending on the characteristics of the dynamics, fixation can be disfavored or unaffected, delayed or accelerated. Therefore, one needs to describe the whole dynamics of the landscape, and to specify characteristic time scales. Fast dynamics are appropriate to model ecological processes such as dispersal and recolonization events (establishment of new colonies and their later fusion (DeHeer and Kamble, 2008; Vasquez and Silverman, 2008) because of their expansion or because one habitat becomes unsuitable), or to model geographical processes such as changes in the fragmentation of habitat due to human action (Davies et al., 2006). In that case, beneficial mutations have “one small chance of doing very well”: fixation is unlikely (frequent founder effects) but very fast if it occurs (limited refuge effects). In contrast, slow dynamics are appropriate to geographical events such as the separation of populations due to glacial events followed by postglacial contact (Young et al., 2002) or such as repetitive fragmentation and fusion of islands (or lakes) due to water level variations caused by climatic events (Owen et al., 1990; Delvaux, 1999; Galis and Metz, 1998; Stiassny and Meyer, 1999; Cook, 2008). In that case, fixation of a beneficial allele would be more likely (rare founder effects) but very slow when it occurs (strong refuge effects). On top of specifying characteristic times scales, knowing how the overall population size varies is essential. In Model 2a the overall carrying capacity is constant whereas it fluctuates in Model 2b. Results of these two models are very close when compared to equivalent static landscapes (i.e. the effect of dynamic landscapes is the same for both models), but they can be significantly different when compared to each other.

Our models are based on some strong assumptions. First, we assume that the pure birth (or death) process used after a bottleneck (Model 1) or a fragmentation or a fusion (Models 2a and 2b) is instantaneous. This assumption is justified because this exponential growth phase is very short compared to the timescale of allele frequency change. Nevertheless, we checked that relaxing this assumption does not significantly change our results. We thus have explicitly computed the durations of the exponential growth and decline phases, which appeared indeed to be much shorter than other processes; fixation times were almost unchanged (data not shown).
Second, Models 2a and 2b assume that in state 2, the two demes do not exchange migrants. This may be a strong assumption, we thus relaxed it (data not shown). Migration has only a small effect on fixation probability: with a migration rate of 0.1 per individual, fixation probabilities are only 10% (at the most) higher than without migration (for the range of values we explored, Table 1). In contrast, high migration rates have a significant effect on time to fixation which approaches the time to fixation in a static landscape (Eq. (2)) when migration increases. When the landscape dynamics generate refuges, each migration event indeed breaks refuges, which shortens the time to fixation. Our results are nevertheless still valid in the case of weak migration: for a per capita migration rate less than 0.01, we still observe refuge effects, i.e. the time to fixation is significantly higher than that in a static landscape. Also note that the effect of migration weakly depends on the fragmentation rate f and on the fusion rate c.

Finally, Models 2a and 2b assume that the population is divided into at most two demes. Fragmented populations generally consist of many subpopulations (Hanski and Gaggiotti, 2004). In such landscapes, the number of founder events and of refuges would thus be higher; advantageous mutations would thus be less likely to be fixed and fixation, when it occurs, would be more delayed. For the extreme scenario of continually increasing fragmentation of the population, fixation could even never occur because refuges would always exist and each one may fix a different allele. However, highly fragmented populations often consist of small and weakly interconnected demes whose existence is ephemeral: subpopulations go extinct stochastically and habitats are recolonized. Extinctions may then allow fixation since it destroys refuges.

Our models need to be improved to take into account more complex dynamics involving for example more demes, extinction and recolonization of demes, or different local selective pressures which would fix different alleles in different demes. They also need to consider that new mutations may arise before fixation of older mutations. In particular new mutants can appear in refuges and invade them. Taking into account these additional features will allow understanding not only allele fixation probability and time to fixation but also fixation flux, genetic diversity or adaptation rate in populations involved in the geographical and biological processes that dynamic landscapes allow one to model.

Acknowledgments

The authors are grateful to four anonymous referees, whose comments significantly improved the scope and quality of our work. We thank Sébastien Ballesteros for helpful discussions. R. A. is supported by a PhD fellowship from the French Ministère de la Recherche et de la Technologie. Traveling was funded by the project Modèles Aléatoires de l’Evolution du Vivant (MAEV) of the French Agence Nationale pour la Recherche (ANR).

References


Small edge populations at risk: Genetic diversity of the green lizard (Lacerta viridis viridis) in Germany and implications for conservation management. Conservation Genetics 8, 555–563.


Ecological speciation in dynamic landscapes

R. AGUILÉ†, A. LAMBERT‡1 & D. CLAESSEN*2

*Laboratoire Ecologie et Évolution, UPMC Univ Paris 06, Ecole Normale Supérieure, CNRS, Paris, France
†Institut des Sciences de l’Évolution de Montpellier, CNRS, Montpellier, France
‡Laboratoire Probabilité et Modèles Aléatoires, UPMC Univ Paris 06, Paris, France

Abstract

Although verbal theories of speciation consider landscape changes, ecological speciation is usually modelled in a fixed geographical arrangement. Yet landscape changes occur, at different spatio-temporal scales, due to geological, climatic or ecological processes, and these changes result in repeated divisions and reconnections of populations. We examine the effect of such landscape dynamics on speciation. We use a stochastic, sexual population model with polygenic inheritance, embedded in a landscape dynamics model (allopatry–sympatry oscillations). We show that, under stabilizing selection, allopatry easily generates diversity, but species coexistence is evolutionarily unsustainable. Allopatry produces refuges whose persistence depends on the characteristic time scales of the landscape dynamics. Under disruptive selection, assuming that sympatric speciation is impossible due to Mendelian inheritance, allopatry is necessary for ecological differentiation. The completion of reproductive isolation, by reinforcement, then requires several sympatric phases. These results demonstrate that the succession of past, current and future geographical arrangements considerably influence the speciation process.

Introduction

Ecological speciation – the evolution of reproductive isolation as a consequence of divergent selection based on ecological mechanisms (Schluter, 2001) – can occur in any geographical arrangement, allopatry, parapatry or sympatry. Allopatric and parapatric speciations have been well accepted for years as plausible modes of speciation. Thanks to the recent accumulation of theoretical models and of empirical evidence, sympatric speciation now seems to be accepted as possible at least (Turelli et al., 2001; Via, 2001; Bolnick & Fitzpatrick, 2007). A debate about its frequency is nevertheless still going on (see e.g. Bolnick & Fitzpatrick, 2007; Fitzpatrick et al., 2009). Some authors (e.g. Fitzpatrick et al., 2008, 2009) argue that classifying speciation events into distinct classes (allopatric, parapatric or sympatric) is unrealistic and potentially misleading. However, the geographical arrangement of speciation candidates remains informative as this selects the possible mechanisms leading to speciation (Rundle & Nosil, 2005; Bolnick & Fitzpatrick, 2007). For example, divergent selection between different environments can drive speciation in allopatry, as opposed to sympatry where all individuals necessarily experience the same environment. On the contrary, ecological interactions between the individuals of a sympatric population, such as competition, can generate reproductive isolation in sympathy, as opposed to allopatry where individuals in different locations do not interact.

It is rather surprising that almost all models of speciation focus on a single geographical arrangement (Fitzpatrick et al., 2009), given that standard verbal models of speciation take into account both allopatry...
and sympatry (Rundle & Nosil, 2005). Ample empirical evidence shows that speciation is often initiated in allopatry and completed in sympatry (e.g. Taylor & McPhail, 2000; Feder et al., 2003; Jordal et al., 2006; Xie et al., 2007; Grant & Grant, 2009), or conversely (e.g. Baack, 2004; Stuessy et al., 2004). The succession of different geographical arrangements seems to be of particular importance in adaptive radiations (Rundell & Price, 2009). In addition, even if ecological speciation can be initiated (i.e. the evolution of weak reproductive isolation and of weak ecological divergence) within only tens of generations (Hendry et al., 2007), divergent selection alone often fails to complete speciation on such a time scale (Nosil et al., 2009). As a result, complete ecological speciation (i.e. the evolution of persistent reproductive isolation) can require a significant amount of time during which several biogeographical changes are likely to influence the speciation process (Bolnick & Fitzpatrick, 2007; Fitzpatrick et al., 2008).

Dynamic landscapes, here defined as the repeated alternation of allopatry and sympatry of populations (Aguilée et al., 2009), are indeed common at different spatio-temporal scales. For example, the connections between populations may vary due to glaciations and post-glacial secondary contacts (Hewitt, 2000; Young et al., 2002; Zhang et al., 2008). Geological processes (e.g. volcanic events) as well as climatic variations can cause sea level changes, resulting in separations or fusions of islands (Cook, 2008; Esselstyn et al., 2009). Similarly, persistent fluctuations of water level causing fragmentation and fusion of lakes are thought to have influenced the radiation of cichlid fishes in the Great African Lakes (Owen et al., 1990; Arnegard et al., 1999; Stiassny & Meyer, 1999; Young et al., 2009). At a different spatio-temporal scale, populations can oscillate between allopatry and sympatry due to the establishment of new colonies by dispersal and their later fusion (DeHeer & Kamble, 2008; Vasquez & Silverman, 2008). Landscapes are also rapidly changed by contemporary fragmentation and reconstruction of habitats due to human activities (Davies et al., 2006). Note that the spatio-temporal scales of landscape dynamics and the nature of the geographical arrangements of populations are relative to their population dynamics and to their evolutionary dynamics.

In the present paper, we address the following question: how do dynamic landscapes, in contrast to static landscape, affect ecological speciation? A well-documented, related question concerns the effect on speciation of secondary contact, i.e. the transition from allopatry to sympatry. Secondary contact can have two opposite effects (Servedio & Kirkpatrick, 1997; Noor, 1999; Servedio & Noor, 2003). First, it allows gene flow between differentiated populations, which homogenizes genotypes and impedes speciation. Second, hybrids produced by differentiated individuals can have a reduced fitness: reinforcement can then potentially complete speciation. Diversity generated in allopatry can thus be maintained or lost at secondary contact, essentially depending on the rate of interbreeding and on how much the fitness of hybrids is reduced compared to their parents (Kirkpatrick, 2000; Servedio & Noor, 2003). The success of speciation at secondary contact also depends on the mechanisms of reinforcement. Consequently, the duration of the allopatric state preceding secondary contact may be crucial. For example, hybrids produced at secondary contact can have a reduced fitness due to genetic incompatibilities between individuals from different former allopatric populations (Orr, 1995), and when hybrids are unviable, postzygotic reproductive isolation is complete. Reinforcement reducing hybrid production is then called intrinsic. Such genetic incompatibilities take long to evolve (Orr & Orr, 1996), so that intrinsic reinforcement is expected at secondary contact only after a long geographical isolation period. Alternatively, hybrids can have a reduced fitness because of ecological interactions (e.g. hybrids are phenotypically intermediate to their parents and consequently worse competitors than their parents). In this case, reinforcement is called extrinsic and can lead to prezygotic reproductive isolation, often much more quickly than the genetic incompatibilities do, for example via the evolution of assortative mating (Servedio & Noor, 2003). In such a case, reinforcement is driven by disruptive selection, selecting for positive assortative mating, which is either directly related to the ecological trait responsible for the reduced fitness of hybrids (‘one-allele’ mechanism), or related to a nonecological, linked and possibly sexual trait (‘two-allele’ mechanism; Felsenstein, 1981; Dobelei & Dieckmann, 2005).

These results on secondary contact are not sufficient to fully answer the question because the whole process is heavily dependent on the time scales of allopatric stages before and after secondary contact. For example, ecological divergence is often assumed to be at equilibrium at secondary contact, whereas the duration of allopatry before secondary contact can be too short for that. Similarly, secondary contact is usually assumed to last long enough to let the population reach an equilibrium state (either failed or permanent speciation), whereas a new landscape change could prevent the population from reaching this equilibrium. Moreover, the effect of successive secondary contacts has not been investigated. This paper examines under which conditions landscape dynamics allow (i) the formation of diversity and (ii) its maintenance (or not) until ecological speciation is complete and persistent. We aim to characterize the likelihood, time scale and predominant underlying mechanisms for each of these two points.

**Model**

To address these questions, our model is built upon the following four guiding assumptions. First, as a first analysis of the effect of landscape dynamics, the landscape dynamics should be as simple as possible: we
assume that the landscape oscillates between an undivided state (sympatry) and a divided state with two subpopulations (allopatry). Second, as the outcome of secondary contact is expected to depend on the fitness landscape, the model should allow us to explore different fitness landscapes. We thus choose a model where the population trait evolves to a singular point (Geritz et al., 1998) and, depending on parameter values, it then evolves under either stabilizing or disruptive selection. Third, we assume that diversification can only occur between allopatric populations. This is performed by generating different environmental conditions for allopatric populations. Sympatric diversification is made unlikely by assuming that phenotypic traits are determined by many independently segregating loci, with small allelic effects. Such genetic constraints are known to impede sympatric divergence (Waxman & Gavrilets, 2005). Fourth, reproductive isolation should be allowed to evolve. To this end, we allow for the evolution of assortative mating based on a one-allele mechanism. Post-zygotic reproductive isolation, for example due to genetic incompatibilities, is assumed to take longer to evolve. To this scale we will consider (Orr & Orr, 1996) and is thus not incorporated into the model.

**Ecological model**

This section describes the population dynamics and evolution in one subpopulation, either the only subpopulation when the landscape is in a sympatric state, or either of the two subpopulations when the landscape is in an allopatric state. The dependence of the parameters on the landscape structure is detailed in Landscape model section.

**Consumer-resource dynamics**

We use a stochastic, individually based model inspired by the models of Claessen et al. (2007, 2008). The consumer population consists of \( n(t) \) discrete individuals. Individuals of the subpopulation under scrutiny compete with each other for two different resources. Each individual \( i \) is characterized by an evolving phenotypic trait \( u_i \) determining its resource utilization strategy (see Table 1 for a summary of the notation). This ecological trait represents a degree of specialization where \( u_i = 0.5 \) represents a generalist strategy, \( u_i = 1 \) and \( u_i = 0 \) represent complete specialization on resource 1 or 2, respectively. It operates through e.g. morphological adaptations influencing the ability to feed on each resource. We assume a power-law trade-off (e.g. Egas et al., 2004; Spichtig & Kawecki, 2004) between the exploitation of the two different resources, specified by a parameter \( z \).

The fitness \( W(u_i) \) of individual \( i \) is a linear combination of performance on either resource:

\[
W(u_i) = \beta(u_i) - d = F_1 u_i^z + F_2 (1 - u_i)^z - d \tag{1}
\]

where \( \beta(u_i) \) is the birth rate of individual \( i \), \( d \) is the constant per-capita death rate, and \( F_1 \) and \( F_2 \) are the densities of resources 1 and 2 available for the subpopulation under scrutiny. The shape of the trade-off depends on the parameter \( z \). When \( 0 < z < 1 \) (resp. \( z > 1 \)), the trade-off is weak (resp. strong; e.g. Egas et al., 2004; Spichtig & Kawecki, 2004): the population is predicted to evolve by directional selection to a singular strategy \( u^* \) where it then experiences stabilizing (resp. disruptive) selection (Rueffler et al., 2004). When \( z = 1 \), the trade-off is linear; at a singular strategy \( u^* \), \( F_1 = F_2 \) and the selection gradient is flat (i.e. no selection).

### Table 1 Notation and numerical values.

<table>
<thead>
<tr>
<th>Evolving trait</th>
<th>Definition</th>
<th>Interpretation</th>
<th>Range of values explored</th>
</tr>
</thead>
<tbody>
<tr>
<td>( u_i )</td>
<td>Ecological trait of individual ( i )</td>
<td>( u_i = 0.5 ): generalist strategy</td>
<td>( 0.2 \leq z \leq 2 )</td>
</tr>
<tr>
<td>( z )</td>
<td>Power-law trade-off parameter</td>
<td>( u_i \neq 0.5 ): specialist strategy</td>
<td>( 0 \leq z \leq 1 )</td>
</tr>
<tr>
<td>( d )</td>
<td>Per-capita death rate</td>
<td>( a_i = 0 ): random mating</td>
<td>( d = 0.1 )</td>
</tr>
<tr>
<td>( L )</td>
<td>Number of diploid loci coding trait ( j )</td>
<td>( a_i &gt; 0 ): assortative mating</td>
<td>( L = 6 \text{ or } L = 12 )</td>
</tr>
<tr>
<td>( \mu )</td>
<td>Trait per-locus mutation probability</td>
<td>( a_i &lt; 0 ): disassortative mating</td>
<td>( 10^{-6} \leq \mu \leq 10^{-1} )</td>
</tr>
<tr>
<td>( \sigma_j^2 )</td>
<td>Phenotypic variance of trait ( j )</td>
<td></td>
<td>( 10^{-6} \leq \sigma_j^2 \leq 0.5 )</td>
</tr>
<tr>
<td>( K_j )</td>
<td>Maximum density of resource ( j ) available in sympatry</td>
<td></td>
<td>( K_1 = K_2 = 1 )</td>
</tr>
<tr>
<td>( V )</td>
<td>Population size scaling parameter in sympatry</td>
<td></td>
<td>( 10 \leq V \leq 100 )</td>
</tr>
<tr>
<td>( h )</td>
<td>Asymmetry of the two resource distributions in allopatry</td>
<td></td>
<td>( 1.1 \leq h \leq 25 )</td>
</tr>
<tr>
<td>( \rho )</td>
<td>Asymmetry of the two subpopulation sizes in allopatry</td>
<td></td>
<td>( 0.5 \leq \rho \leq 0.95 )</td>
</tr>
<tr>
<td>( T_a )</td>
<td>Duration of allopatric phases</td>
<td></td>
<td>( 10 \leq T_a \leq 10^6 ) generations</td>
</tr>
<tr>
<td>( T_s )</td>
<td>Duration of sympatric phases</td>
<td></td>
<td>( 10 \leq T_s \leq 10^6 ) generations</td>
</tr>
<tr>
<td>( T_r )</td>
<td>Duration of partial secondary contact phases</td>
<td></td>
<td>( 0 \leq T_r \leq 10^5 ) generations</td>
</tr>
<tr>
<td>( r )</td>
<td>Hybridization probability during the partial secondary contact window</td>
<td></td>
<td>( 10^{-6} \leq r \leq 0.25 )</td>
</tr>
<tr>
<td>( \delta_i )</td>
<td>Per-capita death rate</td>
<td></td>
<td>( \delta_1 = \delta_2 = 1 )</td>
</tr>
</tbody>
</table>
Assuming that resources follow semi-chemostat dynamics, the densities of resources can be expressed as

\[
\begin{align*}
F_1 &= \frac{K_1}{1 + \frac{1}{2} \sum_{i=1}^{20} u_i^2} \\
F_2 &= \frac{K_2}{1 + \frac{1}{2} \sum_{i=1}^{20} (1 - u_i)^2}
\end{align*}
\]

where \(K_1\) (resp. \(K_2\)) is the maximum density of resource 1 (resp. 2) available for the subpopulation under scrutiny and \(V\) is a scaling parameter allowing us to set the consumer population size relative to the maximum density of resources (see Appendix A for the derivation of eqn 2). Such resource dynamics pertain to, for example, systems of size-selective fish foraging on zooplankton (Persson et al., 1998).

Reproductive isolation

We model prezygotic reproductive isolation by assuming that the population is sexual and that each individual is characterized by a mating trait \(x_i\). At each birth event, the individual \(i\) chosen to reproduce randomly encounters a sexual partner \(j\) among the individuals of the opposite sex in the subpopulation under scrutiny. The pair mates (or not) depending on the mating trait \(x_i\) of individual \(i\) and the difference \(\Delta = u_i - u_j\) between the ecological traits of the two individuals (one-allele mechanism). Individual \(i\) mates with the chosen partner \(j\) with probability

\[
g = \begin{cases} 
(1 - \frac{1}{2} \text{exp} \left( -\frac{x_i^2}{2\sigma_j^2} \right) \text{exp} \left( -\frac{\Delta_i^2}{2\sigma_j^2} \right) & \text{if } x_i > 0 \\
0.5 & \text{if } x_i = 0 \\
1 - (1 - \frac{1}{2} \text{exp} \left( -\frac{x_i^2}{2\sigma_j^2} \right) \text{exp} \left( -\frac{\Delta_i^2}{2\sigma_j^2} \right) & \text{if } x_i < 0
\end{cases}
\]

where \(x_i = 1/(20x_j^2)\). This Gaussian mating function has the minimal biological realism required: it is a continuous function in \(x\), individual \(i\) has no preference when \(x_i = 0\) and mates assortatively (resp. disassortatively) when \(x_i > 0\) (resp. \(x_i < 0\)), and choosiness increases when \(\Delta_j\) increases. We will first analyse our model with \(x\) fixed and identical for all individuals, then we will allow this trait to evolve. In the first case, using a fixed, positive, and identical for all individuals, then we will allow this trait to evolve. In the second case, we model the evolution of exclusively (dis-)assortative mating based on the ecological trait.

When individual \(i\) rejects partner \(j\), another partner is randomly chosen and the process repeats until mating succeeds, or until individual \(i\) has rejected 50 potential partners. This represents a very small cost of (dis-) assortativeness: Schneider & Bürger (2006) and Kopp & Hermisson (2008) showed that giving up mating after rejecting just ten potential partners has a very low cost.

Inheritance rules

The genetic architecture and inheritance rules are based on Claessen et al. (2008). Trait \(j\) (being either the ecological trait \(u\) or the mating trait \(x\)) is determined by \(L_j\) diploid, additive loci on autosomal chromosomes. We assume neither environmental effects nor epistasis nor dominance effects. Each allele can take any real value (restricted to \([0,1]\) for the ecological trait). The value of the phenotypic trait \(j\) is the mean of the \(2L_j\) alleles determining this trait.

We assume independent segregation of each locus; at each locus, one offspring allele is randomly chosen from maternal and paternal alleles. We also assume \(L_j = 6\), so that each allele has a limited effect on the value of the phenotypic trait. Because of these assumptions, when selection is disruptive, sympatric evolutionary diversification is severely delayed (Claessen et al., 2008) and is not expected to happen on the time scales we investigate.

At birth, the offspring’s sex is determined randomly assuming a balanced sex ratio. Mutation occurs at each locus determining trait \(j\) with probability \(\mu_j\). The mutant allele value is drawn from a normal distribution (truncated between \([0,1]\) for trait \(u\) with mean equal to the parental allele value and with standard deviation \(\sigma_j\sqrt{2L_j}\)). This mutation size at the allele level results in a variance \(\sigma_j^2\) at the trait level, regardless of the number of loci \(L_j\) (van Doorn et al., 2004).

Landscape model

Environmental conditions

In allopatry, the population consists of two isolated subpopulations (i.e. without migration between them), referred to as the ‘first’ and ‘second’ subpopulation. Parameters related to the first and second allopatric subpopulations are differentiated by a superscript (1) and (2), respectively; parameters related to the single subpopulation when the landscape is in a sympatric state are indicated without superscript.

In sympatry, we assume that the maximum densities of both resources are the same, i.e. \(K_1 = K_2\). By contrast, in allopatry, we assume that the two subpopulations face different environmental conditions so that allopatric subpopulations are expected to diverge with respect to their ecological trait. This is performed by assuming an asymmetrical distribution of the resources in the two patches: resource 1 is \(h > 1\) times more abundant in the first patch than in the second one, whereas resource 2 is \(h\) times more abundant in the second patch than in the first one. The maximum densities of resources in the allopatric patches are defined by (see Appendix B for the derivation of eqn 4)

\[
\begin{align*}
K_1^{(1)} &= hK_1^{(2)} \\
K_1 &= pK_1^{(1)} + (1 - p)K_1^{(2)} \\
K_2^{(2)} &= hK_2^{(1)} \\
K_2 &= pK_2^{(1)} + (1 - p)K_2^{(2)}
\end{align*}
\]

Parameter \(p\) allows us to set the relative sizes of allopatric subpopulations: the further from 0.5, the more asymmetrical the sizes of the allopatric subpopulations. The scaling parameters for the allopatric subpopulation sizes are expressed accordingly: \(V^{(1)} = pV\) and \(V^{(2)} = (1-p)V\).
Because we assume $K_1 = K_2$, the singular strategy in sympatry corresponds to the generalist strategy $u^* = 0.5$. Because resource 1 is more abundant in the first patch than in the second patch in allopatry, and conversely for resource 2, the singular strategies $u^{(1)}$ and $u^{(2)}$ in allopatry correspond to two more specialized strategies: $u^{(1)} > 0.5$ and $u^{(2)} < 0.5$, respectively. Note that the nature of singular strategies does not depend on the geographical arrangement: under a weak (resp. strong) trade-off, populations at singular strategies experience stabilizing (resp. disruptive) selection both in sympatry and in allopatry. Appendix C gives the detailed adaptive dynamics analysis of the model.

**Landscape dynamics**

We first investigate the effect of a secondary contact, that is, a one-off landscape change from allopatry to sympatry. Later, we assume that the landscape oscillates between sympatry and allopatry.

Allopatric phases last $T_a$ generations. At fragmentation of the landscape, each individual ends up in the first subpopulation with probability $p$ and in the second subpopulation with probability $1-p$. Sympatric phases last $T_s$ generations. During the $T_s$ first generations of sympatric phases ($T_s < T_a$), we assume a reduced mating probability between individuals from different former allopatric subpopulations, in this way mimicking a ‘window of partial secondary contact’. This assumption allows us to slow down the process of hybridization at secondary contact.

During the window of partial secondary contact, although all individuals have access to the same resources (sympatric state), as a result of their specialization on different resources in allopatry, we expect that individuals from a given former allopatric patch reach a contact zone and meet individuals from the other former allopatric patch less often than they meet individuals from their own former patch. Hybrids born in the contact zone are assumed to tend to remain in the contact zone [as do, e.g. hybrids of *Cornus coronae* and *C. cornix* (Saino, 1992)] and thus to meet individuals from each former allopatric patch less often than they meet other hybrids.

We model such partial secondary contact as follows. The model assumptions are those used for the sympatric state (in particular resource abundance and competition for resources), except that, at each birth event, the set of potential sexual partners of the individual chosen to reproduce is a randomly drawn fraction of the population of the opposite sex. For an individual from a given former allopatric patch, this fraction consists of individuals from the same former allopatric patch with probability 1 and of individuals from the other former allopatric patch with probability $r$ ($0 \leq r < 1$) per individual. For a hybrid, this fraction consists of hybrids with probability 1 and of individuals from each former allopatric patch with probability $r$ per individual. Note that such modelling of the window of partial secondary contact does not explicitly take into account the spatially explicit modelling of a contact zone.

**Numerical simulations**

The stochastic model described above is simulated using a birth and death process in continuous time. We used the Gillespie (1977) algorithm. We pick the time until the next event from an exponential distribution with mean equal to $1/(\sum_{i=1}^{M} (b_i u^i + d))$, i.e. the inverse of the total rate at which events occur. The occurring event is randomly chosen proportionally to the rate of each possible event (birth or death).

The weak trade-off case is analysed using a fixed mating trait; the strong trade-off case is first analysed using a fixed mating trait, then this trait is allowed to evolve. The model with a fixed mating trait is simulated by initializing the allele value of the $L_d$ diploid loci of all individuals to the same value and by setting the mutation rate $\mu_a$ to 0.

We measure the time in generations: the generation time is equal to one time unit of the simulation real time divided by the constant death rate $d$. Our numerical simulations use a high mutation rate, $\mu_a = 0.01$ (and $\mu_a = 0.01$ when the mating trait is allowed to evolve), because this considerably speeds up the process we wish to study. We show in Appendix D that using a smaller mutation rate does not change the results, except in terms of time scales: the speed of trait evolution is expected to be proportional to the mutation rate. To minimize the underestimation of time scales, we used a low phenotypic variance of new mutants, $\sigma_u = 0.02$ (and $\sigma_a = 0.02$ when the mating trait is allowed to evolve), when using a high mutation rate (the speed of trait evolution is also expected to be proportional to the phenotypic variance of new mutants).

All figures show either specific time series or means over 50 replications of a simulation (or more replications when indicated as such). In the latter case, we present in the figures the 95% confidence intervals of the estimated means over the replicates.

**Results**

**Weak trade-off case**

Under a weak trade-off between the use of the two resources, we expect from the model definition that the mean ecological trait evolves to – and remains at – a generalist strategy in sympatry. In allopatry, the two subpopulations are expected to evolve to two different specialized strategies, i.e. allopatry is expected to generate diversity. We checked this expected behaviour of the model before analysing the effect of landscape dynamics (not shown).
Secondary contact
At secondary contact, i.e. the transition from allopatry to sympatry, diversity generated in allopatry may be lost. Figure 1 shows that when individuals that are specialized on different resources are not reproductively isolated (fixed mating trait $z < 0.5$), diversity collapses immediately after secondary contact. Their offspring have indeed a generalist strategy; under a weak trade-off ($z = 0.45$ in Fig. 1), selection is stabilizing, so that generalists have a higher fitness than specialists. Intermediate types thus are selected for and rapidly invade the population. Positive assortative mating increases the frequency of extreme phenotypes that are selected against because they depart from the generalist strategy. Assortative mating is thus not expected to evolve at secondary contact under a weak trade-off (see Appendix D, Fig. D1 and Table D1, and e.g. Slatkin, 1979; Dieckmann & Doebeli, 1999; Schneider & Bürger, 2006). Some other mechanisms, not depending on mate choice but depending on the ecological trait, could nevertheless lead to reproductive isolation in allopatry. Temporal isolation and pollinator isolation are such other mechanisms (Coyne & Orr, 2004).

Next, we analyse the effect of secondary contact assuming that during the allopatric stage, one or several mechanisms lead to the appearance of prezygotic reproductive isolation (simulated by assuming a fixed, positive $z$). In this case, Fig. 1 (fixed $z > 1$) shows that diversity in the ecological trait is nevertheless lost after some time. Indeed, the two differentiated species can ecologically coexist at secondary contact as they occupy two different ecological niches, but directional selection acts on their ecological traits, so that they converge towards the same generalist strategy. Typical evolutionary trajectories of the ecological trait shown in Appendix D (Fig. D2) illustrate that one of the two species almost always goes extinct before reaching the generalist strategy. When converging to the generalist strategy, the ecological niches of the two species become more similar. Because of stochasticity in the evolutionary trajectories of the pair of species, one of them reaches the generalist strategy before the other one. The generalist species is slightly better adapted than the other, which consequently goes extinct by competitive exclusion. In brief, the coexistence of two species is thus ecologically possible at secondary contact, but evolutionarily unsustainable.

The speed of diversity loss strongly depends on the asymmetry of the sizes of the subpopulations in allopatry (parameter $p$). At secondary contact, the more different their relative population sizes, the faster one of them goes extinct (Fig. 2). Moreover, the population that goes extinct is most of the time the smallest of the two subpopulations (see typical evolutionary trajectories in Appendix D, Fig. D2). Two reasons explain this result. First, the speed of evolution of a population to the generalist strategy is proportional to its abundance (everything else being equal). The smaller subpopulation thus converges slower to the generalist strategy than the larger one. The smallest subpopulation becomes more maladapted in comparison with the other one, which competitively excludes it more easily. The second reason is a by-product of the model construction: resource abundances in allopatry are defined so that the relative abundance of the two resources is more asymmetrical in the small patch than in the large patch (eqn 4). Consequently, the level of specialization of the smaller subpopulation in allopatry is higher than that of the larger one. Thus, for the same reason, converging to the generalist strategy takes more time for the smaller subpopulation, which risks exclusion by the better adapted, larger subpopulation.
Sympatry-allopatry oscillations

Assuming full landscape dynamics, i.e. oscillations between sympatry and allopatry, the end of a secondary contact is the beginning of an allopatric phase. The latter makes the two subpopulations diverge. Therefore, if subpopulations still coexist at the end of sympatry, their persistence is guaranteed until the next secondary contact. Figure 2 shows the probability that two species derived from former allopatric subpopulations still coexist after a sympatric phase of fixed duration. With the parameter values used in Fig. 2, the probability of diversity maintenance is higher than the probability of diversity loss for at least 1000 generations in sympatry. Consequently, landscape dynamics could maintain diversity if sympatric phases are short enough. The coexistence of two species, although evolutionarily unsustainable in a fixed, sympatric landscape, becomes possible in a dynamic landscape. Appendix D (Fig. D3) shows typical evolutionary trajectories where diversity is maintained in the long term thanks to landscape dynamics and typical evolutionary trajectories where it is not.

The characteristics of landscape dynamics allowing speciation and its maintenance depend on the parameters of the environment and of the population dynamics. When allopatric patches are more different in terms of their resource abundances (larger $h$), subpopulations are more specialized at secondary contact. Figure 3 shows that sympatry can then last a longer time without diversity loss because the convergence to the generalist strategy takes longer. When the population size is small, the stochasticity of the evolutionary trajectory of the pair of species converging to the generalist strategy is high. One of the species is then likely to rapidly become significantly closer to the generalist strategy than the other which then becomes extinct. When population size is small, sympatry should thus be shortlived to avoid diversity loss (Fig. 3). The speed of trait evolution is expected to vary along with the strength of selection. Figure 3 shows that for weaker selection, sympatry can accordingly last longer without diversity loss (see also Appendix D, Fig. D4). Note that our numerical results may underestimate the time scale at which the system maintains diversity because we use a high mutation rate on the ecological trait ($M = 0.01$). We have checked that mutation rates several orders smaller do not change our results, except with respect to absolute time scales (see Appendix D, Fig. D5).

Under a weak trade-off, speciation is impossible in a static, sympatric landscape. Landscape dynamics generate and maintain speciation under four conditions. First, the allopatric phases must be long enough to allow subpopulations to diverge significantly. Second, reproductive isolation must have evolved in allopatry and must be maintained. Third, the sympatric phases must be shorter than the time needed for the species to co-evolve to the same generalist strategy. Last, allopatry must divide the population into two subpopulations whose abundances are of similar sizes. Ecological speciation and its maintenance in a dynamic landscape is thus possible, but it occurs under more restrictive conditions than purely allopatric speciation.

Strong trade-off case

Under a strong trade-off between the use of the two resources, we expect from the model definition that, in sympathy, the mean ecological trait evolves to a generalist strategy. In allopatry, the two subpopulations are expected to evolve to two different specialized strategies. We checked this expected behaviour of the model before analysing the effect of landscape dynamics (not shown). In addition, sympatric evolutionary diversification is not expected to happen on the time scale we investigate: we checked this with simulations lasting $10^6$ generations (see Appendix D, Fig. D6).

Secondary contact

Simulations with a fixed mating trait reveal a sharp transition in the maintenance of diversity at secondary contact: diversity generated in allopatry is maintained in the long term after secondary contact only when the mating trait of the population is above a threshold $z_{lm}$ (Fig. 4, strong trade-off generated with $z = 1.6$). This threshold is the boundary between interbreeding and reproductive isolation of individuals specialized on different resources. Its value thus depends on the ecological divergence of individuals in allopatry: the higher the divergence at equilibrium, the lower the threshold (Fig. 4).

Hybrids produced at secondary contact by specialists have a generalist strategy. Under a strong trade-off, selection is disruptive: hybrids thus have a lower fitness than specialists and should be selected against. In other words, allowing $z$ to evolve, we expect reinforcement to
increase $z$ after secondary contact. However, assuming an instantaneous well-mixed secondary contact (no window of partial secondary contact, $T_J = 0$), hybrids replace the population in only a few generations (<10 generations) for a wide range of parameters (see Appendix D, Table D2). In addition, the mean assortative trait of the population remains very close to 0 before all specialists are replaced by generalist hybrids. Hybrids are only weakly disadvantaged: their birth rate is only a few percent lower than that of specialists, e.g. 10% with $z = 1.8$ and maximal ecological differentiation between specialists. Reinforcement is then weak because this hybrid disadvantage is too small compared to the speed of hybrid production: under random mating ($z = 0$), in each generation, at least half the offspring of each specialist are hybrids.

Figure 5 (right panel) shows that assuming a window of partial secondary contact increases the speciation probability. This is because during the window of partial secondary contact, the production of hybrids by specialists is limited, so that reinforcement has enough time to act, then increasing the mating trait of the population. The longer the duration of the window of partial secondary contact, the longer reinforcement acts, and the higher the mating trait of the population, until it levels off for long windows of partial secondary contact (Fig. 5, left panel). When the mating trait reaches a higher value than the threshold $z_{th}$, speciation is successful, otherwise it is not. After one secondary contact, the probability of successful speciation remains rather small (<0.2, Fig. 5, right panel). As long as a hybrid population persists, it can still grow and replace the population of specialists: the production of generalist hybrids by specialists is reduced, but the production of generalists by hybrids is not. Consequently, if assortative mating does not evolve on a short time scale at secondary contact, the hybrid population is likely to replace the population of specialists before the end of the window of partial secondary contact. Reinforcement is then no longer efficient and speciation is likely to fail. As a result, the probability of speciation remains small, and increasing the duration of partial secondary contact to very high (unrealistic) values does not make speciation more likely (Fig. 5, right panel). Note that the probability of speciation depends on the strength of selection at secondary contact: when disruptive selection is weak, speciation never occurs, and the stronger selection, the higher the probability of speciation (Appendix D, Fig. D7).

**Sympaty-allopatry oscillations**

Despite the impediments to speciation after one secondary contact, persistent ecological speciation is a likely outcome of recurrent landscape dynamics: Fig. 6 (right...
panels) shows that the probability of speciation after six allopatry–sympatry oscillations is higher than 0.45 (vs. 0.2 after one oscillation). At each secondary contact, positive assortative mating is selected. Assuming a negligible cost to assortativeness as we do here, there is neither selection for nor against assortative mating during allopatric phases. After enough allopatry–sympatry oscillations, assortative mating is thus likely to be higher than the threshold \( a_{\text{lim}} \) allowing successful speciation. In other words, oscillation between allopatry and sympatry allows several reinforcement ‘shots’, increasing the likelihood of speciation. Note that the likelihood of speciation is determined by the total duration of reinforcement, which is not equivalent to the total duration of partial secondary contacts. As explained above, generalist hybrids can replace the population of specialists before the end of a window of partial secondary contact, stopping reinforcement. Consequently, many short windows of partial secondary contact due to allopatry–sympatry oscillations lead more efficiently to speciation than a few long windows of partial secondary contact (Fig. 6, top panels versus bottom panels). We have checked that using smaller mutation rates than the mutation rate used in Fig. 6 (\( \mu_a = \mu_s = 0.01 \)) does not change our results, except in terms of absolute time scales (see Appendix D, Fig. D8).

Once ecological differentiation and speciation have occurred, further landscape changes affect populations but not the total level of diversity (see typical evolutionary trajectory in Appendix D, Fig. D9). In sympatry, disruptive selection maintains ecological divergence and a high value of the mating trait. In allopatry, both species can coexist in each patch, but one species may go extinct. The species specialized on the less abundant resource has indeed a small population size, so that it may become extinct by demographic stochasticity. Nevertheless, looking at the whole metapopulation, both species persist.

In a static, allopatric landscape, a population can become ecologically differentiated, but prezygotic reproductive isolation between geographically isolated sub-populations may not be selected for. In a static, sympatric landscape, a population may be stuck at a fitness minimum because of genetic constraints. An equilibrium state with two specialist species exists, but it is unattainable. In a dynamic landscape, a first landscape change allows such a population to diverge ecologically. A second landscape change allows the evolution of reproductive isolation. Eventually, the two-species equilibrium may be attained, and this is more likely after several landscape changes that increase assortative mating sequentially. In conclusion, under a strong trade-off, landscape dynamics can facilitate ecological speciation, a state that may be unattainable in a static, allopatric or sympatric landscape.

**Discussion**

Under stabilizing selection (resulting in our model from a weak trade-off between the use of the two resources), speciation can occur in allopatry, but the coexistence of the two new species is evolutionarily unsustainable in a sympatric landscape (i.e. after secondary contact). We showed that landscape dynamics (allopatry–sympatry oscillations) may facilitate their long-term coexistence. In particular, landscape dynamics preserve allopatric speciation given certain characteristic time scales (long allopatry, short sympatry). Also, the maintenance of speciation is facilitated by similarly sized subpopulations.
upon secondary contact. Under disruptive selection (resulting from a strong trade-off between the use of the two resources), landscape dynamics generate diversity more readily than a fixed, sympatric landscape can: a shift from sympatry to allopatry stops gene flow, allowing ecological divergence that may be impossible in sympatry due to genetic constraints. When a mechanism allowing extrinsic ecological reinforcement to occur at secondary contact exists (e.g. a temporarily reduced meeting probability between ecologically differentiated individuals), speciation is also more likely than in a static, allopatric landscape. Landscape dynamics facilitate the evolution of reproductive isolation between ecologically differentiated subpopulations by offering many opportunities (i.e. at each secondary contact) for reinforcement to be successful.

Standard, verbal scenarios of speciation usually hypothesize that speciation is initiated in allopatry and completed in sympaty, or conversely (Rundle & Nosil, 2005). Accordingly, different authors have pointed out the necessity to take into account the temporal dimension of speciation in models because of likely shifts in the geographical arrangement during the process (e.g. Schluter, 2001; Rundle & Nosil, 2005; Bolnick & Fitzpatrick, 2007; Fitzpatrick et al., 2008). Despite these claims and ample empirical evidence supporting them (e.g. Taylor & McPhail, 2000; Feder et al., 2003; Baack, 2004; Stuessy et al., 2004; Jordal et al., 2006; Xie et al., 2007; Grant & Grant, 2009; Rundell & Price, 2009), almost all models of speciation focus on a single geographical arrangement. Initial ecological and evolutionary conditions are assumed, as well as the evolution of the population in the considered geographical arrangement. Our results show how constraining these assumptions are: past and future geographical arrangements are likely to considerably alter the likelihood of speciation, as well as its long-term maintenance.

Given the way we derived the resource distributions (Appendix B), our model implicitly assumes that individuals are not limited in their intrinsic migration capabilities. Allopatry corresponds to an extrinsic constraint geographically isolating two patches holding two different principal resources. Sympatry corresponds to two patches with unlimited migration between them, justifying the assumption of two resources of the same abundance. Migration is, however, a life-history trait subject to evolution (e.g. Roff, 1990). We could thus have considered a model with oscillations between allopatry and parapatry (with two patches holding two different principal resources) and evolving migration. In this case, under stabilizing selection (weak trade-off case), migration may be selected against at secondary contact because, after specialization in allopatry, migrants are poorly adapted in the patch they reach (Maynard Smith, 1966; Balkau & Feldman, 1973). This would result in permanent (intrinsic) geographical isolation, allowing both specialists to persist in the long term.

In addition, if reproductive isolation is not yet complete at secondary contact, a sufficiently low migration rate between subpopulations may induce their genetic divergence and possibly speciation (e.g. Felsenstein, 1981; Kirkpatrick & Ravigné, 2002; Gavrilets, 2004). Still considering stabilizing selection, specialization may be selected against if migration remains high (Maynard Smith, 1966; Balkau & Feldman, 1973). Weakly specialized individuals should not be too poorly adapted in either habitat to persist. These two alternative strategies (low dispersal, high specialization and high dispersal, low specialization) might also coexist and even appear by evolutionary branching (Mathias et al., 2001). When selection is disruptive in each patch (strong trade-off case), the two different specialists are selected for in each patch. The results assuming allopatry–parapatry oscillations would thus be the same as those reported in the Results section assuming allopatry–sympatry oscillations.

**Landscape dynamics and speciation under stabilizing selection**

Under a weak trade-off, diversity can be maintained at a secondary contact if reproductive isolation has evolved in allopatry and is maintained (i.e. if allopatric speciation has occurred). In our model with an evolving mating trait, reproductive isolation is equivalent to assortative mating between ecologically differentiated individuals. Under a weak trade-off, assortative mating is selected neither in allopatry nor at secondary contact. Because our model does not allow for the evolution of assortative mating based on an ecological trait, we have assumed that reproductive isolation evolves during allopatric stages to be able to study the consequences of secondary contact. Making this assumption does not weaken our results because some other mechanisms, not included in our model, could lead to prezygotic reproductive isolation in allopatry or at secondary contact. Assuming a fixed positive mating trait at secondary contact allows for such mechanisms. For our conclusions to remain valid, such mechanisms must allow the evolution of strong prezygotic reproductive isolation on a short time scale and must satisfy eqn (3) that describes a one-allele mechanism (mating probabilities directly depend on similarity on an ecological trait). These mechanisms may be e.g. temporal or pollinator reproductive isolation (Coyne & Orr, 2004). Equation (3) may also be used for two-allele mechanisms (mating probabilities depend on similarity on a marker trait linked to the ecological trait) when the maker trait is not genetically coded, but determined by parental imprinting (culturally inherited). In this case, recombination does indeed not break the linkage between the ecological trait and the marker trait (note that other mechanisms, such as drift of the marker trait, may nevertheless break the linkage in the long term; eqn (3) may thus be satisfied only on a short time scale). For example, in Darwin’s finches, each ecotype
sings a specific song, and mating is mainly based on similarity on song that is culturally inherited (Grant & Grant, 1996). Founder effects on song when populations become allopatric or at secondary contact may generate strong prezygotic reproductive isolation. An example in the medium ground finches on the Daphne Major island (Galápagos islands) was recently reported by Grant & Grant (2009): in 1981, an immigrant finch with an unusually large beak (ecological trait) and an unusual song (marker trait) arrived on the island; only seven generations after this founder event, the descendents of the immigrant were strongly reproductively isolated from the resident medium ground finches who have a smaller beak and sing a different song.

Assuming a weak trade-off, we showed that the coexistence of two ecologically differentiated species is ecologically possible at secondary contact, but evolutionarily unsustainable. Our results highlight the conditions necessary to maintain diversity in sympathy for a long time: large allopatric divergence, weak stabilizing selection and little genetic drift. Our model assumes that the abundances of the two resources are the same (or at least similar) in sympathy; this assumption facilitates the persistence of species at secondary contact. Without this assumption, the population would evolve to a specialist strategy despite being in sympathy. Consequently, the sets of strategies allowing for an ecological coexistence of the two species would not be symmetrical with respect to the singular strategy in sympathy. Extinction of one of the species in sympathy would thus be more rapid.

Some other model studies have concluded that after an allopatric speciation event, species can ecologically coexist at secondary contact (i.e. no competitive exclusion), but that their coexistence is evolutionarily unsustainable. For example, Mougi & Nishimura (2007) showed that a trade-off on life-history traits not directly related to competition can lead to evolutionarily unsustainable coexistence. As in our model, this result is strengthened by the fact that one subpopulation evolving faster than another drives the latter to rapid extinction. They conclude that evolutionary coexistence is unlikely to occur. We agree with this conclusion, assuming a static, sympatric landscape (and stabilizing selection). As we previously argued, however, landscape dynamics may be common and long-term coexistence could thus be more likely than suggested by previous models.

**Landscape dynamics and speciation under disruptive selection**

Under disruptive selection in sympathy (strong trade-off case), we showed that landscape dynamics allow ecological divergence, i.e. allow the population to escape from a fitness minimum. Populations may find other solutions to escape from a fitness minimum (Rueffler et al., 2006), including the following: evolutionary branching (Geritz et al., 1998; Dieckmann & Doebeli, 1999), the evolution of sexual dimorphism (Bolnick & Doebeli, 2003; van Dooren et al., 2004), the evolution of genetic polymorphism (Kisdi & Geritz, 1999; van Doorn & Dieckmann, 2006; Claessen et al., 2008), the evolution of dominance which allows the emergence of specialists (van Dooren, 1999; Peischl & Bürger, 2008; Durinx & van Dooren, 2009; Peischl & Schneider, 2010), and the migration of a reproductively isolated population leading to character displacement (Aguilé et al., 2011).

The scenario of ecological speciation we proposed differs from adaptive speciation in which speciation is an adaptive response to frequency-dependent biological interactions generating disruptive selection (Dieckmann et al., 2004): our scenario of speciation indeed requires both allopatric and sympatric phases whereas adaptive speciation necessarily occurs in sympathy only. In our scenario, a dynamic landscape allows an escape from a fitness minimum, whereas a static landscape would have fixed the population at the minimum. This situation can happen mainly for two reasons. First, as we have assumed, a population can stay at a fitness minimum without being able to split into two branches (as with evolutionary branching) because of genetic constraints (free recombination and polygenic inheritance with small allelic effects; Waxman & Gavrilets, 2005). Second, a population can be locked on a fitness minimum because of small population size (Claessen et al., 2007, 2008; Johansson et al., 2010). Evolutionary branching can indeed be strongly delayed by demographic stochasticity, which affects small populations with sizes similar to those occurring in our simulations (usually ≤ 1000 individuals). By relaxing the genetic constraints and with increased population sizes, sympatric evolutionary branching would become possible. Because once speciation has occurred, landscape dynamics have no effect on the total level of diversity, the effect of landscape dynamics would then be undetectable (unless the level of ecological differentiation is not the same in sympathy and allopatry, which is not the case in our model).

Two of our model assumptions facilitate the evolution of assortative mating. First, we assumed that the mating trait evolves via a one-allele mechanism, i.e. the assortative mating level is directly related to the ecological trait. Such an ecological trait is sometimes called a ‘magic trait’ (e.g. Gavrilets, 2004): this hypothesis indeed facilitates the evolution of assortative mating (Felsenstein, 1981; Dieckmann & Doebeli, 1999; Servedio, 2000). This assumption has long been debated in the literature and seems now accepted as possible (e.g. Kirkpatrick & Ravigné, 2002; Servedio et al., 2011). Moreover, some authors (Schneider & Bürger, 2006; Kopp & Hermisson, 2008) demonstrated that the evolution of positive assortative mating is difficult with a high cost of being choosy. Because we consider only a small cost to (dis-)assortative mating, we probably overestimate the ease of its evolution compared to natural populations.
We have focused our analysis of the evolution of the mating trait on phases of disruptive selection on the ecological trait. The mating trait may also evolve during phases of directional selection on the ecological trait. Our focus is relevant because in our model, selection on the ecological trait is directional from a landscape change to the moment the population reaches its new singular strategy, and the mating trait changes very weakly during these phases (Appendix D, Fig. D9). Further analysis would nevertheless be helpful to precisely investigate selective pressures on the mating trait when selection on the ecological trait is directional.

We have shown that reinforcement is inefficient when assuming a well-mixed fusion of the allopatric subpopulations at secondary contact. Due to the homogenizing effect of gene flow, full sympatry is indeed known as the environment most hostile to reinforcement (Felsenstein, 1981; Liou & Price, 1994; Servedio & Kirkpatrick, 1997; Kirkpatrick, 2000; Kirkpatrick & Ravigné, 2002). A mechanism allowing reinforcement to be efficient at secondary contact is thus necessary. Here, we propose a window of partial secondary contact, i.e. a temporary reduction in the meeting probability between specialist individuals. We argue that any other assumption generating reinforcement at secondary contacts would have led to the same results. The probability of speciation is ultimately determined by the total time during which reinforcement is efficient. Because reinforcement may fail to lead to speciation even under suitable initial conditions (in particular a large ecological divergence at secondary contact), landscape dynamics facilitate speciation by repetitively generating these suitable initial conditions. Consequently, for a similar duration of possible reinforcement, allopatry–sympatry oscillations lead more easily to speciation than a one-off secondary contact. Note also that, when reinforcement is efficient because of a reduced introgression rate, we found an optimal introgression rate (see Appendix D, Fig. D10).

Kirkpatrick (2001) who proposed another model with reinforcement on an ecological basis also found that reinforcement is possible only under this restrictive assumption (despite important differences with our model: Kirkpatrick’s model assumes e.g. haploid individuals, a fixed phenotypic variance and that hybrids mate with only one of the ancestral subpopulations).

Empirical data on speciation under landscape dynamics

An array of empirical studies support the outcomes of our model. We first discuss examples where allopatry first generates diversity but then sympatry causes its collapse. Next, we show examples where allopatry generates diversity and sympatry maintains part of it, followed by cases resulting in complete and persistent speciation. Finally, we discuss possible signatures of landscape dynamics in the present-day empirical data.

A study by Gow et al. (2006) on three-spined sticklebacks (Gasterosteus aculeatus) in Paxton Lake (Texada Island, Canada) showed oscillations of diversity correlated with landscape dynamics. Due to the adaptation to different habitats within the same lake, sticklebacks were differentiated into limnetic and benthic morphs and were geographically isolated in these habitats until the late 1950s. Then, human disturbance reduced the differences between the habitats of the lake. This landscape change from an allopatric-like state to a sympatric-like state increased the hybridization rate, despite hybrids having a reduced fitness, leading to a strong diversity reduction. Sticklebacks were indeed not reproductively isolated by assortative mating. Disturbance stopped in the 1970s, environmental conditions were then close to initial ones, i.e. differentiated habitats were restored, and sticklebacks have differentiated again: a transition from sympathy to allopatry regenerated the lost diversity. This empirical example illustrates a feature of our model: allopatry easily generates diversity, but in the absence of previously evolved reproductive isolation, the maintenance of incipient species at secondary contact is far from certain, even under disruptive selection. Seehausen et al. (2008a) reviewed other examples of diversity reduction due to hybridization. This is usually associated with a very fast loss of environmental heterogeneity, i.e. secondary contact without reduced introgression. Empirical data reveal that this process occurs very quickly. Accordingly, with the removal of reproductive isolation, our results predict diversity collapse in a few generations at such a secondary contact for cases of both stabilizing and disruptive selection.

Esselstyn et al. (2009) analysed the effect of landscape dynamics on South-east Asian shrews (Crocidura). During the Miocene–Pliocene, volcanic uplift produced many new islands in South-east Asia, and during the Pleistocene, repeated sea level fluctuations have temporarily connected islands. These landscape dynamics have constantly produced new available ecological niches, but the present diversity is less than the number of ecological niches that have probably been produced. In this example, allopatric phases are indeed likely to have been long enough (on the order of thousands of generations) to produce strongly differentiated populations. However, sympatric phases are likely to have been equally long. Our results show that two populations that come into sympatry are expected to persist on such a long time scale only under disruptive selection (and assuming that reproductive isolation has evolved in allopatry: without reproductive isolation, coexistence is impossible regardless of the shape of the fitness landscape). Given the possible high number of secondary contacts and the fact that stabilizing selection may also have been at work, it is unlikely that all populations that have differentiated in allopatry still exist nowadays. Data analyses of Esselstyn et al. (2009) reached the same conclusion. The effect of similar geological and climatic processes (volcanic events...
and sea level fluctuations) have been analysed by Cook (2008) in the Atlantic Madeiran archipelago. She reached similar conclusions: the high diversity of Madeiran land snails is likely to result from numerous geological and climatic changes, mainly because of many allopatric divergence opportunities.

Young et al. (2009) analysed the diversity of mbuna cichlid fishes in lake Malawi in relation to landscape dynamics: water level fluctuations repeatedly divided and reconnected entire communities during some hundreds of years. Cichlid fishes usually mate assortatively, according to their body colours, which is correlated with their ecological behaviour (Seehausen & van Alphen, 1998; Seehausen et al., 2008b; Egger et al., 2010). Based on the analysis of a matrix of pair-wise interaction coefficients for native and transplanted mbuna cichlid species, Young et al. (2009) suggested that the total diversity increased by community splitting, facilitating allopatric divergence, and that the local diversity also increased, at secondary contacts, by bringing reproductively isolated differentiated fishes back together. In this example, sympatric phases are likely to have lasted only a few dozens of generations and, assuming high enough assortative mating, our results show that coexistence in sympathy may indeed be possible on this time scale under both stabilizing and disruptive selection. Young et al. (2009) conclude that landscape dynamics may be the main mechanism responsible for the adaptive radiation of mbuna cichlid fishes of lake Malawi, a radiation that produced hundreds of species.

The different outcomes of our model depend on specific conditions, in particular the time scales of the landscape dynamics. To quantitatively test theoretical predictions of models featuring landscape dynamics, it would be valuable to develop methods allowing us to detect historical landscape dynamics from the present-day empirical data. We give two examples of signatures of landscape dynamics that may be found in data. First, past allopatric phases should make coalescent gene trees strongly deviate from expected unstructured coalescent trees. In particular, as computed in Aguilé et al. (2009), the mean coalescence time of two uniformly sampled neutral genes in a sympatric population can take significantly different values, depending on whether one assumes a past allopatric phase or not. Assuming several past fusions and fissions of populations, these coalescence times are expected to reach unusually high values (see also Jesus et al., 2006). The characterization of the whole shape of the gene tree with landscape dynamics is a particularly challenging task. Second, landscape dynamics could result in different genealogies for different genes, because different genes would coalesce at different sympatric phases, possibly generating incomplete lineage sorting. Quantifying the effect of given landscape dynamics models on these discrepancies would allow us to infer past hybridization periods as well as allopatric phases from genetic data.

Acknowledgments

We thank Frank H. Shaw for helpful suggestions on our manuscript. We are very grateful to two anonymous reviewers whose comments significantly improved the scope and quality of our work. R.A. was supported by a PhD fellowship from the French Ministère de la Recherche et de la Technologie. Travel for R.A. and A.L. was funded by the project Modèles Aléatoires de l’Évolution du Vivant (MAEV) of the French Agence Nationale pour la Recherche (ANR-06-BLAN-3_146282).

References


Under which conditions is character displacement a likely outcome of secondary contact?

Robin Aguilée, Benoît de Becdelièvre, Amaury Lambert and David Claessen

Ecole Normale Supérieure, CERES-ERTI, 24 rue Lhomond, F-75230 Paris Cedex 05, France; Laboratoire Probabilités & Modèles Aléatoires, UMR 7599, UPMC Univ Paris 06, 4 place Jussieu, Case courrier 188, F-75252 Paris Cedex 05, Paris, France

(Received 8 November 2009; final version received 29 April 2010)

Sympatric character displacement is one possible mechanism that prevents competitive exclusion. This mechanism is thought to be behind the radiation of Darwin’s finches, where character displacement is assumed to have followed secondary contact of ecologically similar species. We use a model to evaluate under which ecological and environmental conditions this mechanism is likely. Using the adaptive dynamics theory, we analyse different ecological models embedded in the secondary contact scenario. We highlight two necessary conditions for character displacement in sympathy: (i) very strong premating isolation between the two populations, and (ii) secondary contact to occur at an evolutionary branching point. Character displacement is then driven by adaptation to interspecific competition. We determine how ecological and environmental parameters influence the probability of ecological divergence. Finally, we discuss the likelihood of sympatric character displacement under disruptive selection in natural populations.

Keywords: competitive exclusion; character displacement; ecological speciation; non-adaptive speciation; adaptive dynamics; Darwin’s finches

AMS 2000 Mathematics Subject Classification codes: 92-04; 92D15; 92D25; 92D40

1. Introduction

The proposed scenario for the radiation of Darwin’s finches on the Galápagos islands [14] hypothesizes the divergence of an ecological trait in two populations that came into secondary contact after a migration event [17,31]. This character displacement is thought to be a mechanism that prevents competitive exclusion. As a critical investigation of this scenario, we propose here a model to assess under which conditions the divergence of an ecological trait in two populations that are in sympathy is a likely evolutionary response to interspecific competition during the secondary contact, thereby avoiding competitive exclusion.

The radiation of Darwin’s finches is among the best documented examples of speciation [15]. The currently accepted speciation scenario hypothesizes that the speciation process is initiated...
in allopatry and completed in sympatry according to the three following steps. First, migrants colonize one island of the Galápagos archipelago from the mainland. Second, some individuals disperse onto another island and find a new colony. This step may be repeated several times. Third, migrants from a secondarily colonized island come into sympatry with the original colony. This secondary contact results in successful speciation if two conditions are fulfilled: (a) immigrant and ancestral populations do not interbreed and (b) they stably coexist – that is, no competitive exclusion [8]. Hereafter, we refer to condition (b) as ‘stable coexistence’ as it allows coexistence on an evolutionary time scale.

The second step of this speciation process (i.e. founding of new colonies) may ensure that condition (a) is satisfied. Indeed, finches use their songs – a culturally inherited trait – to discriminate between conspecific and heterospecific individuals. Consequently, song variations should be selected against. Song can nevertheless change at dispersal events as a result of repetitive founder effects. In short, song divergence in allopatry induces premating isolation in sympatry [10–12]. Some morphological differences evolved in allopatry, on which mate choice is based, may also contribute to premating isolation in sympatry [16,18,25].

On the contrary, condition (b) may be difficult to satisfy. One solution would be that migrants occupy an as yet unoccupied ecological niche. The previously cited studies [14,15,17], however, showed that secondary contact causes a divergence in beak size, allowing immigrant and ancestral populations to gradually feed on different resources. This implicitly suggests that no significant ecological divergence had occurred in allopatry. At the time of secondary contact, immigrant and ancestral populations are still ecologically too similar to coexist. They share the same niche and thus one of the populations risks competitive exclusion. According to the accepted scenario, this is avoided because character displacement takes place, in sympatry, such that immigrant and ancestral populations form two different species can stably coexist.

This speciation scenario – evolution of premating isolation in allopatry, followed by sympatry – has been termed ‘non-adaptive speciation’ and has been proposed to be at work for other species too [29]. Rundell and Price [28] suggested that this could be common in some groups of land snails, salamanders, lizards and plants. In each case, a pair of species is ecologically similar while in allopatry. Once in sympatry, competitive interactions result in competitive exclusion. In these cases the question is: can immigrant and ancestral populations avoid competitive exclusion by sympatric character displacement? A success would result in nonadaptive speciation.

We aim here to investigate the conditions under which this speciation scenario is possible. We will only model the key step of secondary contact. We assume premating isolation (possibly partial) of immigrant and ancestral populations. We ask under which ecological and environmental conditions stable coexistence (i.e. on an evolutionary time scale) can be established by character displacement in the two sympatric populations. In the context of the adaptive dynamics theory [9,24], and using numerical simulations, we analyse the secondary contact scenario for two different ecological models, assuming either two discrete resources or a continuum of resources. Our model differs from speciation models built using adaptive dynamics in that (possibly partial) reproductive isolation is already in place at secondary contact. This is a fixed parameter, assumed to have evolved in allopatry. We show that incomplete premating isolation strongly prevents character displacement. Moreover, character displacement is achieved only if populations are stuck at a fitness minimum (i.e. at an evolutionary branching point (EBP)) at the time of secondary contact. Character displacement is then driven by disruptive selection. In these conditions, character displacement is analogous to evolutionary branching, without the need for positive assortative mating to evolve. We determine how other ecological and environmental conditions influence the probability of stable coexistence of the two populations. We finally discuss how the suitable conditions we found for character displacement are likely to be met in natural populations, and in particular in the Galápagos finches populations.
2. Models

2.1. Secondary contact scenario

We consider an initial resident (ancestral) population of \( N_0 \) individuals, monomorphic with ecological trait \( u_0 \). Due to a simple ‘quantitative genetics’ rule for trait inheritance (Section 2.2), the population is no longer monomorphic after a few generations. We let the resident population reach its ecological equilibrium, determined by the interaction with its dynamic food resources. We choose an ecological model such that the trait of the population converges under directional selection to a singular point \( u^* \) [9] where the mutant invasion gradient vanishes (assuming \( 0 < u^* < 1 \)). Depending on our choice of parameter values, selection becomes either stabilizing or disruptive at this point. In the first case, the singular point is a fitness maximum called a ‘continuously stable strategy’ (CSS): all mutants in a resident population at \( u^* \) have a negative fitness, so that they cannot invade the resident population. Selection thus keeps the population at \( u^* \). In the second case, \( u^* \) is a fitness minimum called an ‘evolutionary branching point’ (EBP): all mutants in a resident population at \( u^* \) have a positive fitness, so that they could invade the resident population. At an EBP, an isolated large asexual population is expected to split into two ecologically diverging subpopulations, a phenomenon called ‘evolutionary branching’. We assume that polygenic, diploid inheritance (hybrid formation) prevents evolutionary branching of the resident population, even when the resident is at an EBP. This is achieved by assuming a simple ‘quantitative genetics’ rule for inheritance (Section 2.2), which effectively prevents the population from evolutionary diversification.

Once the resident population is at its ecological equilibrium, \( N_m \) migrants are introduced. Immigrants do not interbreed with residents. In the case of partial premating isolation, this can nevertheless happen with low probability \( 1 - \pi \); parameter \( \pi \) is defined as the level of premating isolation. Immigrants have a trait drawn from a Gaussian distribution with mean \( u_r + d_u \) and standard deviation \( \sigma_m \), where \( u_r \) is the mean trait of the resident population at the time of migration and \( d_u \) is the ecological difference between the resident and immigrant populations. Migrant and resident populations are indeed assumed to be derived from the same ancestral population, as this is the case for Darwin’s finches speciation scenario. The migrant population can have experienced slightly different environmental conditions, resulting in an ecological difference \( d_u \).

A migration event can result in three outcomes: immigrants can either coexist with residents (i.e. absence of competitive exclusion), or go extinct, or replace the residents. If they go extinct, we let the resident population go back to its ecological equilibrium; we then reintroduce migrants. We repeat migration events until successful stable coexistence; or until an arbitrary maximum time limit of 2 million generations is reached. When immigrants replace the residents, the immigrant population is considered as the new resident population and migrants are reintroduced in the same way as previously described.

2.2. Ecological models

The deterministic version of the first ecological model we use is exactly identical to that of Claessen et al. [4]. Let us sum up the main assumptions. Two resources of density \( F_1(t) \) and \( F_2(t) \) are available for a population of density \( N(t) \). The ecological trait \( u \) under evolution is the fraction of time spent foraging on resource 1 (0 \( \leq u \leq 1 \)). Let \( A_1(u) \) and \( A_2(u) \) be the search rate on the two resources, i.e. the volume cleared of resource per time unit per individual with trait \( u \). The foraging ability on a resource is assumed to depend linearly on the time spent foraging on this resource, an assumption that mimics the effect of learning or phenotypic plasticity [4]:

\[
\begin{align*}
A_1(u) &= a_1 + b_1u \\
A_2(u) &= a_2 + b_2u
\end{align*}
\]
The case $b_1 > 0$ and $b_2 < 0$ corresponds to situations such as learning (‘strong trade-off’ on resources, Figure 1). When $b_1 = b_2 = 0$ there is no trade-off on resources, and search rates are independent of $u$. The case $b_1 < 0$ and $b_2 > 0$ means that the foraging ability on a resource decreases with the time spent foraging on this resource; it is biologically less likely than the other cases. Search rates are necessarily positive for all $u$; thus $a_1$ and $a_2$ are positive and $a_1 + b_1 > 0$ and $a_2 + b_2 > 0$. Assuming that the per capita birth rate $\beta(u)$ is proportional to the consumption rate, we have

$$\beta(u) = k_1 F_1(t) A_1(u) u + k_2 F_2(t) A_2(u) (1 - u),$$

where $k_1$ and $k_2$ are the efficiencies of converting food into offspring.

The resources follow a semi-chemostat dynamics. Assuming a monomorphic population of trait $u$, the dynamics of the system is described by

$$\begin{align*}
\frac{dN}{dt} &= (\beta(u) - d) N(t) \\
\frac{dF_1}{dt} &= \delta_1 (K_1 - F_1(t)) - F_1(t) N(t) A_1(u) u \\
\frac{dF_2}{dt} &= \delta_2 (K_2 - F_2(t)) - F_2(t) N(t) A_2(u) (1 - u),
\end{align*}$$

where $d$ is the constant per capita death rate, and $\delta_1$ and $\delta_2$ are the degradation rates of the two resources ($\delta_1 K_1$ and $\delta_2 K_2$ are the renewal rates).

For finite populations, we use a stochastic version of this model. Resources density is assumed to be in quasi-steady state with the current consumer population as in Claessen et al. [4]. The number of individuals depends on a scaling parameter $V$ (‘volume’). We use a birth and death process in continuous time to simulate the evolution of the population. Opposite to Claessen et al. [4], we assume sexual reproduction. Mating pairs are randomly formed according to the premating isolation pattern, and reproduce according to individual birth rates. Offspring trait is drawn from a Gaussian distribution with mean equal to the parental traits arithmetic mean and standard deviation $\sigma$.

We simulate this way the quantitative genetics of the transmission of a polygenic trait. Neither environmental effects nor dominance effects on the ecological phenotype of the offspring are assumed. Under stabilizing selection (CSS), the phenotypic variance of the offspring distribution is expected to decrease, whereas it is expected to increase under disruptive selection (EBP). However, near evolutionary singular points, selection is weak: the variance of the offspring distribution then changes very slowly compared with the population average. As a result, we consider $\sigma_b$ as constant. When premating isolation is not complete ($\pi < 1$), offspring type (resident or immigrant) is sexually inherited. At each birth event, mutation occurs with probability $\mu$, and modifies the offspring trait by a value drawn from a Gaussian distribution with mean 0 and standard deviation $\sigma_\mu$. Offspring sex-ratio is balanced.

Using the adaptive dynamics framework, Claessen et al. [4] showed that the deterministic version of the model has a convergent singular point $u^*$ which is an EBP if $b_1/(a_1 + b_1) > b_2/a_2$ (strong trade-off on resources), and a CSS otherwise (weak trade-off on resources) (Figure 1). In their asexual stochastic version of the model, they get evolutionary branching in an isolated population at an EBP. On the contrary, our sexual stochastic version does not allow for evolutionary branching in a single population. The formation of hybrids of low fitness with intermediate phenotype indeed prevents the trait distribution to split.

In Section 3.3, we compare the results we get with the above two-resource model with a second ecological model assuming a continuous distribution of resources (Roughgarden model [26]). This model assumes a density-dependent logistic growth with carrying capacity $K(u)$ for individuals of trait $u$, a Gaussian function of $u$ with standard deviation $\sigma_K$. Competition is also frequency-dependent: individuals with traits $u$ and $u'$ compete according to a Gaussian kernel $C(u - u')$. 

Figure 1. Trade-off on resources. Left panel: search rates (Equation (1)) $A_1(u)$ on resource 1 (solid line) and $A_2(u)$ on resource 2 (dashed line) vs. fraction $u$ of time spent foraging on resource 1. The top panel shows a situation with a weak trade-off on resources ($a_1 = 1.8, b_1 = 0, a_2 = 2, b_2 = 0.2$), the bottom panel illustrates a situation with a symmetric strong trade-off on resources which mimicks, e.g. learning ($a_1 = 0.25, b_1 = 2, a_2 = 2.25, b_2 = -2$). Right panel: pairwise-invasibility plots (PIPs) with the above trade-offs and $K_1 = K_2 = 2, k_1 = k_2 = 1, \delta_1 = \delta_2 = 1, d = 0.1$. White (resp. black) areas indicate positive (resp. negative) invasion fitness of a rare mutant with trait $u'$, i.e. its per capita growth rate, in a the resident population of trait $u$ at its ecological equilibrium (i.e. along a vertical line). Evolutionary trajectories of the ecological trait can be predicted using PIPs: the population is assumed to evolve by the successive replacements of the resident population by slightly different mutants with positive fitness. In the example with a weak trade-off (top panel), the point $u^* = 0.5$ is a CSS. At this point, all mutants have a negative fitness (i.e. this is a fitness maximum, selection is stabilizing); hence they cannot invade the resident population which should remain on this trait value. In the strong trade-off example (bottom panel) the point $u^* = 0.5$ is an EBP. At this point, all mutants have a positive fitness (i.e. this is a fitness minimum, selection is disruptive): an isolated population could split into two different ecological branches. The way we model sexual reproduction however prevents a single population from evolutionary branching.

The disruptions in ecological traits were simulated with the help of pairwise-invasibility plots (PIPs). Areas in black indicate that the mutant with trait $u'$ can invade the resident population of trait $u$. An isolated population evolves to an EBP if $\sigma_C < \sigma_K$, and to a CSS otherwise [6]. We use a stochastic, sexual version of this model, assuming a polygenic trait $u$ whose transmission occurs as described above for the two-resource model.

### 2.3. Numerical methods

Resources dynamics are assumed to be fast compared with the consumer dynamics ($\delta_i \gg d$). We thus consider that resources are always at their quasi-steady state levels [4]. The birth and death process is simulated as follows. We pick the time until the next event from an exponential distribution with mean equal to the inverse of the total rate at which events occur (sum of individual birth rates $\beta(u)$ and death rates $d$). The occurring event is randomly chosen proportionally to the rate of each possible event (birth or death). When an individual is chosen to reproduce, a potential sexual partner is randomly picked. Potential partners are of the opposite sex and their type (resident or immigrant) is chosen according to the premating isolation pattern.

We assume that migration is a rare event. Consequently, we do not allow more than one immigrant population to exist at the same time. Conditional on non-extinction of the total population, the number $X$ of migration attempts before successful resident–immigrant coexistence follows a
geometric distribution with mean $1/p$ and variance $(1 - p)/p^2$, where $p$ is the probability of stable coexistence after one migration event. For each set of parameter values, we ran 50 replicates. The probability $p$ is estimated by $\hat{p} = 1/\bar{X}$ where $\bar{X}$ denote the mean of $X$ over replicates. The 95% confidence interval $[\hat{p} - 1/(\bar{X} + e); \hat{p} + 1/(\bar{X} - e)]$, where $e = 1.96\sqrt{(1 - \hat{p})/(n\hat{p}^2)}$ are indicated on figures. We consider that resident–immigrant coexistence is stable if both populations are still present after some a priori chosen duration. This duration is chosen long enough to let character displacement be achieved and maintained.

3. Results

By running very long simulations without migration event, we checked that we simulate a stable resident population. As expected, the mean trait value reaches the singular point $u^*$ (CSS and EBP are convergent stable), and then drifts around the singular point indefinitely, because $u^*$ is evolutionarily stable when it is a CSS, and because the formation of hybrids of low fitness prevents the population from evolutionary branching when $u^*$ is an EBP.

3.1. Character displacement under disruptive selection

As expected, stable coexistence of resident and immigrant populations (i.e. on evolutionary time scales) was always associated with character displacement. The mean trait of both populations then diverges on both sides of $u^*$: one is smaller, the other is higher. In other words, the two populations escape from competitive exclusion when they manage to avoid strong competition by specializing on different resources.

When the singular point $u^*$ is a CSS, we never obtain successful coexistence (Figure 2). Either immigrants rapidly go extinct or replace residents. A CSS is indeed a fitness maximum: in a resident population with trait $u^*$, all mutants have a negative (or zero) fitness, i.e. every other strategy is selected against. Character displacement is thus impossible, resident–immigrant coexistence as well.

![Figure 2. Singular point type effect and strength of disruptive selection effect. Probability $p$ of stable coexistence of resident and immigrant populations after one migration attempt vs. absolute values of both slopes $b_1$ and $b_2$ of the trade-off on resources (Equation (1)) with $b_1 = -b_2$. When $b_1 \leq 0$ and $b_2 \geq 0$, the singular point $u^*$ is a CSS. We never observed character displacement in this case. We stopped simulations after 2 millions of generations, which corresponds to a minimum of 10,000 migration attempts. We plotted the corresponding coexistence probability $p$ (necessarily less than $10^{-4}$) as zero. When $b_1 > 0$ and $b_2 < 0$, the singular point $u^*$ is an EBP. Resident–immigrant coexistence is possible in this case. Increasing the absolute values of the slopes of the trade-off on resources with $b_1 = -b_2$ allows one to increase disruptive selection at the singular point $u^*$, and hence probability of resident–immigrant coexistence. Parameter values: $V = 25$ (equilibrium population size $\approx 1000$), $N_m = 40, a_{\infty} = 0.02, a_1 = a_2 - b_1, a_2 = 2.1, K_1 = K_2 = 2, k_1 = k_2 = 1, \delta_1 = \delta_2 = 1, d = 0.1, u^* = 0.5, \mu = 0.01, \sigma_{\mu} = 0.02, \pi = 1, d_u = 0, \sigma_b = 0.01$.](image)
Figure 3. Character displacement under disruptive selection. Typical time series when the singular point $u^*$ is an EBP. The dashed line indicates $u^* = 0.5$. Grey density corresponds to the density of individuals (residents and immigrants summed) of each phenotype. The duration of the simulation is 17,500 generations. Migration events occur at arrows. The fourth migration attempts successfully drives character displacement. Graphs under the time series are the fitness landscape at times indicated by vertical lines. They give the invasion fitness of a rare mutant of trait $u'$ in the resident population at these times. Circles (resp. squares) indicate the mean trait and fitness of the resident (resp. immigrant) population. First graph: directional selection to $u^*$. Second graph: EBP; the fitness of the population sits at a minimum, allowing for character displacement under disruptive selection to happen. Third graph: character displacement at its beginning; fitness associated to the generalist strategy 0.5 and to the weakly specialized residents and immigrants are close. Fourth graph: character displacement ended; immigrant and resident populations lie on a fitness maximum. Parameter values: $N_0 = 10$, $u_0 = 0.05$, $V = 25$ (equilibrium population size $\approx 1000$), $N_m = 40$, $d_0 = 0$, $\sigma_0 = 0.02$, $a_1 = 1.0$, $b_1 = 2$, $a_2 = 2.1$, $b_2 = -2$, $K_1 = K_2 = 2$, $k_1 = k_2 = 1$, $\delta_1 = \delta_2 = 1$, $d = 0.1$, $\sigma_b = 0.01$, $\mu = 0.01$, $\sigma_\mu = 0.02$, $\pi = 1$.

On the contrary, when the singular point $u^*$ is an EBP, character displacement is possible (Figure 2). This point is a fitness minimum: disruptive selection presses to two specialized strategies on both sides of $u^*$. Resident and immigrant populations do not interbreed (or only a little); thus hybrids of intermediate trait are too infrequent to prevent branching of the trait distribution. Consequently the mean resident trait evolves towards a higher or smaller value than $u^*$, the mean immigrant trait to the opposite direction (Figure 3). Disruptive selection drives this character displacement, and then maintains specialization: Figure 3 shows that both branches lie on a maximum of the fitness landscape. Note that the process of character displacement lasts only a few hundreds of generations (assuming a mutation probability $\mu = 0.01$ and a variance of the distribution of mutants $\sigma_\mu = 0.02$; character displacement timescale is proportional to $\mu$ and $(\sigma_\mu)^2$).

3.2. Probability of character displacement

Even if character displacement in sympathy is possible when $u^*$ is an EBP, it is far from being certain. Figure 3 shows, for example, three failed migration attempts. Moreover, the variance of the number of migration events before successful resident–mutant coexistence is rather high. Three elements establish the success or failure of migration: (i) the resident–immigrant hybridization pattern, (ii) the efficiency of disruptive selection and (iii) the strength of drift.

Figure 4 shows that character displacement is likely only for very strong level of premating isolation between resident and immigrant populations. As resident–resident hybrids prevent the resident population from evolutionary branching, resident–immigrant hybrids prevent the two populations from diverging. Hybrids have a generalist strategy and their fitness is close to that of residents and immigrants starting their specialization (see third fitness curve in Figure 3).
Figure 4. The effect of the level of premating isolation. Probability $p$ of stable coexistence of resident and immigrant populations after one migration attempt vs. level of premating isolation $\pi$. When $\pi < 1$, offspring type (resident or immigrant) is sexually inherited (see text). Resident–immigrant coexistence probability decreases to very low values for still very strong premating isolation level. Parameter values: $V = 25$ (equilibrium population size $\approx 1000$), $N_m = 40$, $d_u = 0, \sigma_m = 0.02, a_1 = 0.1, b_1 = 2, a_2 = 2.1, b_2 = -2, K_1 = K_2 = 2, k_1 = k_2 = 1, \delta_1 = \delta_2 = 1, d = 0.1, u^* = 0.5, \sigma_b = 0.01, \mu = 0.01, \sigma_\mu = 0.02$.

Consequently hybrids mate, almost as frequently as non-hybrids, which reduces the overall specialization. As a result, as soon as character displacement begins, hybrids break it. Note that hybrid formation does not break character displacement once the latter has been established: hybrids’ fitness is very low, hence the hybrid population size is negligible, as well as their impact on the trait distribution.

Character displacement in sympatry is easier when disruptive selection is stronger at the time of a migration event, that is, when the competition between individuals at the generalist strategy $u^*$ is more intense and competition between specialist individuals weaker. Figure 2 illustrates this statement. An increase in the absolute values of both slopes $b_1$ and $b_2$ of the trade-off on resources (Equation (1)) means an increase in the ratio of the efficiency of specialist individuals over generalist individuals. As a result, character displacement becomes more likely. Disruptive selection is also more efficient in a population with a high variance of the trait distribution. Then there are more individuals with extreme phenotypes, whose fitness can be significantly higher than individuals of trait close to $u^*$ (see third fitness curve in Figure 3). Consequently they are strongly selected, making character displacement faster and thus easier. Figure 5(a) illustrates this: a high

Figure 5. The effect of the trait distribution. (a) When the variance $\sigma_b$ in the offspring trait distribution is high, individuals of extreme phenotype are frequently formed. They are strongly selected, which speeds up character displacement making it more likely (and even very likely for high $\sigma_b$). (b) A moderate initial ecological difference $d_u$ between residents and immigrants induces a particularly high coexistence probability. The crucial step of character displacement, namely its initiation, is indeed already performed when secondary contact occurs. For $d_u \geq 0.21$, all simulations showed successful coexistence at the first migration attempt. Parameter values: $V = 25$ (equilibrium population size $\approx 1000$), $N_m = 40, \sigma_m = 0.02, a_1 = 0.1, b_1 = 2, a_2 = 2.1, b_2 = -2, K_1 = K_2 = 2, k_1 = k_2 = 1, \delta_1 = \delta_2 = 1, d = 0.1, u^* = 0.5, \sigma_b = 0.01, \mu = 0.01, \sigma_\mu = 0.02, \pi = 1. (a) d_u = 0. (b) d_u = 0.21.
Figure 6. The effect of the strength of drift. (a) Resident–immigrant coexistence is more likely when more immigrants ($N_m$ migrants) arrive at the same time. The risk of extinction of the immigrant population just after its arrival indeed decreases because it experiences less drift. (b) Coexistence is more likely in a large population (high $V$) than in a small one. Parameter values: $d_u = 0, \sigma_m = 0.02, a_1 = 0.1, b_1 = 2, a_2 = 2.1, b_2 = -2, K_1 = K_2 = 2, k_1 = k_2 = 1, \delta_1 = \delta_2 = 1, d = 0.1, u^* = 0.5, \sigma_b = 0.01, \mu = 0.01, \sigma_\mu = 0.02, \pi = 1$. (a) $V = 25$ (equilibrium population size $\approx 1000$). (b) $N_m$ varies with $V$ such that the ratio of immigrant over resident is kept at 0.04. The equilibrium population size varies from about 250 to 2000 for $V$ varying from 6 to 50.

$\sigma_b$ means a strong variance in the trait distribution, so that resident–immigrant coexistence is more likely. Regarding the probability of character displacement, the initial ecological difference $d_u$ between residents and immigrants is one of the most important parameters: depending on its value, coexistence can be almost certain or unlikely (Figure 5(b)). Initiation of character displacement is indeed the most crucial step of character displacement. This can be understood from the fitness curves in Figure 3: the fitness difference between weakly specialized and generalist individuals is much smaller than between fully specialized and generalist individuals. Selection against generalist individuals is thus less efficient at the beginning of character displacement. With a significant initial ecological difference $d_u$, disruptive selection only completes and maintains character displacement.

While disruptive selection induces resident–immigrant coexistence, drift impedes it. A migration attempt can fail because immigrants are initially in small number and hence can become extinct just after migration. A migration event is thus more likely to result in character displacement when many individuals migrate at the same time (Figure 6(a)). If immigrants do not go extinct, stable coexistence can still fail if the immigrants replace the residents. Assuming a weak initial ecological difference between residents and immigrants ($d_u \approx 0$), resident and immigrant fitnesses are approximately equal. Then, if character displacement takes a long time to happen (due to weak disruptive selection), drift is likely to result in ‘fixation’, i.e. extinction of either all immigrants or all residents. Immigrants can thus replace residents purely due to drift. Figure 6(b) illustrates the effect of drift by showing that character displacement is less likely in a small population (low $V$) than in a big one (high $V$).

3.3. Robustness of the results to the ecological model

To check that our results only depend on the secondary contact scenario and not on the ecological model, we proceed with the same analysis with the continuous-resource model. Results are qualitatively similar: (i) character displacement is possible only if the singular point $u^*$ is an EBP (i.e. $\sigma_C < \sigma_K$), (ii) very strong reproductive isolation is needed and (iii) the probability of character displacement depends on a balance between disruptive selection efficiency and intensity of drift. Quantitatively, however, for analogous sets of parameter values, coexistence probability is usually a bit higher with the continuous-resource model than with the two-resource model (not shown). There are indeed only two ecological niches in the two-resource model compared with a
theoretically infinite number of niches in the continuous-resource model. Character displacement is consequently easier with a continuum of resources. Also because of this difference in niche availability, a third type (and more) can settle in the continuous-resource model, whereas only two specialized types can coexist with the two-resource model.

4. Discussion

We have shown that sympatric character displacement in two ecologically similar populations is a mechanism that prevents competitive exclusion under two restrictive conditions. First, populations should be stuck at a fitness minimum, i.e. at an EBP. Second premating isolation between the two populations should be very strong. We have also shown that character displacement is easier when disruptive selection is strong due to intense competitive interactions; when the variance in the offspring trait distribution is high; when ecological divergence has been initiated in allopatry; and when immigrant and/or resident populations are large. Below we argue that these necessary and promoting conditions are likely to be met in natural populations. Note that using two different ecological models and large ranges of parameter values, we have also shown that our results are robust; they do not depend on the ecological details assumed.

While coexistence of resident and immigrant population on an ecological time scale is a prerequisite for character displacement, their coexistence on an evolutionary time scale is a consequence of character displacement. In particular, when the resident is at a CSS, the coexistence of ecologically differentiated immigrants and residents may be possible, but convergent evolution of residents and immigrants will eventually result in the extinction of one of the populations through competitive exclusion. Only when the resident is at an EBP is coexistence probable on both ecological and evolutionary time scales, the latter mediated by character displacement.

An increasing number of field studies have shown that EBPs are not rare [29]. When an EBP exists, populations are expected to converge to it since an EBP is convergent-stable [9]. Populations may find different solutions to escape from this fitness minimum [27]: evolutionary branching, which solved theoretical issues regarding sympatric speciation [6], the evolution of sexual dimorphism [2], the evolution of genetic polymorphism [5, 23] and the evolution of dominance which allows the emergence of specialists [7]. We have characterized here another solution: the migration of a reproductively isolated population, leading to character displacement.

This last scenario requires that the population remains at an EBP until a migration event (i.e. that the other above mechanisms do not act). This can happen for essentially two reasons. First, a population can stay at an EBP without being able to split into two branches because of genetic constraints. Waxman and Gavrilets [32] stressed that this should be the case for traits under polygenic control. Traits establishing the ecological niche of a population are usually complex, so that it is not senseless to think that many genes are involved. In the quite simple case of Darwin’s finches, beak size and shape set the ecological niche through diet. Polygenic control is known for these traits [15]. Second, a population can be locked on an EBP because of small population size [4, 5]. Demographic stochasticity is significant in small populations, which delays evolutionary branching. It may be the case for Darwin’s finches. Grant et al. [20] evaluate that the finch population size of two species (G. fortis and G. scandens) on the island Daphne Major varies from 300 to 3000 individuals (our simulations assume 1000 individuals). This is a small enough population size to observe delayed evolutionary branching according to the studies of Claessen et al.

In agreement with Grant and Grant [11, 15], we have assumed sexual imprinting of mating preferences for resident–immigrant hybrids. However, the result that very strong premating isolation
is required for character displacement does not rest on this hypothesis: as soon as individuals of intermediate phenotype exist (hybrids), character displacement is prevented. Field studies already pointed out the necessity of strong premating isolation. In the case of Darwin’s finches, some of them demonstrate the existence of premating isolation [12] whereas many others attest hybrid formation [10,14–16,19]. It seems thus reasonable to think that assortative mating is not absolute at the time of secondary contact, but that it may evolve through reinforcement. Reinforcement should be fast enough so that the immigrant population does not go extinct or does not replace the resident population. Reinforcement can result from selection against hybrids that carry genetic incompatibilities reducing their fitness (intrinsic reinforcement). Grant and Grant [14], however, demonstrated no genetic loss of fitness in Darwin’s finches hybrids. Here we postulate reinforcement due to increased competition generated by hybrid formation, i.e. extrinsic ecological reinforcement [22,29].

Regarding the initial ecological divergence at the time of secondary contact, our simulations assume the most constraining hypothesis: no ecological divergence. We have however shown that a small ecological difference is enough to significantly increase the probability of character displacement. In this case, populations do not occupy exactly the same niche: they have almost already avoided competitive exclusion. Allopatric populations spreading into sympatry are often known already to have initiated an ecological divergence in allopatry due to different local environmental and/or ecological conditions [28–30].

We have shown that character displacement may occur in only a few hundreds of generations. As a result, even if character displacement is unlikely, considering a reasonable timescale, we can expect many secondary contacts before one of them successfully leads to character displacement. For example, Darwin’s finches radiation has lasted for 2.8 million years [13]. Numerous migration events could have occurred before at least one successful character displacement. Moreover, we may significantly overestimate the time needed for character displacement: Grant and Grant [17] directly observed it on Daphne Major island in a period of time of only 22 years.

We have focused on Darwin’s finches because their radiation scenario is well-documented, allowing us some comparisons with our results. Note, however, that our model may allow to better understand speciation in other species as well. As already mentioned, the same scenario is thought to take place in many non-adaptive radiations [28]. Moreover, ring species, which consist of two reproductively isolated populations connected by a chain of interbreeding populations, are considered as the continental analogues of the Galápagos finches [15,21]. The two reproductively isolated terminal populations that come into secondary contact may be ecologically similar. Competitive interactions may result in competitive exclusion. Ecological interactions could then be a source of disruptive selection.

Finally, we should mention that our model implicitly assumes constant environmental conditions. This should not be the case over a long period of time. For example, during Darwin’s finches radiation, global cooling and warming periods alternated, some islands in the Galápagos archipelago have been submerged, others emerged [3]. It would thus be valuable to incorporate in our model a dynamic landscape framework [1], that is, the opportunity for several populations to merge and split repeatedly. Such dynamic landscapes may lead to repetitive character displacement in a dynamical equilibrium. Populations may then avoid competitive exclusion under much broader environment conditions.

Acknowledgements

Robin Aguilée is supported by a PhD fellowship from the French Ministère de la Recherche et de la Technologie. Travelling was funded by the project Modèles Aléatoire de l’Évolution du Vivant (MAEV) of the French Agence Nationale pour la Recherche (ANR-06-BLAN-3_146282). We thank Marianne Mugabo for helpful comments on previous versions of the manuscript.
References


Landscape dynamics as a mechanism
to explain adaptive radiations

Robin Aguilée\textsuperscript{a,b,*}, David Claessen\textsuperscript{b,1} and Amaury Lambert\textsuperscript{c,2}

November 14, 2011

\textsuperscript{a} Institut des Sciences de l’Évolution de Montpellier (UMR 5554), CNRS, Montpellier, France
\textsuperscript{b} Laboratoire Écologie et Évolution (UMR 7625), UPMC Univ Paris 06, Ecole Normale Supérieure, CNRS, Paris, France
\textsuperscript{c} Laboratoire Probabilités et Modèles Aléatoires (UMR 7599), UPMC Univ Paris 06, Paris, France.

\* Corresponding author. Institut des Sciences de l’Évolution de Montpellier, Université Montpellier II, Place Eugène Bataillon, 34095 Montpellier Cedex 5, France. Tel: +33 4 67 14 32 50. Fax: +33 4 67 14 36 22. E-mail: robin.aguilee@univ-montp2.fr
\\textsuperscript{1} Ecole Normale Supérieure, CERES-ERTI, 24 rue Lhomond, 75230 Paris Cedex 05, France. Tel: +33 1 44 32 27 21. Fax: +33 1 44 32 27 27. E-mail: david.claessen@ens.fr
\\textsuperscript{2} UMR 7599, UPMC, 4 place Jussieu, Case courrier 188, 75252 Paris Cedex 05, France. Tel: +33 1 44 27 85 69. Fax: +33 1 44 27 72 23. E-mail: amaury.lambert@upmc.fr

Abstract

Radiations have been explained assuming either a great diversity of habitats, or sympatric diversification under rather special conditions (e.g. few loci determining phenotypic traits). We propose a new scenario based on the biogeographic changes of the Great African Lakes where cichlid fishes radiated. In our model, the landscape consists of a mosaic of three habitat types which may be separated by geographic barriers or not. We study the effect of the alternation allopatry/sympatry, called “landscape dynamics”, using an individual-based model. We show that landscape dynamics can generate radiations despite a small number of habitat types and inheritance rules impeding sympatric speciation. Diversity is significantly higher than the number of habitats, and higher than diversity reached by sympatric diversification only. Diversity is generated by the joint action of allopatric, ecological divergence, and of disruptive selection in sympathy, allowing for the coexistence of species following reinforcement or character displacement. Landscape dynamics provoke these and other evolutionary mechanisms (e.g. hybridization) in different combinations, possibly increasing diversity at each landscape change. Time scales of the landscape dynamics are the main determinant of the features (asymptotic diversity, time to reach it and its stability) of the radiation. For example, under fast dynamics in a landscape mainly fragmented, the model predicts the temporary collapse of all species into a hybrid swarm.

Running title: Adaptive radiations in dynamic landscapes

Keywords: allopatric divergence, disruptive selection, secondary contact, reinforcement, character displacement, speciation
1 Introduction

Adaptive radiation is the rapid diversification of a single lineage into species with a great diversity of ecological strategies (Schluter, 2000; Rundell and Price, 2009; Losos, 2010). Two main scenarios are currently accepted to explain radiations.

The first scenario involves diversification in sympathy only. Inspired from the radiation of haplochromine cichlid fishes of Lake Victoria, van Doorn et al. (1998) introduced a model for such a radiation. In this model, an ancestral species is subject to disruptive selection on ecological traits, and the evolution of assortative mating is favored by sexual selection. Sympatric speciation then occurs: the population splits into two reproductively isolated populations. The two species stably coexist: their ecological differentiation prevents their extinction by competitive exclusion. Each new species is in turn subject to sympatric speciation, resulting in many sympatric species. This scenario requires however specific conditions, in particular disruptive selection should be strong (Arnegard and Kondrashov, 2004; Coyne and Orr, 2004) and the genetic background of ecological and mating traits should involve very few loci (Coyne and Orr, 2004; Waxman and Gavrilets, 2005).

Another scenario for radiations (Losos and Ricklefs, 2009) assumes that several populations first diverge in allopatry. This divergence involves the ecological traits of individuals adapting to different ecological optima. Divergence in non-ecological traits, involved in reproductive isolation, can also occur (e.g. song drift in allopatric populations of Darwin’s finches (Grant and Grant, 1997)). The second step of this scenario of radiation is secondary contact, which results in sympaty of the differentiated populations. At this stage, reinforcement may reduce the production of unfit hybrids, and/or character displacement may reduce competition between species, ensuring their stable coexistence. Secondary contact repeated between each species differentiated in allopatry is thought to produce a radiation. Under such scenario, the expected number of species in each site equals the number of allopatric sites ecologically differentiated: a radiation requires a great diversity of habitats.

In the latter radiation scenario, the secondary contact step is assumed to be due to a migration event. This is for example the accepted scenario for Darwin’s finches radiation (Grant and Grant, 1997), a scenario which appears to be likely under a broad range of ecological and environmental conditions (Aguilée et al., 2011a). Alternatively, secondary contact may be due to a biogeographic change. A major difference between secondary contact by migration and by biogeographic change is that in the former case, only one species from an allopatric site comes in contact with another species, whereas in the latter case, the entire communities from both sites are in contact. All species may then be subject to new competitive interactions. Each evolutionary processes (i.e. mechanisms leading or impeding the evolution of reproductive isolation and/or ecological differentiation) occurring at secondary contact, in particular character displacement and reinforcement, may involve many species, and each species may be subject to several evolutionary processes. The outcome of such secondary contact (i.e. persistent coexistence of species or not) would be difficult to predict from current theory on the effect of migration, which takes into account only one process at a time, occurring between only two species. A second important difference between migration and biogeographic change is the number of individuals of each species in contact. The coexistence of a migrant population with resident populations may fail due to the small size of the migrant population: the latter may go extinct, e.g. by demographic stochasticity, competitive exclusion or hybridization. At a biogeographic change, populations of sizes of similar order may be in contact, so that extinction may be less likely, or at least slower.

Repetitive biogeographic changes, called “landscape dynamics” and here defined as the repeated alternation of allopatry and sympathy (Aguilée et al., 2009), may be frequent at different spatio-temporal scales. For example, the connections between populations may vary due to glaciations and postglacial secondary contacts (Hewitt, 2000; Young et al., 2002; Zhang et al., 2008). Global climatic variations and geological processes as volcanic events can cause sea level changes, resulting in repetitive separations and fusions of islands (Cook, 2008; Esselstyn et al., 2009). Similarly, persistent fluctuations of water level causing fragmentation and fusion of lakes are known in the Great African Lakes (Owen et al., 1990; Galis and Metz, 1998; Sturmabauer, 1998; Arnegard et al., 1999; Stiassny and Meyer, 1999). At a different spatio-temporal scale, populations can oscillate between allopatry and sympathy due to the establishment of new colonies by dispersal and their later fusion because of e.g. their expansion (DeHeer and Kamble, 2008; Vasquez and Silverman, 2008). The spatial structure of a population can change because of new ecological interactions, e.g. the emergence or extinction of a predator or of a parasite (Batzli, 1992). The contemporary fragmentation and reconstruction of habitats due to human activities also rapidly change the landscapes (Davies et al., 2006).

In the present paper, we address the following questions: Can landscape dynamics cause radiation? If so, how and by which evolutionary processes? We investigate a model with few (three) differentiated habitats, and with polygenic trait inheritance impeding sympatric evolutionary branching.
According to the above scenarios, no radiation should be possible. We will show that landscape dynamics are nevertheless likely to generate a radiation by the joint action of directional selection in allopatry and disruptive selection in sympatry.

## 2 Model and methods

To address our question, our model should be built upon the following five guiding assumptions. First, divergence should be possible in allopatry. This is done by assuming that the landscape consists of six allopatric sites, each one characterized by an ecological optimum chosen among three possible optima defining three habitat types. Second, sympatric divergence should be rare. Sympatric diversification is made unlikely by assuming that phenotypic traits are determined by many independently segregating loci, with small allelic effects (Waxman and Gavrilets, 2005). Third, the evolution of reproductive isolation should be possible. To this end, we allow for the evolution of assortative mating based on ecological traits. Fourth, the model should allow the long-term coexistence of several species in sympatry. We use an ecological model based on Roughgarden (1972)'s model which assumes a continuous distribution of resources and competition for resources, allowing such long-term coexistence. Fifth, the structure and dynamics of the landscape should mimic a documented one. We chose as a reference the Great African Lakes, characterized by repetitive and temporary connections of neighbour sites.

### 2.1 Ecological model

This section describes the population dynamics and evolution in a focal, geographically isolated region. Dependence of the parameters upon landscape structure is detailed in section 2.2 “Landscape model”.

#### 2.1.1 Population dynamics and competition

We use a stochastic, individually-based version of the population growth and competition model of Roughgarden (1972) in a two dimensional ecological traits space (Vukics et al., 2003). In this model, the growth of a population is logistic. Competition for resources is thus density-dependent. Competition is also assumed to be frequency-dependent: individuals with similar traits compete more strongly than dissimilar individuals.

The population in the geographical region under scrutiny consists of \( n(t) \) individuals at time \( t \). Each individual \( i \) is characterized by a vector \( z_i \) of two independently evolving ecological traits \( x_i \) and \( y_i \) determining its resource utilization strategy (see Table 1 for a summary of the notation). These two traits could be for example, for cichlid fishes, their dental morphology and the depth where they feed, which are two traits on which their diet depends (Seehausen and Magalhaes, 2010).

Individual \( i \) gives birth at constant rate \( r \) and dies at rate

\[
d(z_i) = \frac{r}{K(z_i)} \sum_{j=1,j\neq i}^{n(t)} C(z_i - z_j),
\]

where \( K \) is the carrying capacity function and \( C \) is the competition function. Carrying capacity implicitly models a continuous distribution of resources. It is unimodal Gaussian with maximum \( K^* \) at phenotype \( z^* \), referred to as the ecological optimum, and standard deviation \( \sigma_K \):

\[
K(z_i) = K^* \exp \left( -\frac{\|z_i - z^*\|^2}{2\sigma_K^2} \right),
\]

where \( \|z_i - z^*\|^2 = (x_i - x^*)^2 + (y_i - y^*)^2 \). Individuals \( i \) and \( j \) compete according to unimodal Gaussian kernel \( C(z_i - z_j) \) with a standard deviation \( \sigma_C \):

\[
C(z_i - z_j) = \exp \left( -\frac{\|z_i - z_j\|^2}{2\sigma_C^2} \right).
\]

All along this study, we assume that \( \sigma_C < \sigma_K \). Using a deterministic version of the above ecological model (assuming a monomorphic, large population), Vukics et al. (2003) showed that when \( \sigma_C < \sigma_K \) the population is predicted to evolve by directional selection to the ecological optimum \( z^* \) where it then experience disruptive selection. In other words, \( z^* \) is an evolutionary branching point (Geritz et al., 1998).
2.1.2 Reproductive isolation

We model reproductive isolation as in Aguilée et al. (2011b). Pre-zygotic reproductive isolation follows from assortative mating based on magic traits (Servodio et al., 2011). Post-zygotic reproductive isolation is not incorporated into the model. We assume that the population is sexual, and each individual $i$ is characterized by an evolving mating trait $a_i$ determining the degree of choosiness. At each birth event, the individual $i$ chosen to reproduce randomly encounters a sexual partner $j$ among the individuals of the opposite sex in the geographical region under scrutiny. The pair mates with probability $q(i,j)$, which depends on similarity in the ecological traits of the two partners and on choosiness of individual $i$:

$$q(i,j) = \begin{cases} 
\frac{1}{2} \exp \left(-\frac{a_i^2}{2\sigma^2_i}\right) \exp \left(-\frac{\|z_i - z_j\|^2}{2u_i^2}\right) & \text{if } a_i > 0 \\
0.5 & \text{if } a_i = 0 \\
1 - \left(1 - \frac{1}{2} \exp \left(-\frac{a_i^2}{2\sigma^2_i}\right)\right) \exp \left(-\frac{\|z_i - z_j\|^2}{2u_i^2}\right) & \text{if } a_i < 0
\end{cases}$$  \hspace{1cm} (3)

where $u_i = 1/(10\sigma^2_i)$. Individual $i$ has no preference when $a_i = 0$, mates assortatively when $a_i > 0$ and disassortatively when $a_i < 0$. Choosiness increases when $|a_i|$ increases. When individual $i$ rejects partner $j$, another partner is randomly chosen and the process repeats until mating succeeds, or until individual $i$ has rejected 50 potential partners. This represents a very small cost of (dis-)assortativeness: Schneider and Bürgler (2006) and Kopp and Hermisson (2008) showed that giving up mating after rejecting just ten potential partners has a very low cost.

2.1.3 Genetic architecture and inheritance

The genetic architecture and inheritance rules are based on Claessen et al. (2008). Trait $k$ ($k \in \{x,y,a\})$ is determined by $L_k$ diploid, additive loci on autosomal chromosomes. We assume neither environmental effects, nor epistasis, nor dominance effects. Each allele can take any real value. The value of trait $k$ is the mean of the $2L_k$ alleles determining this trait.

We assume independent segregation of each locus; at each locus, one offspring allele is randomly chosen from maternal and paternal alleles. In Results section, we also assume $L_k = 16$, so that each allele has a limited effect on the value of the phenotypic trait. Because of these assumptions, when selection is disruptive, sympatric evolutionary diversification is severely delayed (Claessen et al., 2007, 2008; Johansson et al., 2010) and is expected to happen rarely on the time scales we investigate.

At birth, the offspring’s sex is determined randomly assuming a balanced sex-ratio. Mutation occurs at each locus determining trait $k$ with probability $\mu_k$. The mutant allele value is drawn from a normal distribution with mean equal to the parental allele value and with standard deviation $s_k\sqrt{2L_k}$. This mutation size at the allele level results in a variance $s_k^2$ at the trait $k$ level, regardless of the number of loci (van Doorn et al., 2004).

2.2 Landscape model

In our model, the landscape structure and dynamics mimic, in a very simplified way, the one of the Great African Lakes. These lakes consist of sites holding one of the three major habitat types: a pelagic site surrounded by a ring of rocky sites alternating with sandy sites (Danley and Kocher, 2001; Sturmbauer et al., 2011). Water level fluctuations caused by climatic changes and geological events induced the temporary and repetitive opportunity for individuals to freely move between neighbour sites (Owen et al., 2011). Water level fluctuations caused by climatic changes and geological events induced the temporary and repetitive opportunity for individuals to freely move between neighbour sites (Owen et al., 2011). Water level fluctuations caused by climatic changes and geological events induced the temporary and repetitive opportunity for individuals to freely move between neighbour sites (Owen et al., 2011). Water level fluctuations caused by climatic changes and geological events induced the temporary and repetitive opportunity for individuals to freely move between neighbour sites (Owen et al., 2011).
sum of the carrying capacity functions of each of the sites merged. By successive border disappearance, more than two sites (and up to \( n_s + 1 \) sites) can merge together. Each disappeared border appears again (interpreted as a decrease of the level of water) at constant, exponential rate \( f \). When this event isolates two geographical regions (sites or group of merged sites), individuals are distributed independently of their phenotypes between the two isolated regions in proportion to the maximum carrying capacity of each region.

Given our choice of parameter values in Results section (Table 1), the carrying capacity function is unimodal (but not necessarily Gaussian) whatever the number and the type of habitats of sites merged. In other words, there is always only one ecological optimum for any geographical isolated region. This optimum is intermediate between the optimum of each site merged. For each possible combination of merged sites, we checked numerically that the ecological optimum is an evolutionary branching point under the assumptions of a monomorphic, large population and \( \sigma_C < \sigma_K \) (not shown). As in an isolated site, we thus expect a population in merged sites to evolve by directional selection to an ecological optimum and then to experience disruptive selection.

2.3 Model analysis methods

Our stochastic model is simulated using a birth and death process in continuous time according to Gillespie (1977)’s algorithm. Results are computed over 50 simulation replications running for \( 10^5 \) generations. Details about the simulation procedure can be found in Appendix A.

2.3.1 Types of landscape dynamics

Our model generates different kinds of landscape dynamics depending on the relative and absolute values of parameters \( f \) and \( c \). Because each satellite site is surrounded by 3 borders, when \( f > 3c \) (resp. \( f < 3c \)) sites tend to remain isolated from each other (resp. merged with their neighbour). The higher \( f \) and \( c \), the faster the dynamics. In Results section, we analyze the following five sets of parameter values.

Case 1: fast dynamics in a landscape mainly fragmented, simulated with \( f = 10^{-5} \) and \( c = 10^{-4} \). As \( f \gg 3c \), the landscape is most of the time fragmented, and fusion of sites involves usually no more than two sites (Figure 1a). The expected time spent in states with merged sites is less than 100 generations (Figure 1b), meaning that fragmentation of the landscape rapidly follows fusion of sites. These dynamics are thus fast.

Case 2: as case 1, but slow dynamics \((f = 10^{-3}, c = 10^{-5})\). As \( c \) is small, fusion is a rare event (Figure 1b): on average, more than 10,000 generations are spent in a completely fragmented landscape before a fusion occurs, and fragmentation occurs again after 1,000 generations.

Case 3: fast dynamics in a fragmented landscape with recurrent merged states \((f = 10^{-2}, c = 10^{-3})\). Figure 1a shows that the landscape is most of the time either completely fragmented or only two sites are merged, but a significant proportion of time is also spent in states with more than two sites merged \((f \) is only slightly higher than \( 3c))\). Each state duration is short \((< 100 \) generations, Figure 1b): fragmentation and fusion are thus frequent events.

Case 4: as case 3, but slow dynamics \((f = 10^{-3}, c = 10^{-4})\). Figure 1b shows that each state lasts from 100 to 1000 generations. Fragmentation and fusion are thus rarer events than for case 3.

Case 5: landscape mainly merged \((f = 10^{-3}, c = 10^{-3})\). Because \( f < 3c \), most of the sites are merged most of the time (Figure 1a). Each state lasts from 100 to 1000 generations (Figure 1b).

2.3.2 Diversity estimation

Our estimation of diversity is expressed as a number of species, in the sense of the biological species concept (Mayr, 1942): two populations form distinct species when they are reproductively isolated, or would be if they were into sympatry. Diversity is computed by first grouping together individuals into clusters of phenotypically distinct individuals, then evaluating the level of reproductive isolation between each pair of clusters. A continuous chain of clusters formed by pairs of reproductively not isolated clusters defines a species. This method, detailed in Appendix B, is applied independently in each of the \( n_s + 1 \) sites of the landscape to compute local \((\alpha)\) diversity. Total \((\gamma)\) diversity is computed with the same method, but considering together all individuals of all sites.

2.3.3 Measures used in Results section

We use three measures to characterize the diversity produced in a dynamic landscape: (i) the asymptotic \( \gamma \) diversity which estimates the number of species after the transient phase of diversity increase (i.e. at
the evolutionary equilibrium), (ii) the duration of this transient phase, and (iii) the variance over time of asymptotic \( \gamma \) diversity which quantifies the stability over time of \( \gamma \) diversity after the transient phase.

We used two measures to interpret the above features of the diversity produced in a dynamic landscape: (i) the allopatric divergence which estimates the ecological differentiation between geographically isolated populations subject to directional selection to different ecological optima, and (ii) the smallest ecological distance between species from all sites combined which quantifies the ecological distance between the two phenotypically closest species, whether or not in geographically isolated regions, after the transient phase. This measure is compared to the smallest ecological distance between sympatric species only.

These five measures are averaged over the simulation replications; the method used to compute them is detailed in Appendix C.

3 Results

We first check the expected behavior of the model by analyzing results in a static landscape. Then, these results are used as a benchmark to analyze the effect of landscape dynamics.

3.1 Diversity in a static landscape

Let us first consider a static landscape where all sites are permanently isolated \((f \to \infty, c = 0)\). As expected, allopatric populations in different habitats diverge in their ecological traits under directional selection and rapidly reach their local ecological optimum (in a few thousand of generations, results not shown). Due to populations experiencing disruptive selection when at their ecological optimum, assortative mating based on similarity on the ecological traits increases (Figure 2a), so that after some time, populations in different habitats are reproductively isolated. In other words, allopatric speciation occurs. As we assume 3 different habitats, we expect 3 different species. Figure 2b shows that total \( \gamma \) diversity, averaged over simulation replications, remains however lower than 3. This is due to stochastic reductions of the level of assortative mating that temporarily break reproductive isolation between ecologically differentiated populations. Local \( \alpha \) diversity remains at one species (Figure 2b): as expected from the genetic constraints we assume, sympatric diversification is thus rare.

In a static landscape where all sites are permanently merged \((f = 0, c \to \infty)\), assortative mating increases because of disruptive selection (Figure 2c), but allopatric divergence is obviously impossible. Diversity, averaged over simulation replications, is nevertheless slightly higher than one (Figure 2d) due to sympatric speciation events. Sympatric diversification occurs more frequently than in a static, fragmented landscape because it is favored by a higher local population size (Claessen et al., 2007, 2008; Johansson et al., 2010), and by a higher ratio of the standard deviation of the carrying capacity function over the standard deviation of the competition kernel (Dieckmann and Doebeli, 1999) due to the fusion of all sites together.

3.2 Diversity in a dynamic landscape

Figure 3a shows the time series, averaged over simulation replications, of \( \gamma \) diversity for the five cases of landscape dynamics defined in section 2.3.1 “Types of landscape dynamics”: a dynamic landscape clearly produces a large diversity of species compared to a static landscape, i.e. the total number of species is much higher than the number of habitat types. Table 2 quantifies the three characteristics of \( \gamma \) diversity defined in section 2.3.3 “Measures used in Results section”: asymptotic \( \gamma \) diversity, duration of the transient phase of diversity increase and variance over time of the asymptotic \( \gamma \) diversity depend on the type of landscape dynamics. To understand these differences, let us first focus on the main mechanism producing diversity in a dynamic landscape.

3.2.1 Main diversification mechanism

To understand the mechanism at the root of the increase of diversity, illustrated by Figure 4a, let us first consider a simplified landscape with two sites holding two different habitats. When the landscape is fragmented, allopatric populations in the two differentiated habitats diverge as they adapt to their local ecological optimum. Due to disruptive selection when populations are at their ecological optimum, assortative mating increases. At secondary contact, former allopatric populations may be sufficiently reproductively isolated and ecologically differentiated to stably coexist, i.e. allopatric speciation is successful. At secondary contact, when the two populations are imperfectly reproductively isolated,
reinforcement increasing assortative mating may complete the speciation process, as this happens in the simulation shown in Figure 4a. Alternatively, when the two populations are weakly ecologically differentiated, character displacement may occur, reducing competition between species and allowing their stable coexistence. When the two merged sites become geographically isolated again, the two species coexist in each of the two isolated sites. The two pairs of species then diverge towards the local ecological optimum of each isolated site. In each site, when allopatric divergence ends, as the two local species compete with each other, which generates disruptive selection, none of them is at the local ecological optimum, i.e. where the local species present before fusion of the two sites was. Looking at both sites, the four species present after the second allopatric divergence phase are thus different from the two species present after the first allopatric divergence phase.

Each time this sequence secondary contact – fragmentation is repeated, local diversity may be propagated to other sites, and new species may be generated and maintained by first allopatric divergence, then reinforcement and/or character displacement. Our model considers three differentiated habitats: there is one more direction for allopatric divergence than shown in Figure 4a. When working in our model, the main diversification mechanism described above may thus generate more diversity than in a landscape with two differentiated habitats. In addition, we consider a landscape with \( n_x + 1 = 6 \) sites: local diversity may possibly be different in each site and propagated to other sites by fusion of sites. When working in our model, the main diversification mechanism may thus generate and maintain a higher diversity than in a landscape with only two sites.

Figure 3 (panels a and b) shows that \( \gamma \) and \( \alpha \) diversities do not increase indefinitely, but saturates (except under landscape dynamics of type 2, where diversity does not saturate within \( 10^5 \) generations: this will be discussed later). The main diversification mechanism may indeed fail because of one or several of the three following reasons, illustrated by Figure 4b. First, reinforcement may fail at secondary contact, resulting in the fusion of two populations into a phenotypically intermediate hybrid population (circled populations in Figure 4b). Second, character displacement may fail at secondary contact, so that ecologically close species go extinct due to competitive exclusion (species indicated by an arrow in Figure 4b). Third, when two merged sites holding different habitat types split, one of the two habitats is no more available in each isolated site. As allopatric divergence occurs, species the furthest from the local ecological optimum suffer from maladaptation and may go extinct (squared species in Figure 4b).

Whatever the type of landscape dynamics, the mean mating trait of the population increases to a higher value in a dynamic landscape than in a static landscape (Figure 3c, compared to Figure 2a and 2c). Indeed, in a dynamic landscape, assortative mating increases because populations experience disruptive selection when at their ecological optimum, and also because of reinforcement at each secondary contact.

### 3.2.2 Case 1: Fast dynamics in a landscape mainly fragmented

Table 2 shows that the highest asymptotic \( \gamma \) diversity is generated by fast dynamics in a landscape mainly fragmented (landscape dynamics of type 1, defined in section 2.3.1 “Types of landscape dynamics”). Two reasons explain that this kind of landscape dynamics generates a high diversity.

First, under landscape dynamics of type 1, the main diversification mechanism very efficiently generates and maintains diversity. Indeed, because the landscape is mainly fragmented, populations in different habitats often remain in allopatry long enough to widely diverge (Table 3) and to reach their local ecological optimum where their mating trait increases. Diversity is thus efficiently generated in allopatry, and at secondary contact, populations are likely to be sufficiently ecologically differentiated and reproductively isolated to stably coexist (or, at least, for reinforcement and/or character displacement to be successful).

Second, the set of local species can be different in every site, even in sites holding the same habitat, which results in a \( \gamma \) diversity higher than the \( \alpha \) diversity (Figure 3, panel a vs panel b). Indeed, under landscape dynamics of type 1, sites are geographically isolated most of the time: allopatric divergence is high and the set of local species in sites holding different habitats is thus likely to be different. In addition, sites with the same habitat have the same convergent ecological singular strategy: species in such sites are thus expected to be ecologically very similar. However, because allopatric species do not suffer from competitive exclusion, allopatric sites holding the same habitat can hold different species despite ecologically very close. Table 3 shows this ecological nearness by indicating the smallest ecological distance between species from all sites combined: two species can be ecologically very similar, much more similar than two sympatric species can.

Table 2 shows that the variance over time of the asymptotic \( \gamma \) diversity is the highest under landscape dynamics of type 1. Under such landscape dynamics, the asymptotic level of \( \gamma \) diversity is...
indeed unstable: once diversity has reached its asymptotic level, diversity can temporarily collapse, as this happens in the simulation shown in Figure 3d. Such collapse is due to the fusion of all species into a hybrid swarm, an event illustrated by Figure 4c. As explained above, under landscape dynamics of type 1, ecologically very close species exist in allopatri, but they cannot coexist in sympathy due to competitive exclusion. At secondary contact of such species, either ecological differentiation increases, or species become extinct, or pre-zygotic reproductive isolation decreases. In the latter case, two populations ecologically very close begin to hybridize, which increases the phenotypic variance of the two populations. This consequently decreases the ecological differentiation between them and their ecological neighbors, so that they hybridize with their ecological neighbors also, generating a hybrid swarm which rapidly spreads in this way to the whole niche space. Sympatric speciation finally occurs, generating again some level of diversity. This can however be long to happen: before that, the hybrid swarm may be propagated to all other sites by fusions of sites, destroying all diversity in the metapopulation.

3.2.3 Case 2: Slow dynamics in a landscape mainly fragmented

Under landscape dynamics of type 2, because the landscape is mainly fragmented, diversification is achieved in the same way as under landscape dynamics of type 1. Table 3 shows that allopatric divergence is very high; allopatric populations reach their local ecological optimum where their mating trait increases. Allopatry thus efficiently generates diversity. However, under landscape dynamics of type 2, secondary contacts are rare because the landscape dynamics are slow. Local diversity is thus rarely propagated to other sites, so that the transient phase of $\gamma$ diversity increase is very long (higher than $10^5$ generations, Figure 3a). Because the increase of assortative mating is in part due to reinforcement at secondary contact, assortative mating also increases with delay under slow landscape dynamics compared to fast dynamics (Figure 3c).

3.2.4 Case 3: Fast dynamics in a fragmented landscape with recurrent merged states

Under fast dynamics in a fragmented landscape with recurrent merged states, $\gamma$ diversity is higher than in a static landscape, but remains limited, hardly higher than $\alpha$ diversity (Figure 3, panels a and b). The set of local species are indeed often the same in all sites, even in sites holding different habitats, because of the two following reasons.

First, although allopatric divergence reaches high enough values for the main diversification mechanism to produce diversity, allopatric divergence remains on average restricted (Table 3), so that the set of local species in geographically isolated regions with different habitats are likely to be identical. Allopatric divergence is restricted because fusions of sites are frequent and thus often occur before allopatric populations have ecologically widely diverged. As reproductive isolation is an increasing function of ecological differentiation, when such fusions occur, populations are often not strongly reproductively isolated, and hybridize. The hybrid population may then replace the incipient species, destroying the onset of divergence. The assumption that merged sites, frequent under landscape dynamics of type 3, have intermediate ecological optima also limits allopatric divergence.

Second, allopatric species ecologically very close, who can exist in geographically isolated regions with the same habitat, do not survive for a long time: frequent fusions of sites bring them in contact quickly after their emergence, and competitive exclusion prevents their stable coexistence. As a result, fast landscape dynamics with recurrent merged states maintain a high minimal ecological differentiation between all species from all sites combined, close to the smallest ecological distance between sympatric species (Table 3).

Thanks to this high differentiation between all species, the collapse of diversity because of the fusion of species into a hybrid swarm is unlikely: the low variance over time of the asymptotic $\gamma$ diversity indicated in Table 2 demonstrates that the level of $\gamma$ diversity remains very stable over time, as illustrated by the time series shown in Figure 3d.

Compared to other landscape dynamics types, $\gamma$ diversity reaches its asymptotic level very quickly (Table 2). Indeed, under landscape dynamics of type 3, fusions of sites often occur and often involve many sites, so that as soon as diversity is generated in allopatri, it is propagated to other sites, possibly several sites at the same time, which speeds up diversification. Because of these frequent secondary contacts, reinforcement is frequent, so that the increase of assortative mating is also very fast (Figure 3c).

Despite our assumption that each trait is determined by 16 independently segregating loci with small allelic effects, we observed several sympatric speciation events under landscape dynamics of type 3 (result not shown). Indeed, as allopatric divergence is often restricted, hybridization is frequent at secondary contact. Hybridization increases the genetic variance of the populations. This variability is
often rapidly lost and the hybrid population remains stable. However, a high genetic variance facilitates
the initiation of sympatric speciation, so that when it persists long enough, sympatric speciation occurs,
as illustrated by Figure 4d. Under landscape dynamics of type 3, more than two sites are often merged
together, so that local population size is often high: this also facilitates sympatric speciation.

3.2.5 Case 4: Slow dynamics in a fragmented landscape with recurrent merged states

When the landscape is fragmented with recurrent merged states, the increase of diversity is slower under
slow dynamics than under fast dynamics (Table 3). This is an immediate consequence of the fact that
local diversity is propagated by secondary contacts, which less frequent under slow landscape dynamics.

Compared to landscape dynamics of type 3, allopatric states last longer under landscape
dynamics of type 4, so that allopatric divergence reaches on average higher values (Table 3), and
geographically isolated regions are more likely to hold ecologically close species (Table 3: the smallest
ecological distance between species all sites combined is significantly lower than the smallest ecological
distance between sympatric species). Consequently, geographically isolated regions more often hold
different sets of local species, which results in an higher asymptotic \( \gamma \) diversity (Table 2).

The flip-side of the coin of this low ecological differentiation between all species is that the
stability of the asymptotic \( \gamma \) diversity is weak, as shown by the high variance over time of the asymptotic
\( \gamma \) diversity (Table 2). Hybrid swarms are likely to be generated at secondary contact, destroying diversity,
which explains the great variations of \( \gamma \) diversity of the time series shown in Figure 3d.

3.2.6 Case 5: Landscape dynamics in a landscape mainly merged

Very little \( \gamma \) diversity is generated in a landscape mainly merged (Table 3). Indeed, because most sites
are most of the time merged together, allopatric divergence is weak (Table 3), so that the generation of
new species is rare. As species are most of the time in sympatry, the \( \alpha \) diversity is almost equal to \( \gamma \)
diversity (Figure 3, panel a vs panel b), and all species are highly ecologically differentiated (Table 3).

4 Discussion

Radiations have been explained assuming a great diversity of differentiated habitats (Schluter, 2000).
They have also been explained to occur in sympatry, but under restrictive conditions such as very few
loci determining phenotypic traits (van Doorn et al., 1998; Coyne and Orr, 2004; Waxman and Gavrilets,
2005). Our model makes more parsimonious assumptions: we assume few (three) differentiated habitats,
and many (16) loci determining each trait. We have shown that landscape dynamics — chosen to mimic
the biogeographic changes of the East African Great Lakes — can nevertheless generate a radiation.

During such radiation, diversity is generated by the joint action of allopatric, ecological divergence
under directional selection towards different ecological optima, and of disruptive selection in sympathy
leading to reinforcement and character displacement. Landscape dynamics generate also other mechanisms
producing or restricting diversity: hybridization, sympatric evolutionary branching (induced by hybridization),
and extinction. In a dynamic landscape, all these mechanisms are combined repetitively. Because of their
interactions, they constantly alter the (co)existing species. This results in different initial conditions at
each secondary contact: although secondary contact always occurs between the same three habitat types,
its outcome (i.e. stable coexistence or not of species) changes each time. Consequently, after several
secondary contacts, the conditions necessary to reach each ecological niche are likely to have been
fulfilled, so that the number of species in each habitat can be significantly higher than the number of
differentiated habitats in the whole landscape. In addition, our results show that landscape dynamics
may enable radiation in cases where sympatric speciation is unlikely. The resulting diversity is higher
when radiation is caused by landscape dynamics than when it results from sympatric speciation in a
static landscape: assuming default parameter values (Table 1) except \( L_x = L_y = L_a = 1 \), sympatric
speciation is very easy and produces an asymptotic diversity of, on average, 3.3 species in a static,
merged landscape. Indeed, some ecological niches, attainable by character displacement occurring in a
dynamic landscape, are not attainable by sympatric speciation only.

The characteristics of the diversity produced by a landscape dynamics mechanism are strongly
correlated to the landscape structure and to the characteristic time scales of the landscape dynamics
(Figure 3, Table 2). In particular, the highest asymptotic diversity is generated by fast dynamics in a
landscape almost always fragmented, but diversity is then unstable: it is likely to temporarily collapse
into a hybrid swarm. Diversity is generated the fastest under fast dynamics in a fragmented landscape
subject to fusion of many sites at the same time. In this case, the level of diversity remains very stable,
but limited. Dynamics in a landscape rarely fragmented generate only little diversity.
Models of radiation in differentiated habitats consider a metapopulation with migration. In this case, a subtle balance of migration is necessary to allow diversification (Heaney, 2000; Parent et al., 2008; Losos and Ricklefs, 2009). Assuming a small migration rate, a migrant population is likely to go extinct by demographic stochasticity, or to fail to initiate character displacement when the migrant population is ecologically close to the resident population (Gillespie et al., 2008; Aguilée et al., 2011a). On the contrary, a high migration rate may allow the migrant population to persist. However, allopatric divergence is expected to be strongly limited even for an intermediate migration rate. Consequently, allopatric divergence would be too limited to allow the generation of a radiation, as it is the case for landscape dynamics with sites merged most of the time (case 5 defined in section 2.3.1 Types of landscape dynamics). In addition, migration brings only one species at a time in a new habitat. Only one evolutionary mechanism would then occur. If migration fails, it will occur again, but under the same conditions. Consequently, secondary contact may allow only a limited diversification: some ecological niches may be unattainable because the initial conditions necessary to reach them cannot be generated by a static landscape with migration. Such constraint does not hold in a dynamic landscape.

Mathematical and computational models of speciation usually focus on one specific geographical background (but see Aguilée et al., 2011b), either allopatry or sympatry, more rarely parapatry. Contrary to formal models, standard verbal models of speciation often take into account both allopatry and sympatry (Rundel and Nosil, 2005). Many empirical studies have indeed shown that speciation can be initiated in allopatry and completed in sympatry (e.g. Taylor and McPhail, 2000; Feder et al., 2003; Jordal et al., 2006; Xie et al., 2007; Rundell and Price, 2009), or conversely (e.g. Baack, 2004; Stuessy et al., 2004). In addition, complete speciation, i.e., the evolution of strong and persistent reproductive isolation, can take a significant time during which several biogeographic changes are likely to occur (Bolnick and Fitzpatrick, 2007; Butlin et al., 2008; Fitzpatrick et al., 2008).

Our model constitutes a step towards the modeling of speciation considering biogeographic changes. It includes both allopatric and sympatric speciation. In our model, most speciation events, from their initiation to their completion, result from the combination of evolutionary mechanisms occurring in allopatry (e.g. local adaptation) and in sympatry (e.g. character displacement). While a debate about the relative frequency of allopatric and sympatric speciation is still going on (see e.g. Bolnick and Fitzpatrick, 2007; Fitzpatrick et al., 2009), some authors argue that classifying each speciation event into discrete classes does not help our understanding of the mechanisms of speciation (e.g. Butlin et al., 2008; Fitzpatrick et al., 2008, 2009). We think that defining the geographic background at each step of a speciation event remains informative since this sets the possible mechanisms acting (Rundel and Nosil, 2005; Bolnick and Fitzpatrick, 2007). Our results highlight that the interactions between evolutionary mechanisms are also important to understand how new species arise, and should not be neglected. The study of radiations in dynamic landscapes offer a great opportunity to understand simultaneously the biogeography and the evolutionary mechanisms of speciation.

4.1 Model assumptions and natural populations

Our results assume that populations evolve under disruptive selection. Radiations are thought to occur mainly under such a fitness landscape (Rosenzweig, 1978; Christiansen, 1991; Abrams et al., 1993; Geritz et al., 1997, 1998; Cohen et al., 1999; Ito and Dieckmann, 2007; Rundell and Price, 2009; Losos and Ricklefs, 2009). In contrast, stabilizing selection is unlikely to lead to a radiation, at least via a landscape dynamics mechanism. After allopatric divergence, two species in secondary contact are expected to converge towards the same ecological optimum and may hybridize (Aguilée et al., 2011b). In addition, when they are reproductively isolated, one of them finally goes extinct due to competitive exclusion. Thus, local diversity can increase only temporarily, and the total number of species is expected to remain approximately equal to the number of differentiated habitats. In real landscapes, both disruptive and stabilizing selection may be at work on different ecological traits. Some ecological traits may also evolve under weak directional selection which can favor the generation of diversity by sympatric evolutionary branching (Ito and Dieckmann, 2007).

We assumed that each trait is determined by 16 loci with small allelic effects, which impedes sympatric evolutionary branching (Dieckmann and Doebeli, 1999; Waxman and Gavrilets, 2005; Claessen et al., 2008). In addition, we assume small population sizes (about 500 individuals for each species), so that demographic stochasticity is significant. This is known to strongly delay sympatric evolutionary branching (Claessen et al., 2007, 2008; Johansson et al., 2010). Sympatric evolutionary branching is thus rare in our model, except during periods of strong hybridization. Relaxing constraints impeding sympatric evolutionary branching does not change the stationary state of diversity in a dynamic landscape (data not shown). Diversity is however faster generated, so that the asymptotic diversity level is reached on short time scales, even under slow landscape dynamics.
We defined environmental conditions such that resource distributions significantly overlap between habitat types (Table 1). This assumption is necessary to allow the propagation to other sites of local diversity: without resource distribution overlap, a species adapted to a specific habitat would be too maladapted in another habitat to survive at the time the species reaches this new habitat. Therefore, even if several species could stably coexist in a given habitat, its colonization by different species would systematically fail.

The exact local and total number of species resulting from landscape dynamics in our model should not be understood as predictions. This number is set by environmental parameters ($K^*$, $\sigma_K$ and the number of differentiated habitats) and by the competition parameter ($\sigma_C$). These parameters have been arbitrarily chosen. The ratio $\sigma_C/\sigma_K$ may be smaller in natural populations than in our model, which is expected to lead to a higher diversity. In addition, we assume only three habitat types, whereas more habitats can exist in natural landscapes, generating more allopatric diversity than in our model.

We assumed that the assortative mating is based on the difference between the ecological traits of individuals. This is analogous to a one-allele mechanism (Felsenstein, 1981). Such ecological traits are sometimes called “magic traits” (e.g. Gavrilets, 2004): this hypothesis indeed facilitates the evolution of assortative mating (Dieckmann and Doebeli, 1999; Servedio, 2000). This assumption has long been debated in the literature and seems now accepted as possible (Servedio et al., 2011). Alternatively, sexual selection can drive the evolution of assortative mating by a two-alleles mechanisms. van Doorn et al. (2009) demonstrated that disruptive selection can select sexual preferences for ornaments correlated to local adaptation, inducing this way the evolution of assortative mating. This mechanism is known to have had a great importance for example in the radiation haplochromine cichlid fishes in the East African Great Lakes (Seehausen et al., 1997; Galis and Metz, 1998): male body color is correlated to local adaptation, and females choose their partner according to their color. In brief, assortative mating is likely to evolve under disruptive selection, in some way or other.

4.2 Landscape dynamics and radiations in empirical data

The East African Great Lakes constitute a good example of a dynamic landscape. Water level has fluctuated in each lake, most recently during the Pleistocene glaciation (Owen et al., 1990). As a result, satellite lakes have been repeatedly created and destroyed, intralacustrine islands and new areas of shoreline temporarily appeared, as well as differentiated habitats along the lakeshore. The radiation of cichlid fishes in these lakes is one of the most impressive, with hundreds of closely related species. As noticed above, sexual selection on body colors may have triggered sympatric speciation. Whether this radiation has been sympatric or allopatric remains however discussed (see e.g. the review in Coyne and Orr, 2004). Young et al. (2009) analyzed the origin of this radiation in Lake Malawi using a matrix of pair-wise interaction coefficients for native and transplanted mbuna cichlid species. They suggested that water level fluctuations has divided and reconnected entire communities, this way increasing total diversity by allopatric divergence and local diversity by secondary contact (this hypothesis was first introduced by Greenwood (1965)). Our results show that this mechanism, involving both allopatric and sympatric stages, can indeed generate a radiation under many landscape dynamics time scales. Barson et al. (2007) showed that the body color of cichlid fishes of lake Malawi may be determined by four to seven loci. Under such conditions, diversification in sympathy by evolutionary branching may be possible, but not very frequent: landscape dynamics could thus have significantly contributed to this radiation.

We have focused on landscape dynamics of the Great African Lakes, but similar dynamics in other regions are thought to have generated radiations. For example, Cook (2008) analyzed Madeiran land snail diversity in relation to landscape dynamics. The topology of the Madeiran archipelago has changed due to repeated volcanic events, sea-level changes caused by climatic fluctuations and strong erosion. She demonstrated that the high diversity observed in these islands results from the generation of many opportunities for allopatric divergence on one hand, and interactions between species at secondary contact on the other hand. She suggested that biodiversity can be explained thanks to Hubbell’s neutral model (Hubbell, 2001) if a “rate of geodetic change” is added to take into account for landscape changes. In accordance with our results, she asserted that slow landscape dynamics would generate only limited diversity, and that fast landscape dynamics are likely to induce a catastrophic decrease of diversity.

Acknowledgments

We are very grateful to Martine E. Maan, Etienne Bezault, Ole Seehausen and Carlos J. Melian for insightful discussion about the Great African Lakes and the cichlid fishes. We thank Johan A. J. (Hans) Metz for helpful discussion about the estimation of diversity in our model. R. A. was supported by a
PhD fellowship from the French Ministère de la Recherche et de la Technologie and by a postdoctoral fellowship from the EVORANGE project (ANR-09-PEXT-01102) from the French Agence Nationale de la Recherche. Traveling was funded by the project Modèles Aléatoires de l’Évolution du Vivant (MAEV) from the French Agence Nationale de la Recherche (ANR-06-BLAN-3_146282) for R. A. and A. L., and by the EVORANGE project for R. A.

References


Figure 1. Types of landscape dynamics and time scales. Panel (a): proportion of the time during which the landscape is divided into a given number of geographically isolated regions, indicated on the x-axis. The landscape consists of $n_s + 1 = 6$ sites. The number next to each line indicates the type of dynamics, as defined in section 2.3.1 “Types of landscape dynamics”. Panel (b): mean duration (in generations, on a logarithmic scale) of time intervals during which the landscape consists of a given number of geographically isolated regions. For cases 1 and 2, the landscape never consists of only 1 or 2 geographically isolated regions.

Figures
Figure 2. Panel (a): time series, averaged over simulation replications, of the mean mating trait of all individuals in a fragmented, static landscape. Panel (b): time series, averaged over simulation replications, of diversity in the same landscape. Solid line: \( \gamma \) diversity. Dotted line: \( \alpha \) diversity. Panels (c) and (d) are replications of, respectively, panels (a) and (b), for a static landscape where all sites are merged together. For such landscape, there is only one local population; \( \alpha \) and \( \gamma \) diversities indicate the same quantity. Parameter values: see Table 1. The equilibrium population size is about 400 individuals per site in a fragmented landscape, and about 2000 individuals in a landscape with all sites merged. When sympatric evolutionary branching occurs, local population size increases by about 60%.
Figure 3. Panels (a), (b) and (c) show the time series averaged over simulation replications of, respectively, γ diversity, α diversity and the population mean mating trait. Panel (d) plots time series of one generic simulation replication. The number next to each line indicates the type of dynamics, as defined in section 2.3.1 “Types of landscape dynamics”. Parameter values: see Table 1. The total population size varies from about 2000 to 8000 individuals, depending of the number of species. Table 2 quantifies some characteristics of γ diversity shown in panel (a).
Figure 4. Some typical outcomes of secondary contact and fragmentation. The mechanism illustrated on each panel is described in the text. Each graph corresponds to a geographically isolated region, here depicted by a dot. An individual in the ecological space (x,y) indicates the ecological optimum in a habitat (x-axis, y-axis). The tick on the x-axis (resp. y-axis) indicates the ecological optimum of a habitat P (resp. S). The type of habitat in isolated regions is indicated on top of each panel: PPR indicates the fusion of one site with a habitat P, PR indicates the fusion of two sites with a habitat P, and RR indicates the fusion of two sites with a habitat R. On each panel, the first line shows the state of the population one generation before fusion of the two isolated regions depicted. Second line: a few generations after fusion (number of generation indicated on the left of each graph). Third line: long after fusion, corresponding to one generation before fragmentation of the landscape (fragmentation results in the same landscape structure as depicted on the first line). Fourth line: long after fragmentation. The mean mating trait $\bar{a}$ of the population in each isolated region is indicated on each graph.

Parameter values: see Table 1. The total population size is about: (a) 1200, (b) 2000, (c) 1800, and (d) 2600 individuals.
## Tables

### Table 1. Notation and numerical values

<table>
<thead>
<tr>
<th>Evolving trait</th>
<th>Definition</th>
<th>Values explored; Value used in Results section</th>
</tr>
</thead>
<tbody>
<tr>
<td>$z_i = (x_i, y_i)$</td>
<td>Individual $i$ vector of ecological traits $x_i, y_i$</td>
<td></td>
</tr>
<tr>
<td>$a_i$</td>
<td>Individual $i$ mating trait</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Definition</th>
<th>Values explored; Value used in Results section</th>
</tr>
</thead>
<tbody>
<tr>
<td>$r$</td>
<td>Per capita birth rate</td>
<td>$r = 1$</td>
</tr>
<tr>
<td>$K^*$</td>
<td>Maximum of the carrying capacity function</td>
<td>$10 \leq K^* \leq 5000$; $K^* = 400$</td>
</tr>
<tr>
<td>$\sigma_K$</td>
<td>Standard deviation of the carrying capacity function</td>
<td>$0.1 \leq \sigma_K \leq 2$; $\sigma_K = 1$</td>
</tr>
<tr>
<td>$z^*J = (x^*J, y^*J)$</td>
<td>Ecological optimum in a habitat $J$</td>
<td>$z^*P = (0, 0), z^*R = (1, 0), z^*S = (0, 1)$</td>
</tr>
<tr>
<td>$\sigma_C$</td>
<td>Standard deviation of the competition kernel</td>
<td>$0.0001 \sigma_K \leq \sigma_C \leq \sigma_K; \sigma_C = 0.5$</td>
</tr>
<tr>
<td>$L_k$</td>
<td>Number of diploid loci determining trait</td>
<td>$1 \leq L_k \leq 50$; $L_k = 16$</td>
</tr>
<tr>
<td>$\mu_k$</td>
<td>Mutation rate at each locus of trait</td>
<td>$10^{-8} \leq \mu_k \leq 10^{-2}$; $\mu_k = 10^{-3}$</td>
</tr>
<tr>
<td>$s_k$</td>
<td>Expected phenotypic variance of trait</td>
<td>$10^{-3} \leq s_k \leq 0.5$; $s_k = s_a = 0.04, s_a = 0.1$</td>
</tr>
<tr>
<td>$n_s$</td>
<td>Number of satellite sites</td>
<td>$2 \leq n_s \leq 10$; $n_s = 5$</td>
</tr>
<tr>
<td>$f$</td>
<td>Rate at which a disappeared border appears again</td>
<td>$10^{-6} \leq f \leq 10^{-1}$; $10^{-3} \leq f \leq 10^{-2}$</td>
</tr>
<tr>
<td>$c$</td>
<td>Rate at which an existing border disappears</td>
<td>$10^{-6} \leq c \leq 10^{-1}$; $10^{-5} \leq c \leq 10^{-3}$</td>
</tr>
</tbody>
</table>

### Table 2. Characteristics of $\gamma$ diversity in a dynamic landscape

<table>
<thead>
<tr>
<th>Case of landscape dynamics</th>
<th>Asymptotic $\gamma$ diversity</th>
<th>Duration of the transient phase</th>
<th>Variance over time of the asymptotic $\gamma$ diversity</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>$12.88 \pm 2.22$</td>
<td>$30.13 \pm 9.17$</td>
<td>$5.71 \pm 3.49$</td>
</tr>
<tr>
<td>2</td>
<td>-</td>
<td>$&gt; 100$</td>
<td>-</td>
</tr>
<tr>
<td>3</td>
<td>$5.99 \pm 0.99$</td>
<td>$13.91 \pm 8.84$</td>
<td>$0.46 \pm 0.42$</td>
</tr>
<tr>
<td>4</td>
<td>$10.59 \pm 1.66$</td>
<td>$28.87 \pm 10.97$</td>
<td>$4.01 \pm 2.43$</td>
</tr>
<tr>
<td>5</td>
<td>$2.34 \pm 0.81$</td>
<td>$32.58 \pm 23.69$</td>
<td>$0.03 \pm 0.05$</td>
</tr>
</tbody>
</table>

The cases of landscape dynamics are those defined in section 2.3.1 “Types of landscape dynamics”. The characteristics of $\gamma$ diversity indicated in this table are defined in section 2.3.3 “Measures used in Results section”. The symbol $\pm$ is followed by the standard deviation among simulation replications of each estimation. The duration of the transient phase of diversity increase is indicated in thousand of generations. Under landscape dynamics of type 2, the asymptotic $\gamma$ diversity is not yet reached when simulations end, i.e. after 100,000 generations (Figure 3a); the asymptotic $\gamma$ diversity and its variance are thus not computed.
Table 3. Allopatric divergence and smallest ecological distance between species in a dynamic landscape

<table>
<thead>
<tr>
<th>Case of landscape dynamics</th>
<th>Allopatric divergence (maximum: 0.85 ± 0.10)</th>
<th>Smallest ecological distance between all species</th>
<th>Smallest ecological distance between sympatric species</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.67 ± 0.13</td>
<td>0.16 ± 0.04</td>
<td>0.68 ± 0.03</td>
</tr>
<tr>
<td>2</td>
<td>0.77 ± 0.14</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3</td>
<td>0.52 ± 0.12</td>
<td>0.81 ± 0.07</td>
<td>0.88 ± 0.04</td>
</tr>
<tr>
<td>4</td>
<td>0.62 ± 0.12</td>
<td>0.31 ± 0.09</td>
<td>0.74 ± 0.03</td>
</tr>
<tr>
<td>5</td>
<td>0.25 ± 0.07</td>
<td>0.86 ± 0.27</td>
<td>0.98 ± 0.10</td>
</tr>
</tbody>
</table>

The cases of landscape dynamics are those defined in section 2.3.1 “Types of landscape dynamics”. The measures computed in this table are defined in section 2.3.3 “Measures used in Results section”. The symbol ± is followed by the standard deviation among simulation replications of each estimation. The values of allopatric divergence are to compare to 0.85±0.10 which is the allopatric divergence measured in a fragmented, static landscape (computed from simulations of Figure 2), i.e. the maximum of allopatric divergence under the assumptions of our model. The values of smallest ecological distance between all species (i.e. from all sites combined) are to compare to the smallest ecological distance between sympatric species of the same landscape dynamics type, because the latter depends on the level of assortative mating (which differs between the landscape dynamics types). The smallest ecological distance is computed after the transient phase ends; under landscape dynamics of type 2, the asymptotic level of γ diversity is not yet reached at the end of simulations (Figure 3a), so that no smallest ecological distance is not computed.

Appendix

A Simulations

Our stochastic model is simulated using a birth and death process in continuous time according to Gillespie (1977)’s algorithm. At any time, four events can happen: the birth of an individual (occurring at rate $N_r$ where $N$ is the total population size), the death of an individual (rate $\sum_{i=1}^{N} d(z_i)$ where $d(z_i)$ is defined by Eq 1), the disappearance of a border (rate $cN_a$ where $N_a$ is the number of existing borders), and the appearance of a border (rate $fN_d$ where $N_d$ is the number of disappeared borders). We pick the time until the next event from an exponential distribution with mean equal $1/(N_r + \sum_{i=1}^{N} d(z_i) + cN_a + fN_d)$. The occurring event is then randomly chosen proportionally to the rate of each possible event.

Initial, each border exists with probability $f/(f + c)$, which corresponds to the stationary state of the landscape. The central site is defined as a habitat P, the habitat of each satellite site is randomly chosen at the beginning of each simulation run as a habitat either R or S. Each site is initialized with $K_0$ individuals whose sex is randomly chosen and whose alleles values at each locus determining trait $k$ ($k \in (x, y, a)$) are chosen in a centered normal distribution with standard deviation $\sigma_k$.

We measure time in generations: the generation time is equal to one time unit of the simulation real time divided by the per capita death rate. As the death rate differs between individuals, we approximate it by the per capita birth rate, which is expected to have the same value when the population is at its ecological equilibrium.

The simulation program is coded with the C language using the GNU Scientific Library (Galassi et al., 2009) for random number generation. The computation of diversity is coded with the R language (R Development Core Team, 2010). Source code is under GNU General Public License, and is available upon request to the corresponding author.

B Diversity estimation

Our estimation of diversity is expressed as a number of species, in the sense of the biological species concept (Mayr, 1942). We proceed with the three following steps.

Step (i): definition of clusters of individuals whose ecological traits are close. The phenotypic space $(x, y)$ is divided into cells of area $s_x \times s_y$. We define “well-occupied cells” as cells whose density is higher than the mean density computed over non-empty cells. Contiguous well-occupied cells form a phenotypic cluster (diagonal cells are considered as contiguous). Individuals from other cells belong in the cluster whose phenotypic center is the closest.
Step (ii): evaluation of the level of reproductive isolation between each pair of clusters. For each pair of clusters, we use Eq 3 to compute the mating probability $\pi$ of two individuals randomly sampled from distinct clusters.

Step (iii): computation of the number of distinct species. Two distinct phenotypic clusters are considered as reproductively isolated when $\pi$ is below the threshold $v = 0.01$. A continuous chain of clusters formed by pairs of reproductively not isolated clusters defines a species.

Local ($\alpha$) diversity is computed by applying this method independently in each of the $n_s + 1$ sites, then averaging the $n_s + 1$ values. To evaluate total ($\gamma$) diversity, we hypothesize that all individuals of the metapopulation are in sympathy, and then apply the above method. If such instantaneous secondary contact of all populations occurred, new competitive interactions and/or habitat changes would possibly lead evolutionary processes to change the number of species. Therefore, our estimation of $\gamma$ diversity does not indicate the evolutionary equilibrium, but the instantaneous number of species at such secondary contact. Note that our method imply that two phenotypic clusters in a same site can be considered as two distinct species when computing $\alpha$ diversity, but as the same species when computing $\gamma$ diversity if an intermediate phenotypic cluster exists in another site.

Step (i) of the above method ensures to exclude rare individuals isolated from others individuals in the ecological space from being considered as a full-fledged population. This avoids two issues: considering as a species a single individual unusually far from other individuals in the ecological space, and considering as the same species two phenotypic clusters with a stable and high level of reproductive isolation between them because of a single, exceptional hybrid. Step (iii) uses a threshold $v$ whose value is arbitrarily fixed. Gavrilets (1999) used an analogous threshold to distinguish species. He showed that the number of species is weakly dependent on its value, even when the threshold value varies by several orders of magnitude. We checked that this statement is valid for our simulations: using $v = 0.001$ instead of our default value ($v = 0.01$) in some generic simulations, there were no detectable qualitative change in the results, and very weak quantitative effects (results not shown).

C \hspace{1cm} \textbf{Method used to compute the measures defined in section 2.3.3}

\textbf{“Measures used in Results section”}

Asymptotic $\gamma$ diversity. For each simulation replication, we compute the $\gamma$ diversity averaged over time, ignoring the 50,000 first generations in order to exclude the transient phase of diversity increase. The asymptotic $\gamma$ diversity is then estimated as the average over simulation replications of these $\gamma$ diversities.

Duration the transient phase. For each simulation replicate, we compute the number of generations until $\gamma$ diversity reaches its asymptotic level. The duration the transient phase is then estimated as the average over simulation replications of these numbers of generations.

Variance over time of the asymptotic $\gamma$ diversity. For each simulation replicate, we compute the variance over time of the $\gamma$ diversity, from the time $\gamma$ diversity reaches its asymptotic level to the end of the simulation. The variance over time of the asymptotic $\gamma$ diversity is then estimated as the average over simulation replications of these variances.

Allopatric divergence. For each simulation replication, at each time, we compute the ecological distance between pairs of populations from geographically isolated regions as the distance between the centers of their empirical ecological traits distributions. This ecological distance is computed only between pairs of population from geographically isolated regions with different ecological optima because no allopatric divergence is expected between geographically isolated region with the same ecological optimum. For each simulation replication, at each time, all ecological distances computed are averaged. The allopatric divergence is then estimated as the average over time and over simulation replications of these mean ecological distances.

Smallest ecological distance. For each simulation replicate, at each time after the transient phase, we compute the ecological distance between all pairs of species from all sites of the landscape combined as the distance between the center of their ecological traits distribution. At each time step, we take the minimum of all the computed distances. The smallest ecological distance between species is then estimated as the average over time and over simulation replications of these ecological distances. This measure is compared to the smallest ecological distance between sympatric species, which is computed similarly but considering ecological distances between sympatric species only.
References


A.3 Current and future research (section 4)

1. Phytback research proposal, funded by the ANR.
PHYTBACK – project description

**PHYTBACK** - Ecology-climate feedbacks due to evolution of phytoplankton cell size and shape.

Written by David Claessen

1. **The research proposal (from the ANR proposal) ......................................................... 1**
   1.1. Background, state of the art.................................................................................. 2
   1.2. Objectives............................................................................................................ 4
   1.3. Scientific programme, specific aims of the proposal.............................................. 6
       2.1 Task 2: Generic model development................................................................. 8
       2.2 Task 3: Marine system...................................................................................... 10
       2.3 Task 4: Freshwater system.............................................................................. 13

2. **References.............................................................................................................. 14**

### 1. THE RESEARCH PROPOSAL (FROM THE ANR PROPOSAL)

**General presentation of the problem and context**

Some of the strongest interactions between ecological and climatic processes concern phytoplankton. While the importance of phytoplankton ecology for the global carbon cycle is well established, the role of their evolution is much less so. Adaptation is of particular importance in predicting the system’s response to climate change, since it will modulate the ecological response to environmental change. Recent global ocean circulation models account for phytoplankton ecology. Here we propose to refine the definition of ecological processes and to allow for adaptation of phytoplankton cell size and shape in such models, as well as in more strategic models for freshwater systems. Phytoplankton communities are size-structured, and ecological functioning depends strongly on cell size and shape. Furthermore, phytoplankton size will influence the effectiveness of the biological carbon pump, through which carbon is sequestered from the atmosphere into the ocean interior by cell sinking. In addition, phytoplankton dynamics and evolution depend on interactions with higher trophic levels in the pelagic food web and these ecological interactions are generally also size structured. All these properties are shared between marine and freshwater systems. Phytoplankton ecology will be modelled by accounting for physiological structure (cell size, shape, nutrient quota) of phytoplankton communities and the size structure of the entire food web. We will study a range of models covering spatial scales from the global ocean to lakes. Different theoretical issues will be tackled using models at different spatial and temporal scales. The models will be used to formulate quantitative, testable predictions, that will be put to the test in experimental setups (outdoor freshwater mesocosms) and by using ecological and genomic data from the Tara Oceans expedition. The overall theoretical issue to be addressed is: does adaptation accelerate or mitigate the impact of climate change on the global carbon cycle?
1.1. BACKGROUND, STATE OF THE ART

Global climate-phytoplankton feedbacks in both marine and freshwater ecosystem are crucial for understanding and predicting climate change and its consequences. -- Some of the strongest interactions between ecological and climatic processes concern the role of marine and freshwater phytoplankton in the carbon cycle (Falkowski et al 1998, Arrigo et al 1999; Le Quéré et al 2005, Meskhidze and Nenes 2007; Cermen et al 2008). Phytoplankton produce almost half of the Earth's primary production and are a significant factor in the regulation of global atmospheric CO₂ (Falkowski et al 1998, Cole et al 2007, Smetacek and Cloern 2008). A major influence of ocean phytoplankton on climate is the “biological pump” (Falkowski and Oliver 2007): phytoplankton populations fix CO₂ through photosynthesis, causing a drawdown of atmospheric CO₂, and part of the primary production is exported to the ocean interior through the sinking of phytoplankton cells and faecal pellets from zooplankton grazers. In this way, a flux of carbon from the atmosphere to the ocean interior is established, and the carbon is sequestered for very long periods in ocean sediments. The efficiency of the carbon pump depends on abiotic and biotic factors, including the nutricline depth, zooplankton grazing, taxonomic composition, and cell size and shape (Falkowski and Oliver 2007, Cermen et al 2008). Cell size is important for the carbon cycle since larger cells sink faster than smaller ones, whereas cell shape is important because shape alters the interaction of a cell with its surrounding environment.

Recent carbon cycle models account for phytoplankton ecology -- Such models usually incorporate a small number of phytoplankton functional groups, characterised by physiological parameters for nutrient uptake, nutrient requirements, sinking rate and maximum population growth rate (Le Quéré et al 2005). The approach has been significantly improved by groundbreaking methods developed by Mick Follows at MIT, who seeded a marine ecosystem model of the global ocean with 78 different phytoplankton types whose physiological traits were assigned stochastically from ranges derived from field and laboratory observations. The model was successfully able to generate an emergent community structure and biogeography consistent with observed global phytoplankton distributions (Follows et al 2007). This flexible representation of community structure can therefore be used to explore relations between ecosystems, biogeochemical cycles, and climate change, and can also incorporate the growing body of genomic and metagenomic data mapping the oceans in terms of genes and their encoded physiological functionality.

Yet current models are inaccurate because they do not account for adaptation of phytoplankton traits. -- What is lacking in such models, however, is the ability of phytoplankton species to adapt to current and changing environmental conditions. The latter is of particular importance in predicting the system’s response to climate change, since gradual adaptation of species will modulate the ecological response to environmental change. For example the Follows et al (2007) model of ocean circulation and phytoplankton ecology can simulate the response of the ocean ecosystem to climate change but it does not account for gradual evolution of the present phytoplankton types to changing conditions. The predicted response of the ecosystem will hence be biased by the strong constraint of the absence of adaptation. Yet recent theoretical work on simple, strategic models of phytoplankton adaptation shows that cell size, being an important trait due to allometric scaling of many physiological parameters, is likely to evolve in response to changes in conditions such as nutrient levels (Litchman et al 2009, Verdy et al 2009) or grazing (Jiang et al 2005). Since the ability of phytoplankton to sequester carbon (through sinking) depends on
cell size and shape, evolution of these traits in response to climate change will influence the feedback between climate and the pelagic ecosystem. Given the recently discovered widespread infra-specific genetic diversity in marine phytoplankton (Simon et al. 2009), and the scope for epigenetic adaptation in diatoms and other phytoplankton (Bowler et al. 2010, Jablonka and Gaz 2009), adaptation on time scales from years to centuries seems likely, and may significantly influence the climate-ecosystem’s response to human impact.

Here we propose to incorporate the effect of adaptation of phytoplankton (cell size and shape) in models of feedbacks between climate and ecosystems

Evolution of phytoplankton traits: why focus on cell size and shape? -- Cell size and shape are known to influence the sinking rate of phytoplankton (Litchman and Klausmeier 2008). In turn, the sinking rate is known to be critical for the efficacy of the biological carbon pump as well as for the stability of the vertical stratification of plankton and nutrients, and for phytoplankton species composition (Huisman et al. 2006). Also, cell size and shape strongly determine ecological functioning of phytoplankton in terms of resource acquisition, photosynthesis, maximum growth rate, etc (Litchman and Klausmeier 2008). Grazing by zooplankton depends on the cell size of phytoplankton as well as the body size of zooplankton (Reynolds 2006). Smaller cells are predicted to be more efficient in nutrient uptake than big ones, a conclusion that has motivated many authors to find explanations for why one does find large celled phytoplankton (Chisholm 1992): predator-prey coevolution (Jiang et al. 2005), the use of non-limiting substrates (Thingstad et al. 2005), the size-scaling of diffusive transport (Yoshiyama and Klausmeier 2008), nitrogen limitation and pulsed supply of this nutrient (Litchman et al. 2009), and the size scaling of the nutrient uptake kinetics vs metabolism (Verdy et al. 2009). Cell shape influences the sinking rate but also the relation between cell surface and cell volume. The optimal cell size (for given environmental conditions) is therefore different for spherical and elongated shapes. The evolution of cell shape has not, however, been taken into account in theoretical studies.

Phytoplankton cell size and the role of size-dependent food web interactions -- Taking a cell-size perspective on phytoplankton ecology and evolution raises the issue of size-dependent ecological interactions. Phytoplankton communities are strongly structured by cell volume (Cermano et al. 2006, Irwin et al. 2006). Also, food web interactions depend strongly on organism size (Reynolds 2006). Whereas the role of cell size for allometry is well understood (Litchman et al. 2007), the implications of intra-population cell size variability for population and food web dynamics are still largely unexplored. A theoretical framework capable of rigourously modelling the consequences of intra-population size structure, in a food web context, is the theory of physiologically structured population models (PSP models) (Metz and Dieckmann 1986). Intra-population size variability is well known to have important implications for population and community dynamics (Claessen et al. 2000, 2002, De Roos and Persson 2001, Jansson et al. 2007).

Epigenetic inheritance -- Epigenetic inheritance is likely to be important in the co-evolutionary dynamics of phytoplankton (Bowler et al. 2010). While evidence for its occurrence in phytoplankton is still sporadic (Jablonska and Gaz 2009), epigenetics may explain part of the observed plastic response of phytoplankton to environmental change in both marine and freshwater systems (Bowler et al. 2010). Epigenetic inheritance has been suggested to be an efficient mechanism of adaptation on an intermediate time scale (say one to hundreds of generations), in particular in environmentally fluctuating conditions. The essential aspects of epigenetic inheritance, compared to genetic inheritance, are: rapid adaptation; on/off switching of genes (analogous to large mutation steps); reversibility; and
direct and heritable influence of environment on the trait (Bossdorf et al 2008, Jablonka and Gaz 2009). Epigenetics has been ignored in the adaptive dynamics literature and, more generally, the population-level consequence of epigenetic variation remains largely unexplored (Richards 2008).

**Marine and freshwater systems** -- The recent theory of trait-based community ecology of phytoplankton (Litchman and Klausmeier 2008) shifts the attention from a taxonomic to a functional vision, and provides a unified view of phytoplankton and their embedding food webs. In both marine and freshwater systems, cell size and shape, and size-dependent grazing by zooplankton are of fundamental importance. Here, we propose to adopt such a unified perspective and study marine and freshwater systems in parallel, with the promise of cross-fertilisation. In particular, the manageable spatial scale of freshwater systems allows us to do things experimentally that can be of great conceptual use in marine systems. In this vein, the aim is to develop analogous models for the dynamics and evolution of marine and freshwater systems, but also to develop new experimental knowledge linking size-dependent food web interactions and phytoplankton adaptation. Results from these empirical methods will feed the modelling tasks of both freshwater and marine systems.

In order to be able to predict the phytoplankton’s adaptive response, it is necessary to link cell physiology to fitness, to population dynamics, to food web interactions. The trait-based community approach serves as a useful, simplifying conceptual framework, and proposes cell size as a major trait that influences cell characteristics, in conjunction with cell shape. Yet the consequences of intra-population variation in cell size and intracellular nutrient contents for population dynamics, important for the assessment of fitness and biogeochemical cycling, are yet poorly understood. Two important and recent ecological theories seem very relevant and promising to further the conceptual study of these issues: on the one hand the theory of physiologically-structured population (PSP) models, which is well suited to tackle the intra-population structure issue, as well as size-structured food web interactions; on the other hand the theory of adaptive dynamics which is well suited to link ecological mechanisms and interactions to fitness and short-term evolutionary dynamics (Geritz et al 1998).

### 1.2. Objectives

The above state-of-the-art leads us to propose the following objectives of our research proposal:

*Scientific objectives*

1. To develop a generic model for phytoplankton populations structured by cell size, shape and nutrient-contents, using the framework of physiologically structured population (PSP) models;
2. To study these models in the framework of adaptive dynamics, using cell size and shape as evolutionary traits, incorporating both genetic and epigenetic inheritance;
3. To apply this approach to an existing model of global ocean circulation and ecology (Follows et al 2007) to predict the consequences of phytoplankton adaptation for ecology-climate feedback;
4. To compare the emergent ecotypes to *in situ* measures of marine phytoplankton size and shape, and pinpoint the regions where genomic analysis may be crucial to coupled biogeochemical models;
PHYTBACk – project description

5. To apply this approach to an experimental freshwater system to explicitly test the influence of size-dependent food web structure, and the role of allochthonous energy and carbon subsidy, on phytoplankton adaptation and ecology-climate feedback.

Scientific obstacles and challenges
The project faces three main challenges:

1. How to define fitness, and to simulate evolution, in complex structured and non-stationary populations? This is a fundamental issue in adaptive dynamics theory in recent years (Metz et al 1992, Ferrière and Gatto 1995, Metz and Gyllenberg 2001, Durinx et al 2008). The proposed context will require the development of new theory and algorithms to define and simulate fitness in global circulation models.

2. Experimental assessment of phytoplankton size distributions, adaptive responses, and size-dependent food web interactions (grazing and competition). These challenges will be tackled by using an advanced cell-counting and cell-sizing device for sampling the phytoplankton community, in addition to intense sampling of the size structured food web.

3. Our mechanistic understanding of the role of epigenetic vs genetic adaptation in phytoplankton is currently insufficient. Here we propose to complement our theoretical work program with an empirical approach (experimental evolution) that will supply crucial information on the genomic and epigenomic aspects of adaptation in a marine diatom.

Anticipated results
This is a purely scientific project and hence the “final products” are in the form of scientific results: new theory, new data, new insights, new methodology:

1. An ocean model taking into account the adaptive response of phytoplankton to climate change. This is a climate-ecology feedback currently not taken into account. This will enhance our understanding of the consequences and feedbacks involved in climate change.

2. New theory on physiologically structured population models, including spatial structure and applied to the phytoplankton level (previously applied to zooplankton and fish compartments of the ecosystem). In particular accounting for the intra-population size (and shape) variation of phytoplankton -- essential for a good understanding of the evolution of phytoplankton cell size and shape

3. A common approach to marine and freshwater systems, based on the “trait-based community ecology” perspective, which is expected to cross-fertilize currently separate scientific communities. Also, this will provide us with experimental data (high resolution size-structure analysis of the food web dynamics; functional genomics; food web manipulation) on phytoplankton adaptation and food web interactions, impossible to obtain in a marine context.

4. New adaptive dynamics theory on the consequences and evolution of epigenetics. So far, only genetic inheritance has been studied in this theoretical context.

5. Confrontation of model predictions with experimental data, as well as with a unique data set covering all Earth’s oceans and a wide scope of environmental, ecological and genomic variables (the Tara Oceans expedition).
1.3. SCIENTIFIC PROGRAMME, SPECIFIC AIMS OF THE PROPOSAL

Scientific programme, methodology and project structure

The central question of this research proposal is: What is the role of adaptation (genetic and epigenetic) in the phytoplankton community in the feedbacks between aquatic (marine and terrestrial) ecosystems and climate change? The idea behind the scientific programme is to evaluate, on the one hand, the role of size- and nutrient-structure in phytoplankton populations for their ecological dynamics in the context of their size-structured food web; and on the other hand, to study the consequences of adaptation of cell size and shape for the phytoplankton’s ecological dynamics. Finally, the role of abiotic (climatic) forcing in these two aspects will be evaluated; as well as the feedbacks of the ecological and evolutionary dynamics for climate regulation through biogeochemical cycling.

The project is predominantly theoretical, using mathematical modeling and data analysis to seek answers to this question, yet it is complemented by an important experimental approach. This division between modeling and empirical work is reflected in the composition of the team (Claessen, Ferriere, De Monte, Legendre, d’Ovidio and Follows are modellers; whereas Bowler, Trichine, Zabulon, Edeline and Lacroix are experimentalists) and the requested funding for personnel (postdoc 1: 36 months modeling, postdoc 2: 24 months experiments) (see Fig 2 in section 3.2).

The modeling work uses four main methodologies:

a. The theory of physiologically-structured population (PSP) models (David Claessen, Regis Ferriere, Task 2, 3, 4);

b. The MIT global circulation model (MITgcm), developed at MIT (Mick Follows, David Claessen, Task 3);

c. Adaptive dynamics theory (David Claessen, Regis Ferriere, Stephane Legendre, Silvia De Monte, Tasks 2, 3 and 4). Methods (a) and (b) are basically models of ecology (population dynamics and nutrient cycling). The theory of adaptive dynamics can be used to predict (for any model of population dynamics such as (a) and (b)) the course and endpoint of co-evolution of the ecosystem (including speciation, extinction and species coexistence);

d. The theory of finite-size and finite-time Lyapunov exponents (Francesco d’Ovidio and Silvia De Monte) which will be used for marine data analysis (Tara Oceans expedition and remote sensing, Task 3).

The experimental work uses two main methodologies:

e. Functional genomics to assess genetic and epigenetic adaptation under stress (Chris Bowler, Task 3) in a long-term lab experiment;

f. Automated phytoplankton cell counting and cell-size measuring, in the context of food web manipulation and exhaustive ecosystem and genetic sampling (Eric Edeline, Gerard Lacroix, Task 4)

Details on the methodologies are given in the Task descriptions below.
PHYTBACK – project description

Justification of Tasks in the light of the objectives

The project consists of four Tasks (in parentheses: the responsible scientist):

1. Coordination (David Claessen)
2. Generic model development (David Claessen)
3. Marine system (Chris Bowler)
4. Freshwater system (Eric Edeline)

While each Task has its own sub-set of research questions, they all interact closely with each other (Fig 1). Task 2 seeks to develop general theory about how to model size-structured phytoplankton, how to define fitness and to model evolution of cell size and shape, in a range of models from simple to complex. Fundamental questions on the evolution of cell size and shape will be addressed. Task 3 applies adaptive dynamics theory and methods directly to the MITgcm model (Follows et al 2007). Results from Task 2 are expected to guide the analysis of this highly complex model, as well as to help with the interpretation of the (computationally heavy) simulations of this Task. This Task will give explicit answers to the question of the importance of adaptation for ecology-climate interactions through the carbon pump. The modified MITgcm model will allow us to simulate evolving phytoplankton in the context of climate change. The experimental leg of Task 3 will provide invaluable information on the adaptive response of phytoplankton to changing conditions, both in functional terms (which traits change) as well as underlying molecular biology (observed genetic and epigenetic changes). These experimental results will feed the modeling work. Model predictions will be confronted to in situ and satellite data on ocean phytoplankton. Task 4 uses the freshwater experimental system as a test case: models will be developed to
PHYTBACK – project description

predict phytoplankton evolution and size-structured dynamics, and predictions will be tested in terms of phytoplankton dynamics (abundance and size distributions), food web dynamics, phytoplankton functional genomics, and nutrient cycling.

The integration of Tasks 2, 3 and 4 will hence give a comprehensive view of the role of phytoplankton evolution in marine and freshwater ecosystems, and is expected to shed new light on the posed research question.

2.1 TASK 2: GENERIC MODEL DEVELOPMENT

Responsible: David Claessen (partner 1)

Objectives: to study the implications of intra-population variability between individuals, using the theoretical framework of physiologically structured population models (PSP-models), on both ecological and evolutionary time scales. The idea is to start simple and to study progressively more complex models; at each step asking the questions: (i) What are the consequences of intra-population variability for population and community dynamics? (ii) How can we define fitness in a consistent way, using the theory of adaptive dynamics? (iii) How do cell size and shape evolve assuming either genetic or epigenetic adaptation, respectively? Under which conditions is epigenetic inheritance expected to evolve/persist?

The proposed sequence of increasingly complex models is as follows. Model 1: The well-known Droop model (Grover 1991) will serve as the reference model (Klausmeier et al 2007, Yoshiyama and Klausmeier 2008, Litchman et al 2009, Verdy et al 2009). Model 2: A physiologically structured version of model (1), accounting for two dimensions of population structure: cell size and cell nutrient quota. Model 3: As model (2) but in an explicit water column (1D vertical space). Model 4: As (3) but with forced abiotic conditions, in particular with an annual temperature and nutrient influx cycle, assuming periodic forcing which enables us to use Floquet theory to define invasion fitness (Klausmeier 2008).

In response to question (i) above, models (2) to (4) will be critically examined in the light of the reference model (1) in order to establish which aspects are truly inherent to the physiological structure of the population. Whenever the dynamics of the physiologically structured model are qualitatively similar to the reference model, this lends plausibility to the unstructured Droop model. Whenever important differences are found, this means that the simple reference model may not be reliable to predict biological consequences of oceanographical and human impact on phytoplankton ecology, such as the occurrence of phytoplankton functional types across the globe (Follows et al 2007) or consequences of food web manipulations for phytoplankton adaptation.

Question (ii), computing fitness for complex ecological scenarios is a central, non-trivial objective to this research proposal, with important implications for the study of adaptation in Axes “Marine system” and “Freshwater system”. How to define fitness? The theoretically most elegant fitness definition is “invasion fitness”, the corner stone of adaptive dynamics theory. Invasion fitness is defined as the exponential population growth rate of an infinitely diluted invader (e.g., mutant) population in the environmental conditions set by the current resident population (Metz et al 1992). (Technically, invasion fitness is the dominant Lyapunov exponent). Here “infinitely diluted” means that during the initial phase of invasion (or extinction) of a newly arrived type (albeit through mutation, epigenetics or immigration) its population size is too small to have a significant density-dependent impact. During the invasion process, the mutant population experiences density-dependence from
PHYTBACK – project description

the resident, but does not exert any density dependence on itself nor on the resident. The invader population grows hence exponentially, and the exponential growth rate is defined as its fitness (negative fitness means certain extinction, positive fitness means likely exponential increase). The invasion fitness can hence be derived directly from knowledge of the ecological interactions among individuals, and in particular of the density-dependent interactions. Theoretically, invasion fitness can be determined for any model of population dynamics, irrespective of its details. In the long term, the invasion fitness definition accurately predicts the direction of evolution during the monomorphic, directional phase of evolution (Metz et al 1992). That is, during the phases that can be approximated by a trait-substitution sequence, i.e., a sequence of resident types that each persists until the next mutant invades and replaces it, becoming the next resident itself. It also allows identifying at which points the directional evolution comes to a halt (Geritz et al 1998). At these so-called singular points the directional selection gradient vanishes. The invasion fitness concept allows one to classify the nature of the singular point as either a Continuously Stable Strategy (CSS; a convergent stable fitness maximum) or as an Evolutionary Branching Point (EBP; a convergent stable fitness minimum; Geritz et al 1998).

This fitness concept is consistent and straightforward. For simple models, it is relatively easy to compute and to use in simulation or analytical models. Yet in more complex models, for example in structured metapopulation models (Metz and Gyllenberg 2001), its derivation from the underlying ecological model can be a non-trivial challenge. In particular, in spatially structured populations with externally forced, varying abiotic conditions, it may be a real challenge to derive a coherent definition of invasion fitness. It will be one of the objectives of this project to define fitness for those cases, based on insight gathered from the entire range of studied model complexity. In the most complex cases, the only straightforward solution is to simulate explicitly the “fate” of resident and invader, i.e., to simulate the entire invasion and competition process between invader and resident, but this may be extremely inefficient from a computational point of view. In particular, this could be the case for the most complex of models considered in this proposal: the 3D ocean circulation and phytoplankton ecology model of Follows et al (2007). For this model it is challenging to define invasion fitness other than by explicit simulation. To illustrate the problem, consider following reasoning. At a given time and place in the ocean, the model computes the local rate of increase of a phytoplankton population. Locally, integrated over an annual period, this can be considered to be its invasion fitness. However, the long term rate of increase of the type under consideration depends on immigration from other locations, as well as on non-periodic fluctuations in the abiotic and biotic environment. For example, while its local rate of increase due to reproduction and mortality may be negative, the net rate of change may be positive if the sinking rate from above exceeds the local rate of decrease. Hence the invasion fitness should be evaluated over the entire spatial range of the invader, and over the entire periodicity of fluctuation of its environment. Of course, for aperiodic (or chaotic) fluctuations, this means that the invader must be tracked over a very long period. This example illustrates that blunt simulation of the mutant-resident interaction to assess invasion fitness is computationally extremely costly. For this purpose, defining fitness for complex ecological scenarios is a central objective to this research proposal.

The theory of finite-size and finite-time Lyapunov exponents (Aurel et al 1997) or their more recent developments (De Monte et al 2005, Tailleur and Kurchan 2007) have been successfully used to estimate exponential separation rates in high-dimensional, time-varying dynamical systems. Moreover, they are more and more commonly employed in analyzing
empirical data, in particular remote-sensing measures of the ocean surface (d'Ovidio et al 2004, d'Ovidio and De Monte, submitted). This project envisages their use both for implementing the definition of fitness in complex models (finite-size Lyapunov exponents being particularly important in modelling non-infinitesimal phenotypic changes), and for the comparison of the model expectations with the measures in the marine environment.

The answer to question (iii) above will also have important repercussion Tasks 3 and 4. The results of these strategic, relatively simple models, will serve as conceptual search images for the analysis of the more complex marine and freshwater models. Recently, several results have already been obtained for the evolution of cell size in relatively simple scenarios using the Droop model and an adaptive dynamics approach (Jiang et al 2005, Klausmeier et al 2007, Yoshiyama and Klausmeier 2008, Litchman et al 2009, Verdy et al 2009), but the role of intra-population physiologically structure has not been treated in those studies. Further, the focus on cell shape of this proposal is original. Finally, the explicit distinction between genetic and epigenetic inheritance is completely new in the context of both the modelling of phytoplankton evolution and in adaptive dynamics theory in general.

The distinction between genetic and epigenetic inheritance will be modelled by incorporating both genetic traits and epigenetic traits in the definition of the individual state variables. The inclusion of genetic traits is of course standard procedure in adaptive dynamics theory, but epigenetic traits have not been included before. The epigenetic traits should be modelled in such a way as to capture the essential processes of epigenetic inheritance: rapid adaptation; on/off switching of genes (analogous to large mutation steps as opposed to the common assumption of tiny mutations in adaptive dynamics); reversibility; and the possibility for direct and heritable influence of environment on the trait (Bossdorf et al 2007).

2.2 Task 3: Marine system

Responsible: Chris Bowler (partner 2)

Objective 1: to investigate the evolution of phytoplankton cell size and cell shape in a global model of ocean circulation and ecology and to study the implications for the carbon cycle; Objective 2: long-term lab experiment of (epi)genetic adaptation in a marine diatom; Objective 3: confront model predictions with in situ and satellite data of ocean phytoplankton.

The general question underlying Task 3 is: what is the role of adaptation in the emergence of oceanic phytoplankton communities, their biogeography, and their reaction to climate change? It has recently been shown by Follows et al (2007) that well-known functional types of phytoplankton emerge spontaneously as being the dominant constituents of phytoplankton communities in a simulated global ocean model. The Follows model is seeded with a large number (78) of hypothetical phytoplankton species, both prokaryotes and eukaryotes, of which the physiological parameters are defined stochastically by randomly drawing values from realistic ranges based on observed distributions of the values of these parameters. The model includes a realistic representation of ocean geography, currents and abiotic conditions (including nutrient concentrations and fluxes, temperature, salinity, etc). Together with these parameters, ecological interactions between different phytoplankton species and with their abiotic environment are computed for a 10-year interval. In each of many model runs, the final state of the phytoplankton community consists of the same overall functional groups, and the emergent spatial distribution in these simulations corresponds well to the actual, observed biogeography of these functional types. This flexible representation of community structure can therefore be used to explore relations between ecosystems, biogeochemical
cycles, and climate change. For example, given the prominent role of ocean phytoplankton, and that of diatoms in particular, for the global carbon and other biogeochemical cycles, the model can help in predicting the future role of the ocean in the stocking or release of greenhouse gases and hence in the regulation of climate.

What is lacking in these studies up until now is an adaptive response of phytoplankton to current and changing environmental conditions: although the initial state contains considerable genetic variability (i.e., the 78 initial species), this variability erodes systematically since the genomes are not allowed to evolve in the modeled ocean. This means that their response is constrained by the initial genetic variation. Yet biotic conditions change continuously throughout the 10-year period (which represents many generations), and hence the final conditions may be favorable to types that have gone extinct in an early phase. Furthermore, organisms are likely to adapt/evolve via heritable genetic or epigenetic changes. Indeed, even in the absence of abiotic change, evolutionary theory shows that evolutionary traits may continue to fluctuate and are not at equilibrium (Jiang et al. 2005). This shortcoming is especially limiting for studies of environmental change, such as simulated climate change, because phytoplankton adaptation could have important ecological consequences. For example, adaptation may prevent extinction of species, in which case the evolutionary model may predict higher biodiversity, and hence no loss of the ecological functioning of the marine ecosystem. In other words, we will ask the question: how robust are the predictions of the purely “ecological” model of Follows et al. (2007) when the seeded species are allowed to evolve?

We propose to refine the Follow model by equipping the organisms with the ability to evolve, and thereby adapt, to changing environmental conditions. More specifically we will allow different phytoplankton groups to evolve by one of two mechanisms known to drive heritable adaptation: irreversible genetic point-mutation based processes and reversible more rapid epi-genetic processes. The evolutionary traits we will use are cell size and cell shape because they are well known to influence key processes such as nutrient uptake and sinking rates.

This work will allow us to investigate the role of adaptation and the continuous input of genetic and epigenetic variability on the emergence and dynamics of phytoplankton communities in a realistic, spatially explicit ocean model. In particular we will focus on spatial patterns that may emerge due to the presence of spatial gradients in light intensity and nutrient concentrations. Cell size (evolutionary trait) is known to influence the buoyancy of cells and hence the sinking rate of phytoplankton (Litchman and Klausmeier 2008) and is hence critical for the efficacy of the biological carbon pump as well as for the stability of the vertical stratification of plankton and nutrients, and for phytoplankton species composition (Huisman et al. 2006). Furthermore, the ecological effects of cell shape has received much less attention than that of cell size (Litchman and Klausmeier 2008) even though it also has important ecological consequences, for example, cell shape is known to affect how physiological parameters scale with cell volume. The existing literature on the evolution of cell size does indeed suggest that rich adaptive dynamics can occur (Jiang et al. 2005, Litchman et al. 2009, Verdy et al. 2009).

To this end, an important part of Task 3 will consist of developing a mathematical model of marine phytoplankton dynamics and evolution, coupled to oceanographic dynamics of currents, nutrient upwelling and seasonal changes in abiotic conditions. Recently, signalling the start of this collaborative work, Mick Follows has provided to David Claessen the code for a 1D-configuration of the 3D-model used in his groundbreaking
publication (Follows et al 2007), and Claessen and a Master student are currently exploring initial trials of implementing adaptation. This simplified model represents a vertical column of the ocean and its phytoplankton ecosystem in one particular region, specifically the Norwegian Sea. Although simplified, this 1D-model does include (i) the random definition of the phytoplankton species initially present; (ii) the ecological interactions (competition for multiple nutrients and light); (iii) explicit nutrient dynamics; (iv) vertical gradients of light intensity and nutrient concentrations; (v) vertical movement of phytoplankton (sinking and diffusion) and nutrients (diffusion); all as specified in Follows et al (2007).

The Task will consist of the following steps: (1) Introduction of mutation into the 1D model and analysis of the model in the framework of adaptive dynamics, and assessment of the relative significance of genetic and epigenetic phenomena. (2) Expansion of the phytoplankton model by explicitly modelling nutrient quotas (3) Formulation of a strategic 1D model by reduction of number of nutrients and simplification of spatiotemporal variability of abiotic factors, in order to capture some of the essential dynamics of the full 1D model while allowing a more thorough theoretical analysis. (4) Introduction of adaptation in the full 3D ocean model. (5) Comparison of the model predictions with empirical data obtained from three sources: a study of genome and epigenome evolution and adaptation in a long-term experiment with the model diatom *Phaeodactylum tricornutum*; the Tara Oceans expedition; and biophysical multisatellite analysis.

In the first experiment, we will examine the genetic and epigenetic changes occurring following a long-term adaptation experiment in *P. tricornutum*. Cells will be exposed to an environmentally relevant stress (eg, nitrogen starvation, elevated CO₂, or the allelochemical decadienal (Vardi et al 2006) for at least 1000 generations, as previously reported for *E. coli* and *Chlamydomonas* (Barrick et al 2009; Collins and Bell 2004). Following the treatment, the population will be examined by genome sequencing, DNA methylation analysis, and chromatin immunoprecipitation to determine the genome wide patterns of key histone marks such as H3K4(me)2 and H3K9(me)2. The patterns will be compared with control untreated cells. Such an experiment is feasible because genome and epigenome maps of this diatom are available (Bowler et al 2008; Bowler, unpublished), and the results will reveal the extent of genetic and epigenetic modifications that have occurred during the adaptation period, together with their functional implications. Such data will be of great benefit for improving our models of the consequences of such phenomena in phytoplankton populations.

The second source of empirical information will derive from the Tara Oceans expedition. During its circum-navigation, data will become available on: phytoplankton concentrations in surface waters; detailed microscopic analysis of phytoplankton populations at the species level both at the surface and in the Deep Chlorophyll Maximum; phytoplankton sizes and shapes in different conditions; functional-genyll Maximum; phytoplankton sizes and shapes in different conditions; functional-genomics based quantification of species abundance at the molecular level; gene expression profiles in different oceanic contexts; a suite of biological (including zooplankton), environmental and climatic indicators. The provided data will allow detailed comparisons with model predictions in terms of the relation between biotic and abiotic factors and species distribution. In particular, the model predictions on the role and evolution of epigenetic adaptation will be confronted to the environmental genomics data from the expedition.

The third source is empirical data from a biophysical multisatellite analysis. The multisatellite analysis will be based on a combination of physical and biological data, respectively surface current from altimetry satellites and water-leaving radiance from ocean
color satellites. Once reprocessed with nonlinear algorithms, these two satellite products have been recently shown to provide information on the spatiotemporal structure of planktonic communities at the global scale and on its relation with physical drivers like turbulent dispersion (Alvain et al 2006, De Monte et al 2009, d’Ovidio et al 2009). This information will be complemented with other emerging experimental products, which provide an estimation of particle size distribution from satellite-based optical measurements (Kostadinov et al 2009). This empirical approach is also expected to contribute to the understanding of the competition and dispersal process in the ocean, two fundamental components of community evolution.

2.3 Task 4: Freshwater System

Responsible: Eric Edeline (partner 3)

Objective 1: to determine the effects of size-dependent grazing by zooplankton on size-structured phytoplankton dynamics and evolution;

Objective 2: to confront model predictions to new experimental knowledge on size-dependent phytoplankton-food web interactions, using mesocosm food web manipulations.

Models developed in Task 2 “Generic model development” will be parameterised to an experimental freshwater system. The models will be used to predict the ecological and evolutionary consequences of two experimental treatments: food web configuration (presence/absence of fish); and allochthonous carbon influx (add organic matter/not). The presence of fish will influence zooplankton size structure. Allochthonous organic matter (OM) subsidy is expected to influence the competitive relation between phytoplankton and heterotrophic producers in favour of the latter and to interfere with the trophic cascade from fish to phytoplankton. The experiment proposed in this Task tries to disentangle these two important aspects of lake systems.

The model analysis will yield testable, quantitative predictions that will be confronted to experimental data. The model analysis is also expected to lead to the design of additional experiments. The steps in this Task are: (1) Models from Task 2 will be parameterised for the freshwater system including phytoplankton, zooplankton and fish. A heterotrophic compartment will be added, essential in the dynamics of lake food webs (Jansson et al 2007). (2) The food web dynamics will be quantified in response to the two experimental treatments (modelling). (3) The results will be confronted with experimental results on size-structured, community and food-web dynamics (experimental). (4) The effects of phytoplankton adaptation on nutrient cycling will be investigated theoretically, by comparing the models with and without genetic or epigenetic adaptation, and by assessment of the implications for ecology-climate feedback through the carbon cycle.

This task will constitute a mixed theory-experiment collaboration between modellers of Partner 1 (including postdoc 1), postdoc 2 (Partner 2) and ENS researchers Gerard Lacroix and Eric Edeline (Partner 3). Lacroix and Edeline have recently developed an experimental setup for freshwater ecosystems at the ENS-CNRS experimental station (CEREED-Ecotron Ile de France, www.foljuif.ens.fr). The existing experimental facilities will be used to manipulate the freshwater pelagic food web, using four treatments (no fish, planktivorous fish) x (no allochthonous OM, allochthonous OM), and three replicates of each treatment.

Postdoc 1 will formulate models of size-structured food webs, based on the previous work on PSP models for lake ecosystems that have been used to study the dynamics of either size-structured zooplankton or fish populations, embedded in food chains (De Roos et al 1992, Persson et al 1998, 2003, 2007, Claessen et al 2000, 2002). The novelty here is the taking
into account of physiological structure (size, shape and nutrient quota) in the phytoplankton compartment. In addition to the population-based PSP model, a phytoplankton community-based model will be developed (i.e., a size-spectrum model lumping species; Andersen and Beyer 2006). Models of increasing complexity will be investigated, starting from a single phytoplankton species and single nutrient, to multiple phytoplankton species and nutrients. The work programme of the PSP modelling in this task follows the steps outlined in Task 2.

The tank experiments (postdoc 2 with Eric Edeline and Gérard Lacroix)
The experimental component of this Task will be performed in 12 circular tanks (1.5 m depth, 8 m3, see Fig.2) mimicking small, shallow water ponds. The first experiment will last for 1 year and will include 4 experimental treatments: (No fish, planktivorous fish) x (no allochthonous OM, allochthonous OM) x 3 replicates. Analyses performed on the tanks and variables measured will be: Water physics and chemistry - Primary production, Bacterial production, Coloured dissolved Organic Matter - Chemical nature of allochthonous, dissolved and sedimented organic matter (lipid component analysis by GC- and GC-ms), stable carbon and nitrogen isotope analysis, organic matter degradability (respirometry experiments) - Microbial diversity (PCR-DGGE, clone libraries and T-RFLP...) - Abundance of bacteria, ciliates, flagellates, phyto- and zoo plankton, periphyton, and macroinvertebrates - Size distribution of phytoplankton - Fish (small-sized roach, Rutilus rutilus) survival, somatic growth and fecundity. The results of this first 1-year experiment will provide the necessary information to parameterize the models, or, if further data is needed, to tune relevant protocols for new experiments.

Remark: The time allocation indicated by the members of Partner 3 to the project corresponds only to the additional time necessary for participating to the present project (i.e. the phytoplankton sampling), and not to the total time effectively allocated for preparing and realizing the field experiment. This part of the experiment has already been prepared and accounted for independently.

2. REFERENCES
PHYTBACK – project description


PHYTBACK – project description


Persson, L; De Roos, AM; Claessen, D; Bystrom, P; Lovgren, J; Sjogren, S; Svanback, R; Wahlstrom, E; Westman, E. (2003). Gigantic cannibals driving a whole-lake trophic cascade. Proceedings of the National Academy of Sciences 100 (7): 4035-4039.


A.4 Other work (section 5)


Which traits promote persistence of feral GM crops? Part 1: implications of environmental stochasticity

David Claessen, Christopher A. Gilligan, Peter J. W. Lutman and Frank van den Bosch


Transgenes in plants affect life history traits including seed survival and germination. With stochastic matrix models we predict population-level consequences of transgene induced life history changes. We assess systematically which changes in life history traits, resulting from genetic modification, may increase the risk of invasion and persistence of feral crops or increase fitness in case of introgression from arable fields into conspecific, feral populations. We apply our method to feral populations of oilseed rape. Like many annual weeds, oilseed rape depends critically on disturbance; in undisturbed habitats it is generally outcompeted by perennials. The associated inherent variability and unpredictability render deterministic models inappropriate. With a stochastic matrix model we study population growth rate, elasticities and quasi-extinction times. Our results indicate that changes in survival in the seed bank impact population growth and persistence most. Less important are dormancy, fecundity and seedling survival. The predicted distribution of extinction times is highly skewed, with some patches persisting for decades.

D. Claessen, IBED/Population Biology, Univ. of Amsterdam, Kruislaan 320, NL-1098 SM Amsterdam, the Netherlands, P. J. W. Lutman and F. van den Bosch, Rothamsted Research, Harpenden AL5 2JQ, UK (claessen@science.uva.nl). – D. Claessen and C. A. Gilligan, Dept of Plant Sciences, Univ. of Cambridge, Downing Street, Cambridge, CB2 3EA, UK.

There is much concern on spread of genetically modified (GM) plants and their introduced foreign genes (‘transgenes’) into the environment. Spread of transgenes from crops can originate from hybridisation of transgenic pollen with wild or cultivated relatives, or from transgenic seeds that disperse and potentially establish a persisting feral population of transgenic plants (Wolfenbarger and Phifer 2000). In either case, however, invasion and persistence of a transgene will depend on the effect it has on the fitness of the emerging transgenic feral population, be it the crop or a wild relative (Cummings et al. 2002). The likelihood of invasion and the expected persistence time of a transgenic feral population are crucial in a risk assessment of the environmental impact of a transgene.

One of the concerns about transgenic crops is that a GM crop may be more invasive than the original crop and hence constitute a persistent weed problem (Bullock 1999). In order to assess environmental risks of adopting GM crops it is crucial to link transgene induced life history changes (Table 1) to population dynamics. Field-release experiments such as described by Crawley et al. (1993, 2001) and Hails et al. (1997) are the most reliable way to assess the fate of specific GM plants in cultivated or natural environments. Yet, such experiments cannot be used to predict systematically which changes in life history are more likely to produce persistence than others. A useful tool for this purpose is the analysis of matrix population models (Bullock 1999, Caswell 2001).

Previous studies have used deterministic models to estimate the population growth rate of feral transgenic crops (Crawley et al. 1993, Parker and Kareiva 1996, Bullock 1999). However, both routes of spread of
transgenes (via pollen or seed) are subject to environmental stochasticity. Many crops are annuals derived from weedy ephemeral plants such as oilseed rape and sunflower (Linder and Schmitt 1995), combining a high potential reproductive output with low competitive ability. Their strong dependence on disturbance implies that variability and unpredictability are essential features of populations of such plants, which renders deterministic models inappropriate to describe their dynamics. Here we show how to incorporate environmental stochasticity to allow for disturbance of patches and good and bad years for seed production into a parsimonious model that relates simple life history traits to the population dynamics of transgenic plants from which it is possible to assess the risk of invasion and persistence of these plants. We parameterise the model for oilseed rape (*Brassica napus* L.), which is at the focus of current discussions on GM crops, mimicking two common habitats: (i) a feral population outside arable fields and (ii) a volunteer population inside an arable field (Lutman 1993, Crawley and Brown 1995, Pessel et al. 2001).

Specifically we use the model to ask the following questions. (a) How does disturbance affect the population growth rate and hence the probability of invasion? Is there a minimum rate of disturbance for population increase? (b) Which components of the crop life history have most effect on population growth rate? How is this affected by environmental stochasticity? (c) What is the distribution of extinction times for feral and volunteer patches? How is this affected by changes in life history trait? (d) For feral patches typical of those arising from seed spill from lorries, what is the effect of initial conditions (seed number) on patch persistence and the chance that patches will be extinct after five, ten or 20 years? (e) How robust are our results to parameter uncertainty? The results are discussed in the context of risk assessment and experimental work on the spread of transgenes.

Our intention is to provide a theoretical framework to complement the earlier, deterministic studies of an ecologically important and pressing problem. Our focus on the implications of environmental variability, inherent in the dynamics of ephemeral crop plants, and transient dynamics provides a new and relevant background to evaluate the risks of adopting transgenic crops. The relevance of using a stochastic approach can be deduced from the list of questions above: they can only be studied in a framework that incorporates environmental variability.

### Model and methods

We use a stochastic matrix model to project the state of the population from one year to the next (Caswell 2001). The state of the population in year *t* just before seed shed is represented by the number of flowering adults, *n*1(+), the number of seeds in the top layer of the seed bank, *n*3(+), and the number of seeds in the deep layer of the seed bank, *n*3(−). The top layer of the seed bank is defined as the range of depths from which germination can result in successful establishment; for oilseed rape...
this means the deep layer starts at a depth of approximately 9 cm (Lutman 1993). Our assumptions on the demographic processes within a single patch of an annual plant with a structured seed bank are summarised schematically in Fig. 1. The values of the parameters for oilseed rape in feral and volunteer habitats are listed in Table 1.

We assume that environmental stochasticity affects fecundity, germination and seedling survival; we use subscript t to identify these stochastic processes. First, in most plants fecundity depends on weather factors (Harper 1977) and we incorporate this variability in a simplified way by assuming that the number of seeds per plant, F_t, is high (F_G) in some years (referred to as good years) and low (F_B) in others (bad years). Second, the fraction of seeds that germinate, G_t, and the proportion of seedlings that survive to become flowering plants, S_t, depend on whether the habitat is disturbed (G_D and S_D) or undisturbed (G_U and S_U) in year t. In feral patches a disturbance should be thought of as a temporary reduction of interspecific competition, such as the removal or cutting of perennial grasses by mowing or the activity of rabbits. This is assumed to increase both germination and seedling survival (i.e. GD and S_D are high in disturbed habitats). We assume that disturbances have no effect on plants beyond the seedling stage. In volunteer patches where disturbance occurs by cultivation the success of volunteer oilseed rape depends on (i) weed control and (ii) the current crop, both affecting seedling survival. Common crops in rotation with oilseed rape are cereals and field beans. In cereal fields, volunteer oilseed rape seedlings perish due to competition and the possibility of herbicide application, while in field beans or oilseed rape crops they are likely to survive and set seed. Seedling survival is hence high only if the volunteer patch escapes weed control and the current crop is noncereal. We refer to this combination of factors as a ‘favourable opportunity’ and model it analogously to a disturbance in feral patches (Table 1).

We denote the probability of a disturbance (feral habitat) or favourable opportunity (volunteer habitat) by p and the probability of a good year by q. The two sources of environmental stochasticity are assumed to be independent of each other, such that the environment may be in one of four states at each time step. We assume that there are no temporal correlations in the condition of the environment, i.e. that the stochastic process is independent and identically distributed (IID). We have no good estimate of p, but it is thought to be low in both habitats. We will vary p and study its effect on population dynamics. The probability of good years is assumed to be q = 0.5, but preliminary sensitivity analysis shows that our results are qualitatively robust to variation in q; the value of q affects the dynamics mainly through its effect on mean fecundity.

The three variables n_1(t), n_2(t) and n_3(t) together make up the population vector n(t). The transition of the population from one year to the next, as outlined in Fig. 1, can be summarised as a vector-matrix product (Caswell 2001):

\[ n(t + 1) = X_t \cdot n(t) \]

where \( X_t \) is a stochastic, 3 \times 3 projection matrix. From Fig. 1 we can derive the elements of the matrix \( X_t \):

\[
X_t = \begin{pmatrix}
(1 - \mu) F_t (1 - d) \sigma_1 + \delta d \sigma_2 G_t S_t & \delta d \sigma_2 G_t S_t & \delta d \sigma_2 G_t S_t \\
(1 - \mu) F_t d \delta \sigma_2 (1 - G_t) s & \delta \sigma_2 (1 - G_t) s & \delta \sigma_2 (1 - G_t) s \\
(1 - \mu) F_t d \delta \sigma_3 & \delta \sigma_3 & \delta \sigma_3 \\
\end{pmatrix}
\]

At each time step the values for F_t, G_t and S_t are selected corresponding to the current state of the environment. This approach is equivalent to selecting entire matrices at each time step (Caswell 2001).
The deterministic equivalent model uses the mean matrix U (weighted by p) at each time step:
\[ n(t + 1) = U \cdot n(t), \tag{2} \]
with \( U = \varepsilon(X_t) \) where \( \varepsilon(X_t) \) denotes the expected value of the stochastic matrix \( X_t \).

**Population growth rate and fitness**

In a deterministic matrix model, the population growth rate equals the logarithm of the dominant eigenvalue of the (mean) matrix \( U \), denoted \( \log \lambda_1 \) (Caswell 2001). Note that \( \log \) denotes the natural logarithm throughout this article. In a stochastic environment the most relevant measure of population growth is the mean long-term population growth rate, \( \log \lambda_s \) (Tuljapurkar 1997, Caswell 2001), which can be estimated as

\[ \log \lambda_s = \frac{1}{T} \sum_{t=0}^{T-1} r_t \tag{3} \]

from a time series with any initial condition, where \( r_t \) is the growth factor at time \( t \), defined as \( r_t = \log (N(t + 1)/N(t)) \) and \( N(t) = n_1(t) + n_2(t) + n_3(t) \). We estimated \( \log \lambda_s \) with simulations of \( T = 50,000 \) time steps. In density-independent population models, such as Eq. 1, individual fitness is equivalent to the population growth rate.

**Elasticity analysis**

Elasticity analysis is used to identify components of the life cycle to which the population growth rate is most sensitive (de Kroon et al. 1986). The elasticity of a life history parameter is defined as the proportional increase in the population growth rate in response to a proportional increase in the parameter. It is thus a form of sensitivity. The elasticity to, for example, parameter \( d \) is therefore

\[ e_d = \frac{\partial \log \lambda_s}{\partial \log d} \tag{4} \]

Hence an elasticity of \( e_d = 0.2 \) means that if \( d \) were increased by, say, \( 3\% \), then \( \lambda_s \) would increase by \( 0.2 \times 3\% = 0.6\% \). (Note that this is true only by approximation, as the relation between \( \log \lambda_s \) and the vital rates is generally nonlinear). By calculating the elasticities to all life history parameters, we can identify the ones that, if changed, have the largest effect on the population growth rate. Altering such parameters by GM is likely to have most impact on population dynamics (Bullock 1999). There is no a priori reason to believe that the effect of GM is best described by proportional changes of the vital rates. Yet elasticities can even be used to assess the effect of any experimentally measured change in a vital rate on the absolute change in population growth rate using the relation \( \frac{\delta \lambda}{\delta x} = e_x \frac{\lambda}{x} \), where \( x \) is the vital rate and \( e_x \) denotes its elasticity. We focus on elasticity analysis because it offers more straightforward comparison of the different vital rates than sensitivity analysis.

Contrary to reports by Caswell (2001) and others, elasticities in stochastic models can be very different from elasticities in deterministic models (Tuljapurkar et al. 2003). Below, we therefore present results based on the full, stochastic model, but for comparison we present results of the mean matrix model as well. We estimated elasticities with time series of \( T = 50,000 \) time steps, using the method described by Caswell (2001).

Note that we use the kind of stochastic elasticity which assumes a proportional perturbation of both the mean and standard deviation of a matrix element, and which is denoted by \( E^S \) by (Tuljapurkar et al. 2003). This is equivalent to assuming that the value of a vital rate is perturbed by the same proportion in each possible state of the environment. This kind of stochastic elasticity is the one most commonly used (Caswell 2001).

**Quasi-extinction times**

In our model the number of individuals can become trivially small without ever becoming zero because we do not incorporate demographic stochasticity. However, we can specify a critical abundance, below which we consider the population extinct; this approach is referred to as quasi extinction (Caswell 2001). We consider a local population extinct if the total number of individuals drops below one, \( n_1(t) + n_2(t) + n_3(t) < 1 \). We record the time to extinction, \( \hat{t}_q \), in 20,000 runs with a fixed initial condition of 100 individuals in the shallow seed bank, that is, \( n(0) = (0, 100, 0)^T \), where \( T \) denotes transpose. The smoothness of the estimate of \( \hat{t}_q \) versus \( p \) (Results) suggests that 20,000 runs is sufficient.

From simulations we compute the elasticity of extinction time to the life history parameters as:

\[ e_x = \frac{x}{\hat{t}_q} \frac{\Delta \hat{t}_q}{\Delta x} \tag{5} \]

where \( x \) represents the considered life history parameter, \( \hat{t}_q \) the mean extinction time in 10,000 runs, \( \Delta \hat{t}_q \) the difference in mean extinction time obtained from simulations with the values \( x \) and \( x + \Delta x \), respectively.

**Effect of initial conditions**

To study the effect of seed spills, we calculate the short-term prevalence and persistence times for a range of initial conditions. The initial conditions are meant to mimic a lorry seeding event; seeds are deposited on the
surface, some of which will germinate immediately, and some of which will be incorporated into the top layer of the seed bank. Assuming that $\Delta(0)$ seeds are deposited in year $t=0$ in an empty patch ($n(0)=0$), the population vector in year $t=1$ is defined according to Fig. 1; for example, the number of seeds in the shallow seed bank will be $n_s(1) = \Delta(0) \times \delta_{22} \sigma_s(1-G_0)s$. The vector $n(1)$ was used as initial condition in Eq. 1. Results are based on 10,000 runs per initial condition.

Robustness to model assumptions

We test the robustness of model results to parameter uncertainty. In principle the results will depend on the specific values for the life history parameters (Table 1) and it is therefore important to assess the sensitivity of our results to unavoidable uncertainty in these estimates. To systematically test the robustness we created 1,000 different parameter sets by choosing all parameter values randomly from a uniform distribution ranging $\pm 25\%$ around the original estimate for feral habitat (Table 1). For each random parameter set, stochastic elasticities were computed as outlined above.

In a second test of robustness we relaxed the assumption of constant movement in the seed bank. If vertical seed movement is related to the occurrence of disturbance, we expect more seeds to move in disturbed habitat. We checked this by reducing $\delta_{22}$ and $\delta_{33}$ by $80\%$ or $50\%$ in years with a disturbance.

Results

How does disturbance affect population growth rate?

The stochastic growth rate is always lower than (or equal to) the deterministic growth rate for both volunteer and feral populations (Fig. 2), as predicted by general theory (Tuljapurkar 1997). A volunteer population is predicted to increase ($\log \lambda_s>0$) only if the probability of a favourable opportunity ($p$) exceeds $9\%$ (Fig. 2a). The fact that $\log \lambda_s<0$ for small $p$ shows that cultivation can be an effective method to control volunteer oilseed rape. A feral population is predicted to grow if the disturbance rate exceeds $28\%$ (Fig. 2b). For very low $p$ there is no difference between $\log \lambda_s$ and $\log \lambda_1$ which is due to the absence of variability in the deep layer of the seed bank.

In the absence of reliable estimates for the disturbance rate $p$ we can obtain a rough estimate for feral patches based on Crawley and Brown (1995). They recorded oilseed rape density (discretised into eight density classes) in each 100 by 1 m road verge along the M25 motorway (UK) in 1993 and 1994. They note that almost $20\%$ of sites that were empty in 1993 were occupied in 1994. Assuming (i) that establishment requires disturbance and (ii) the presence of a seed bank (i.e. no seed limitation), then $20\%$ of sites must have been disturbed and a first estimate is hence $p \approx 0.2$. A second and independent estimate can be made based on the assumptions that only sites which are occupied in 1993 and which are subject to a disturbance, will produce a higher density in 1994. Using Table 1 in Crawley and Brown (1995), by summing the probabilities of all

![Fig. 2. The effect of changing $p$, the probability of a favourable opportunity (volunteer patch) or a disturbance (feral patch) on the population growth rate. (a): volunteer patch. (b): feral patch. Open symbols: $\log \lambda$, of the mean matrix model (Eq. 2). Filled symbols: $\log \lambda$, of the stochastic matrix model (Eq. 1). Error bars: 95\% confidence interval of $\log \lambda$, estimated with the method of Caswell (2001). (c) and (d): distribution of quasi extinction times; mean (filled symbols) and 95\% range, based on initial condition of $n(0)=(0, 100, 0)^t$, where $t$ denotes transpose. (c): volunteer habitat. (d): feral habitat. Parameters as in Table 1 and $q = 0.5$.](image-url)
transitions from density classes 1–7 in 1993 to a higher density class in 1994, we obtain \( p = 0.11 \). We thus conclude that realistic values of \( p \) are likely to be smaller than the extinction threshold (\( p < 0.28 \)). This result is consistent with the general perception that most feral oilseed rape populations are ephemeral (Crawley and Brown 1995).

**Which components of the life history have most effect on population growth rate?**

The elasticities to the life history parameters in a feral, stochastic habitat (i.e. Eq. 1) are plotted in Fig. 3a for a range of \( p \)-values. The analysis shows that survival in the seed bank is the most important life history aspect, although the emphasis shifts from the deep (\( \sigma_3 \)) to the shallow (\( \sigma_2 \)) layer of the seed bank as \( p \) increases. For very low disturbance rates, \( \sigma_3 \) is practically the only parameter of importance. The increase with \( p \) of the elasticity of \( \sigma_2 \), as well as of dormancy \( (d) \), fecundity \( (F_t) \) and seedling survival \( (S_t) \), coincides with a shift from a declining population to a growing population (Fig. 2). The elasticity of \( \mu \) is negligibly small and not shown in the figure. It should be noted, however, that the importance of seed dispersal cannot be assessed with our model since dispersing seeds are disregarded (below).

If we ignore environmental stochasticity by using the mean matrix model (Eq. 2), we find a very different pattern of elasticity (Fig. 3b). As in the stochastic model, for \( p < 0.05 \), the highest elasticity is found for seed bank survival \( \sigma_3 \). But for \( p > 0.05 \) high elasticity is found for \( \sigma_2 \), dormancy \( d \) and seedling survival \( S_t \) (Fig. 3b). The mean matrix model also identifies germination \( G_t \) as an important parameter. By ignoring stochasticity one thus reaches a different conclusion about which traits are essential for feral oilseed rape dynamics. For a given value of \( p \), comparison of panels a and b in Fig. 3 shows that the parameters which are subject to stochastic variation \( (F_t, S_t, G_t) \) have a much lower elasticity in the stochastic matrix model than in the mean matrix model. This illustrates the more general finding that increasing the variability in a life history parameter reduces its elasticity (Caswell 2001).

In volunteer habitat with \( p = 0.1 \) the stochastic elasticities are, in decreasing order: \( \sigma_3 \) (0.76), \( \sigma_2 \) (0.22), \( d \) (0.2), \( S_t \) (0.12), \( F_t \) (0.11), \( G_t \) (0.06), \( s \) (0.02), \( \sigma_1 \) (0.02) and \( \mu \) (−0.002). The order of elasticities is the same across the whole range of \( p \) and the elasticities do not change much with \( p \) (data not shown). Again, survival in the seed bank is most important, followed by dormancy, seedling survival and fecundity.

![Fig. 3. Elasticities, broken down to life history parameters, depending on \( p \). Parameters are for a feral patch (Table 1) and \( q = 0.5 \). (a) Stochastic matrix model (Eq.1). (b) Mean matrix model (Eq. 2).](image)

**What is the distribution of extinction times for feral and volunteer patches?**

This question is relevant since feral and volunteer patches are predicted to go extinct for low \( p \) (Fig. 2). For the same range of \( p \) values as in Fig. 2a and b the extinction times are plotted against the corresponding value of \( \log \lambda_e \) in Fig. 2c and d. The constant values of the lower limit of the 95% range of the distribution correspond to the ‘worst case scenarios’ in feral and volunteer patches, respectively. That is, they represent the minimum time to extinction, obtained by selecting the worst growing conditions each year. The figure illustrates a number of results. First, the distribution of extinction times is highly skewed; most patches go extinct quickly, but some persist for decades. Second, in the range \(-0.2 < \log \lambda_e < 0\) the mean extinction time is approximately the same for feral and for volunteer populations. These two observations confirm the theoretical prediction that the quasi-extinction time approaches an inverse Gaussian distribution, the mean of which depends on \( \log \lambda_e \), only, not on the specific life history parameters (Caswell 2001).

For feral patches with low disturbance rates (\( p \approx 0.05–0.1 \)) the expected time to extinction is 6–10 years, while 95% of patches will go extinct within 20–40 years (Fig. 2d). This is longer than the estimates that patches
are unlikely to persist for more than two years given by Crawley et al. (2001), but the discrepancy may lie in the working definitions used for extinction. If, for example, we define extinction as two consecutive years without adult plants, that is, \( n_1(t_1) < 0.5 \) and \( n_1(t_1 + 1) < 0.5 \), the predicted mean extinction time is below two years for \( p = 0.1 \). However, for management and risk assessment purposes the presence of transgenic seeds in the seed bank should be taken into account (Pessel et al. 2001) and therefore we prefer to define extinction as presented in Fig. 2.

For volunteer patches it is predicted that it takes at least 10 years before the seed bank drops below the extinction threshold. Even with infrequent favourable opportunities (e.g. \( p = 0.02; \log \lambda_s = -0.26 \)) a volunteer patch may persist for up to several decades (Fig. 2).

We examined the effect of changes in the life history parameters on the distribution of mean extinction time \( t_q \) for a limited set of parameter values (feral patch: \( p = 0.1, p = 0.2 \) and \( p = 0.25 \); volunteer patch: \( p = 0.05 \)). The ranking of life history parameters according to this elasticity was consistently: \( \sigma_3 \) (8.25), \( \sigma_2 \) (1.27), \( d \) (0.82), \( S_1 \) (0.54), \( F_1 \) (0.41), \( G_1 \) (0.18), \( \sigma_1 \) (0.06), \( \mu \) (0.01), where values in parentheses are the elasticity values for \( p = 0.1 \) in feral habitat. With the exception of \( s \), the ranking is identical to that of elasticity of population growth rate (above). We hence found that the elasticity of \( \log \lambda_s \) was a good predictor for the relative magnitude of the elasticity of \( t_q \).

**What is the fate of patches founded by seed spills?**

Although seeds are dispersed by birds or by other natural mechanisms, it is generally thought that the main origin of feral patches of oilseed rape is seed spill from farm machinery and from lorries during transport from farms to seed processing plants (Crawley and Brown 1995). Here we ask the questions: how does initial density of seeds affect the fate of a newly seeded patch? What is the probability that a patch will still be occupied a number of years later? What kind of abundances can we expect with which frequency?

For a fixed disturbance rate (\( p = 0.1 \)) but a range of initial conditions, Fig. 4a shows the probability of reaching 1, 10 or 100 adult plants within 10 years after the seeding event. It shows that if fewer than \( 10^3 \) seeds are spilled, the establishment of a patch of 10 plants is unlikely, although a single plant may be observed. Only spills of at least \( 10^4 \) seeds are likely to result in a patch with more than 100 plants. Another way to characterise short term dynamics is the probability of extinction within a fixed number of years. Figure 4b shows that half of patches seeded by \( 10^3 \) seeds are extinct within five years but nearly all are extinct in 20 years. Spills of more than \( 10^4 \) seeds persist at least 10 years and possibly longer. For a volunteer patch with \( p = 0.03 \) results very similar to Fig. 4a are found, but extinction probability is much lower than in Fig. 4b, as can be expected from Fig. 2c.

**How robust are our results to parameter uncertainty?**

A first indication of the robustness of our results is that despite substantial differences between parameters for volunteer and feral habitat the ranking of life history parameters according to their elasticities are remarkably similar (above).

The results of the systematic test of robustness to parameter uncertainty are given in Fig. 5 and Table 2 for \( p = 0.15 \). The large uncertainty in parameter values results in considerable variation in elasticities, as illustrated by the frequency distribution of elasticities found for the seed bank survival parameters \( \sigma_2 \) and \( \sigma_3 \) (Fig. 5a) and dormancy \( d \) and seedling spring survival \( S \) (Fig. 5b). Table 2 lists the frequency of rankings of the parameters according to their elasticity from highest (rank 1) to lowest (rank 8). Despite large variation in parameter values, the qualitative pattern of elasticity in terms of ranking is remarkably robust, with parameters \( \sigma_3, \sigma_2 \) and \( d \) occupying the first, second and third place.

---

**Fig. 4.** The short term fate of a newly founded feral patch as a function of the initial number of founding seeds, for \( p = 0.1 \) and \( q = 0.5 \). (a) The probability of establishment within 10 years, defined as the fraction of runs in which the number of flowers exceed a threshold value: \( n_1(t) > n_{\text{est}} \), in at least one year, with respectively \( n_{\text{est}} = 1 \) (■), \( n_{\text{est}} = 10^2 \) (×), \( n_{\text{est}} = 10^3 \) (△). (b) The probability of extinction, defined as the fraction of runs in which \( n_1 + n_2 + n_3 < 1 \) within \( T \) years, for respectively \( T = 5 \) (■), \( T = 10 \) (×), \( T = 20 \) (△).
in more than 90% of the random parameter sets. Hence the general pattern of elasticity is robust against simultaneous uncertainty in all parameters.

This robust ranking may come at some surprise when considering the observed variation in elasticity in some of the parameters (Fig. 5a, b). The consistency in ranking is explained by strong correlations between elasticities, as illustrated in Fig. 5c for d and S. It appears that the elasticities of all parameters correlate strongly ($r > 0.84$) with that of $\sigma_2$, except that of $\sigma_1$ which has a correlation coefficient of $-1$ (Fig. 5c). Thus, a random parameter set which leads to a high elasticity in, for example, d is associated with a high elasticity of $\sigma_2$ as well. These correlations maintain the ranking of the parameters in spite of variation. Strong positive correlations between all parameters except $\sigma_1$ confirm the antagonistic relation between this parameter and the rest as observed in Fig. 3.

Relaxing the assumption of constant movement in the seed bank had only a minor, quantitative effect on elasticities but did not change the overall pattern (data not shown).

**Discussion**

We have shown how stochastic matrix population models can be used to predict population level consequences of transgene-induced life history changes. For oilseed rape, seed bank survival is predicted to be the most critical aspect of the life cycle (Fig. 3). The importance of seed bank survival reflects that (i) life history parameters with little or no variability are less affected by the usually negative effect of stochasticity on elasticity; and (ii) local populations of oilseed rape function as sinks: they are expected to go extinct and the time to extinction is mainly determined by persistence in the seed bank. In more favourable habitat (i.e. high disturbance rate p), local seed production contributes significantly to population dynamics. Even in this case, it appears that seed bank survival is the critical life history process although dormancy and, to a lesser extent, fecundity and seedling survival are important aspects as well.
If stochastic variation of life history parameters is independent and identically distributed (IID), then the more variation a parameter has, the lower its elasticity is (Caswell 2001). This explains why \( F_t \), \( S_t \) and \( G_t \) have lower elasticity in the stochastic model than in the mean matrix model. It is surprising, however, that stochasticity so strongly affects the elasticity of dormancy (d), which is assumed to be constant. The explanation is that the effect of stochasticity on life history transitions applies to the entire life history pathway to which the transition belongs (Claessen 2005). For example d is part of the annual pathway via the seed bank (i.e. new seeds that overwinter in the top layer of the seed bank and germinate the following spring). This pathway is subject to large variability which affects all parameters involved. The low elasticity of d in the stochastic case is hence the consequence of the large variability in seed production, germination and seedling survival. This result is the first demonstration of the important role of life history pathways (or `loops', van Groenendael et al., 1994) for elasticities in stochastic matrix models (Claessen 2005).

A transgene that, intentionally or not, improves seed survival is likely to increase persistence of oilseed rape. This means that transgenic lines should be tested for seed survival. It also means that it is advisable to use cultivars with low seed survival as the basis for transgenic lines. Depending on the expected level of disturbance (p) dormancy, fecundity and seedling survival may also contribute significantly to population growth. Yet, the low sensitivity of fecundity found for small p shown by the elasticities in Fig. 3 means that reduced fecundity of a transgenic line is no guarantee of a low fitness, as increased seed survival may outweigh the decreased fecundity.

By considering known effects of transgenes on life history in oilseed rape (Table 1), we conclude that the positive effect of transgenic oil-modifications (in particular high stearate: HS in Table 1) on seed survival and dormancy could slow population decline and thereby increase persistence of transgenic feral and volunteer populations. Bt transgenes increase performance through plant survival and fecundity (Table 1), which are predicted to have less impact on fitness. It will therefore depend to a large extent on the unintentional effects on seed survival and dormancy whether Bt transgenes increase or decrease fitness. To our best knowledge, there is currently no data available on the effect on these traits.

A central question in risk assessment of GM crops is: will a transgenic crop be able to persist as a weedy population? In many studies this question has been approached by comparing the ecological performance or fitness of the GM crop relative to the original crop or wild relatives (Crawley et al. 1993, Linder and Schmitt 1995, Fredshavn and Poulsen 1996, Hails et al. 1997, Snow et al. 1999). However, our results stress that even if the fitness or population growth rate of a GM crop is lower than that of its conventional counterpart, or even negative, it may persist longer than is acceptable (Fig. 2). In undertaking risk assessment it is therefore essential to specify what level of prevalence is acceptable, and during what period of time. With our approach it can then be estimated whether these criteria are likely to be met.

In the case of oilseed rape, a complete risk assessment of the spread of transgenes will require knowledge of the frequency distribution of seed spills (\( \Delta t \)) in Fig. 1). Although there is indirect evidence for seeds spill from lorries (Crawley and Brown 1995), a reliable quantification is not currently available. A reduction of seed spills from lorries may be a necessary measure to limit transgene spread.

To parameterise our model we used estimates drawn from different literature sources (Table 1). Because experimental conditions are not constant between these different sources, it is necessary to check for the sensitivity of our results to parameter uncertainty. Although uncertainty in parameter values implies uncertainty in their elasticities (Fig. 5a, b) correlations between the elasticity of different parameters (Fig. 5c) lead to a consistent ranking of the life history parameters in terms of their elasticity despite such uncertainty. We have thus shown that our results are robust to considerable parameter uncertainty.

Our matrix model relies on two simplifying assumptions: density-independent population growth and spatial homogeneity, which allow us to use the tools for analysis described above. Density-independent population growth may be a reasonable assumption in the context of invasion and extinction since densities are expected to be low during these processes. We need, however, to check for the robustness of this assumption. Spatial structure may be an important ecological aspect for feral crop populations due to their ephemeral character. Elsewhere we report a check of the robustness of our results to relaxing both simplifications by studying a density dependent, spatially-structured population model consisting of a linear array of a large number of sites coupled by migration, thus mimicking a string of roadside populations (Claessen et al. unpubl.). With this extended model, we consistently found the highest elasticity for seed bank survival (\( \sigma_3 \)) followed by dormancy (d), seedling spring survival (\( S_0 \)) and seed production (\( F_1 \)), independently of the size of seed spills. These results show that the ranking of most important vital rates does not depend critically on the two simplifying assumptions of the matrix model.

Although we apply our method to a crop plant, in principle the effect of transgene introgression into a wild species can be studied by our method as well. This requires parameterisation of the model for the wild species of interest, but otherwise the same analyses. The applied method cannot say anything about the
likelihood of introgression, but it can say something about the expected effect of transgenes on the fitness of individuals that harbour the transgene.

In conclusion, our study shows that environmental stochasticity has implications for GM risk analysis, because the stochastic model gives other results than the deterministic model. Stochasticity has a major impact on the contribution of life history traits to population dynamics. By ignoring stochasticity one reaches a different conclusion about which traits are essential for feral oilseed rape dynamics. We have thus shown that for risk analysis of transgenic crops with an ephemeral character it is crucial to take environmental stochasticity into account. In addition, we have demonstrated that by adopting a stochastic approach, one can address issues of particular interest to risk assessment, such as the distribution of persistence times and the probability of invasion. We performed extensive tests of robustness to make sure our claims are not sensitive to parameter uncertainty. These tests demonstrated remarkable robustness, suggesting that seed bank survival and dormancy are the most important contributors to population growth and persistence times also for other crop species with a life history comparable to oilseed rape.

Acknowledgements – Rothamsted Research is supported by the Biotechnology and Biological Sciences Research Council (BBSRC).

References


Subject Editor: Tim Benton
Which traits promote persistence of feral GM crops? Part 2: implications of metapopulation structure

David Claessen, Christopher A. Gilligan and Frank van den Bosch


Transgenes may spread from crops into the environment via the establishment of feral populations, often initiated by seed spill from transport lorries or farm machinery. Locally, such populations are often subject to large environmental variability and usually do not persist longer than a few years. Because secondary feral populations may arise from seed dispersal to adjacent sites, the dynamics of such populations should be studied in a metapopulation context. We study a structured metapopulation model with local dispersal, mimicking a string of roadside subpopulations of a feral crop. Population growth is assumed to be subject to local disturbances, introducing spatially random environmental stochasticity. Our aim is to understand the role of dispersal and environmental variability in the dynamics of such ephemeral populations. We determine the effect of dispersal on the extinction boundary and on the distribution of persistence times, and investigate the influence of spatially correlated disturbances as opposed to spatially random disturbances. We find that, given spatially random disturbances, dispersal slows down the decline of the metapopulation and results in the occurrence of long-lasting local populations which remain more or less static in space. We identify which life history traits, if changed by genetic modification, have the largest impact on the population growth rate and persistence times. For oilseed rape, these are seed bank survival and dormancy. Combining our findings with literature data on transgene-induced life history changes, we predict that persistence is promoted by transgenes for oil-modifications (high stearate or high laurate) and, possibly, for insect resistance (Bt). Transgenic tolerance to glufosinate herbicide is predicted to reduce persistence.

Feral populations of transgenic crops may serve as sources or stepping stones of gene flow of transgenes from crops to wild relatives. Because genetic modification of plants has direct and indirect effects on life history traits such as germination, dormancy, seed survival and seed production (Crawley et al. 1993, 2001, Parker and Kareiva 1996, Hails et al. 1997, Wolfenbarger and Phifer 2000, Snow et al. 2003) it is important to know which of these changes are likely to increase or decrease the persistence of feral populations. This gives insight in whether the feral crop populations are likely to cause problems such as gene flow to wild relatives, becoming persistent weeds in arable fields or becoming dominant plants in wild vegetation with consequences for native plants or herbivorous insects.

A commonly used approach for assessing the risk of spread of feral, transgenic crops is to estimate the population growth rate, often referred to as $\lambda$ or its...
logarithm, \( \log \lambda \), of the plants in (semi) natural habitats (Crawley et al. 1993, 2001, Parker and Kareiva 1996). Positive population growth and persistence of the population are predicted if the condition \( \log \lambda > 0 \) is satisfied, while the population is expected to go extinct if \( \log \lambda < 0 \). Matrix population models are a useful tool to compute \( \log \lambda \) based on empirical data of life history processes (Caswell 2001), as well as the sensitivity or elasticity of the population growth rate to changes in life history parameters (de Kroon et al. 1986). Elasticity analysis has been used to determine which traits, if changed by genetic modification, are likely to have a large impact on the population growth rate (Bullock 1999, Claessen et al. 2005).

For the case of oilseed rape both empirical and modelling studies predict that single populations are likely to become extinct. Crawley et al. (1993, 2001) found that experimentally established populations of oilseed rape do not persist for more than a few years, which is consistent with empirical observations of short-lived roadside populations of the same species (Crawley and Brown 1995, 2004). In a recent study of a stochastic matrix population model we have predicted a negative population growth rate unless the habitat is disturbed very frequently (Claessen et al. 2005), reflecting the poor ability of oilseed rape to compete with existing vegetation (Crawley et al. 2001, Walker et al. 2004). Moreover we showed that allowance for environmental stochasticity changed the relative importance of life history traits for the population growth rate when compared with a deterministic model (Claessen 2005, Claessen et al. 2005).

With the exception of Crawley and Brown (1995, 2004), previous analyses of invasion and persistence of feral crop populations have been constrained by focusing on a single, isolated population. The theory of metapopulation dynamics predicts that by ignoring spatial structure and dispersal one underestimates the potential of a spatially subdivided population to persist (Hanski and Gilpin 1997). The best example is the classical metapopulation model of Levins (1969, 1970), which shows that a collection of local subpopulations, each of which will go extinct with certainty, can persist if the rate of recolonisation of unoccupied sites exceeds the rate of local extinctions. Here we extend our original model (Claessen et al. 2005) to allow for metapopulation dynamics.

Many roadside feral populations of oilseed rape originate from seed spillage from lorries that transport harvested seeds to seed crushing plants (Crawley and Brown 1995, Pessel et al. 2001). A typical scenario we study is where a certain (e.g. transgenic) cultivar is being used for a number of years such that seed spills from lorries result in the emergence of patches of oilseed rape along a network of roads, initialising the metapopulation. When the use of the cultivar is stopped the dynamics of the metapopulation are determined by local dynamics and seed dispersal only. Here we study the effect of dispersal on the population growth rate, on the predicted extinction boundary and on the expected time to extinction of the oilseed rape populations.

Noting that longterm persistence was possible only with a high disturbance rate in our model for an isolated, single population (Claessen et al. 2005), we now ask the following questions. Will sufficient dispersal enable the metapopulation to persist indefinitely? Does the relative importance of life history traits for population growth and persistence time change with metapopulation structure and hence what are the implications of genetic modification? In other words, how robust is the analysis of the stochastic matrix model (Claessen et al. 2005) to the introduction of density dependence and metapopulation structure? Of particular interest is the interaction between environmental variability and dispersal, previously investigated in the simpler context of only two patches by Wiener and Tuljapurkar (1994). They found that dispersal may increase the population growth rate by providing “insurance” against unfavourable conditions. Should we expect this “insurance effect” to be important for feral oilseed rape? In our model the number of sites is very large and dispersal is local, as opposed to the simple two-patch model of Wiener and Tuljapurkar (1994). Does the insurance effect result in the emergence of characteristic spatial patterns and, if so, can they help us detect the presence of this effect?

The model

We extend a stochastic matrix population model for a population in a single site as studied by Claessen et al. (2005) in two directions, to include metapopulation structure and density dependence. Our model incorporates (i) population structure in terms of a seed bank and flowering plants; (ii) dispersal between neighbouring sites; (iii) environmental stochasticity at two spatial scales (local site and whole metapopulation) affecting life history processes. Each local population is characterised in terms of abundance (not merely presence or absence) so our model may be classified as a spatially explicit, structured metapopulation model. We assume that habitat quality is high if the site is disturbed, since this favours the survival of seedlings, and low otherwise, while disturbances are assumed to be random and relatively rare. The chain of sites can hence be seen as a dynamic landscape in which the focus population lives. Yet our model differs from other dynamic landscape models in that we do not consider catastrophic extinctions of sites. For example, in the models of Keymer et al. (2000) and Johst and Drechsler (2003) a local population cannot survive beyond the life span of the habitat. By contrast, in our model a local population
may survive a run of unfavourable years in between disturbances, most likely in the seed bank. Local extinctions may occur if insufficient seed immigration and infrequent disturbances result in local population decline.

Spatial structure
To mimic a string of roadside populations we assume that the metapopulation consists of a chain of k sites, which initially are unoccupied by the focal cultivar. A site is assumed to stretch 100 × 1 m along a road. The model structure is summarised in Fig. 1 and the model parameters in Table 1. The state variables comprise, the number of plants and the number of seeds in the seed bank and are denoted by \(a_i(t)\) and \(b_i(t)\), respectively, where \(i = 1\ldots k\) refers to the location of the site in the metapopulation. Dispersal is possible between neighbouring sites only. In addition, a site may receive seeds from spillage off lorries (Fig. 1b). We connect the first and last sites as if the string were circular, thus avoiding boundary effects. The structure of the metapopulation therefore resembles the geometry of the string of oilseed rape populations along the M25 motorway orbiting London, UK (Crawley and Brown 1995).

The following derived variables are used: the total abundance in site \(i\), defined as \(n_i(t) = a_i(t) + b_i(t)\); and the total fraction of occupied sites \(m(t)\), defined as the fraction of sites with \(n_i(t) > 0\).

**Dynamics of plants and seeds**
We describe the dynamics of \(a_i(t)\) and \(b_i(t)\) with difference equations, projecting the population at the time of seed shed from year \(t\) to year \(t + 1\). The dynamics of \(a_i(t)\) can be deduced from considering the number of seedlings that settle in site \(i\). Seedlings may emerge from locally produced seed, from immigrating seed, or from the seed bank. Total local seed production is given by \(a_i(t)f(a_i(t))(1 - \mu)\), where \(f(a_i(t))\) is the density-dependent seed production per adult plant, discussed below, and \(1 - \mu\) is the fraction of seeds that do not disperse. The total number of seeds that immigrate into patch \(i\), the sum of seed spillage and seed dispersal, is denoted by \(I_i\), and will be specified later. Together, locally produced seeds and immigrating seeds are called new seeds (Fig. 1a), of which a fraction \(d\) will become dormant and enter the seed bank. Of the non-dormant fraction \(1 - d\), a proportion \(\sigma_1\) survives the winter as seedlings. Finally, of the seeds in the seed bank, comprising both the old ones and the newly incorporated ones, a fraction \(\sigma_2 g_{i,t}\) survives the winter and germinates in spring. Taking all these different pathways together, and multiplying with the the seedling survival rate, denoted by \(S_{1,i}\), we arrive at the equation for the number of flowering plants in year \(t + 1\) in site \(i\):

\[
a_i(t + 1) = s_i [a_i(t) f(a_i(t)) (1 - \mu) + I_{i,t}] \\
(1 - d)\sigma_1 + d \sigma_2 g_{i,t} + b_i(t) \sigma_2 g_{i,t} \]

(1)

The number of seeds in the seed bank next year equals the sum of the new seeds that are incorporated into the seed bank and the old seeds that do not germinate (Fig. 1a):

\[
b_i(t + 1) = [(a_i(t) f(a_i(t))(1 - \mu) + I_{i,t})d + b_i(t)] \sigma_3 (1 - g_{i,t}) \sigma_3
\]

(2)

where \(\sigma_3\) is the survival fraction in the seed bank in the summer.

**Density dependence: fecundity**
We assume that fecundity is density dependent owing to intra-specific competition. The number of viable seeds per flowering plant is assumed to follow

\[
f(a_i(t)) = \frac{F_i}{1 + c_F a_i(t)}
\]

(3)

where \(F_i\) is the maximum viable seed production in year \(t\), reached only in the absence of intra-specific competition, and \(c_F\) a coefficient for the intensity of competition. The functional form of Eq. 3 provides a good fit to the data of Mendham et al. (1981) on arable oilseed rape. Viability of seeds incorporates the probability to find suitable habitat (i.e. to stay within the roadside verge) which is assumed to be high since most seeds will land close to the mother plant. Dispersal has no effect on viability since the distinction between dispersing and non-dispersing seeds is only made for the sake of bookkeeping: the total habitat (the roadside
The metapopulation dynamics are hence governed by the quasi-extinction concept (Caswell 2001) and define a continuous variable not discrete individuals. Instead we use population abundance in terms of density, i.e. a continuous (not) trivial threshold $\chi$. Set to $\chi = 0.5$, after which the abundance remains zero until the site is recolonised. Extinction

We do not consider true extinction because we model population abundance in terms of density, i.e. a continuous variable not discrete individuals. Instead we use the quasi-extinction concept (Caswell 2001) and define a site extinct if the total abundance $n_i(t)$ falls below a trivial threshold $\chi$, set to $\chi = 0.5$, after which the abundance remains zero until the site is recolonised. The metapopulation dynamics are hence governed by

Migration: dispersal and seed spillage

Sites receive seeds that disperse from neighbouring sites (Fig. 1b). In addition, sites may receive seeds that are spilled from lorries. The number of seeds spilled into site $i$ in year $t$ is denoted $\phi_{i,t}$ and is assumed to be a stochastic variable. The total migration into site $i$ is thus:

$$I_{i,t} = \phi_{i,t} + \frac{\mu}{2} [a_{i,t}(t)f(a_{i,t}(t)) + a_{i,t+1}(t)f(a_{i,t+1}(t))]$$

Seed spill occurs in a site with probability $P_{\text{spill}}$. If a spillage occurs, the number of seeds that enter the site is assumed to be a random number between zero and a maximum. Thus,

$$\phi_{i,t} = \begin{cases} \Phi z_t & \text{with probability } P_{\text{spill}} \\ 0 & \text{otherwise,} \end{cases}$$

where $\Phi$ is the maximum number of seeds per seed spillage and $z_t$ is a random number between 0 and 1 drawn from a uniform distribution.

Environmental stochasticity: fecundity, germination and seedling survival

We include two types of environmental stochasticity (Claessen et al. 2005); (i) good years and bad years for seed production resulting in variation in $F_G$; and (ii) disturbance of the vegetation cover affecting germination and seedling survival. Good and bad years are common to the whole metapopulation and are assumed to occur with equal probability. The maximum fecundity $F_t$ equals $F_G$ in good years and $F_B$ in bad ones, with $F_G > F_B$.

Disturbances are site specific. Each year, disturbances are assumed to occur in all sites independently with a probability $p$. The effect of spatially correlated disturbances is discussed later. Germination and seedling survival in spring are assumed to depend on disturbance of the existing vegetation cover. A disturbance is assumed to reduce interspecific competition by removing part of the competitively superior vegetation cover (mostly perennial grasses). We assume that both the germination rate $g_{i,t}$ and the fraction of seedlings that can reach the flowering stage, $s_{i,t}$, are high in disturbed habitat and low otherwise,

$$(g_{i,t}, s_{i,t}) = \begin{cases} (g_D, s_D) & \text{if site } i \text{ is disturbed} \\ (g_U, s_U) & \text{otherwise} \end{cases}$$

where $g$ and $s$ are the germination and seedling survival rates, respectively. For simplicity, we assume that $g_D = g_U$, the germination rate is constant, and the fraction of seedlings that can reach the flowering stage is always high in disturbed habitat and low otherwise. The number of time steps $T$ is varied from 100 to 1000, the extinction threshold $\chi$ is set to $\chi = 0.5$, the disturbance probability $p$ is varied from 0.1 to 0.5, and the number of sites $k$ is varied from 100 to 1000.
Eq. 1, 2, for \( i = 1 \ldots k \), in combination with the rule for quasi-extinction, which resets \( a_i(t) = 0 \) and \( b_i(t) = 0 \) whenever the total density in that site falls below \( \chi \).

**Spatially correlated disturbances**

In simulations to study the effect of geographical correlation of disturbances, we assumed that the occurrence of disturbances is locally correlated. We implemented spatial autocorrelation by using a Markov chain with states ‘disturbed’ and ‘undisturbed’ and transition probabilities \( P_1 \) and \( P_2 \) such that if site \( i \) is disturbed, then site \( i + 1 \) is undisturbed with probability \( P_1 \). Conversely, if site \( i \) is undisturbed, then site \( i + 1 \) is disturbed with probability \( P_2 \). The process is characterised by the Markov chain transition matrix

\[
P = \begin{pmatrix} 1 - P_1 & P_2 \\ P_1 & 1 - P_2 \end{pmatrix}
\] (4)

The expected frequencies of disturbed and undisturbed sites resulting from this Markov process equals the normalised right eigenvector \( w \) of \( P \), while the correlation between the state in neighbouring sites, denoted \( r \), equals the subdominant eigenvalue of \( P \) (Tuljapurkar and Orzack 1980, Caswell 2001). We define the transition matrix (Eq. 4) in terms of the required stationary frequency of disturbed sites \( p \) and the spatial autocorrelation \( r \), by choosing \( P_1 = 1 - r - p \) \((1 - r)\) and \( P_2 = p \) \((1 - r)\), taking care that \( P_1 \) and \( P_2 \) remain within \((0, 1)\).

**Parameter values**

Standard values of the parameters are listed in Table 1. References for the parameterisation can be found in Claessen et al. (2005). The value of \( c_F \) is based on experiments in arable fields (Mendham et al. 1981). Our choice of \( c_F = 0.001 \) slightly overestimates the effect of density dependence.

In the absence of reliable estimates for the disturbance rate \( p \) and the dispersal fraction \( \mu \) we can obtain a rough estimate based on Crawley and Brown (1995). They recorded oilseed rape density (discretised into eight density classes) in each 100 by 1 m road verge along the M25 motorway (UK) in 1993 and 1994. They note that almost 20% of sites that were empty in 1993 were occupied in 1994. Assuming (i) that establishment requires disturbance and (ii) the presence of a seed bank (i.e. no seed limitation), then 20% of sites must have been disturbed and a first estimate is hence \( p \approx 0.2 \). A second and independent estimate can be made based on the assumptions that only sites which are occupied in 1993 and which are subject to a disturbance, will produce a higher density in 1994. Using Table 1 in Crawley and Brown (1995), by summing the probabilities of all transitions from density classes 1–7 in 1993 to a higher density class in 1994, we obtain \( p = 0.11 \). Based on these rough estimates, we assume \( p = 0.15 \) in our calculations. Most results have, however, been checked for dependence on \( p \). It appeared that the effect of \( \mu \) on population growth and persistence responds to changes in \( p \) in the same way as found for the stochastic matrix model of a single isolated population studied in Claessen et al. (2005).

Since we have very little quantitative information for estimating the dispersal fraction \( \mu \), we choose to study the population dynamics for a wide range of \( \mu \). The sites in our model are assumed to represent adjacent sites of \( 100 \times 1 \) m stretches of roadside verge. We assume that most seeds remain within a few meters of the mother plant. It hence follows that at most a few percent of all the seeds disperse to either neighbour. In analyses for a fixed dispersal rate we therefore choose \( \mu = 0.01 \) as a default value or \( \mu = 0.05 \) to illustrate the effect of high dispersal.

The habitat is assumed to be a spatially continuous strip, flanked by unsuitable habitat. Because new seeds are distributed up to a few meters from the mother plant, a fraction of seeds is likely to end up in unsuitable habitat. This form of seed mortality is included in the definition of “viability”, and its effect incorporated in the value of \( F_t \) (Eq. 3). We assume the probability to end up in unsuitable habitat to be low \((<0.2)\) since most oilseed rape seeds land close to the mother plant. This loss of seeds is equal for all mother plants: the borders that subdivide the habitat into sites are chosen arbitrarily, and therefore dispersal between sites imposes no additional mortality.

**Methods of analysis**

**Simulation scenario**

The results below were obtained from studying a scenario of using a cultivar for a number of years (10 unless stated otherwise), after which the use is terminated. Each simulation run hence consists of two phases; an initial phase with seed spills and a final phase without seed spills. Seed spillage during the initial phase results in the emergence of local populations in the roadside metapopulation. The resulting state of the metapopulation is random owing to the stochastic nature of seed spills, and mimics the relevant initial condition for practical questions related to the control of potentially unwanted feral crops. After the termination of seed spillage, the metapopulation persists for a number of years. During the final phase, we keep track of population growth rates and persistence times in the metapopulation, as described below. The length of simulation runs is denoted \( T \) and we use values between 200 to \( 10^4 \) to estimate the various measures of metapopulation dynamics. The number of
sites is also varied between \(k = 200\) and \(10^4\). The model was implemented and analysed with the software package MATLAB.

**Characterising metapopulation dynamics**

**Persistence time: \(T_S\) and \(T_L\)**

We use two different measure of persistence: site-level persistence \(T_S\) and the time until last occupancy of a site \((T_L)\). The persistence time at site-level, denoted by \(T_S\), is defined as the time elapsed between colonisation of a site and the first subsequent (quasi-) extinction of the local population in that site. Note that during a single simulation a site can be colonised and go extinct several times. We estimate the expected site-level persistence time \(\varepsilon(T_S)\) as the average of all measured persistence times in a metapopulation of \(k = 1000\) sites in 200 model runs with \(T = 500\).

For each site in the metapopulation we record the last year in which it was occupied, denoted \(T_L\). Unlike \(T_S\), the value of \(T_L\) of a site is influenced by potential recolonisations. The expected time until last occupancy, denoted by \(\varepsilon(T_L)\), we compute as the average of all non-zero \(T_L\) values in the metapopulation in a number of model runs. This is a more global measure of persistence than \(\varepsilon(T_S)\), while still being practically independent of the number of sites \(k\) (unlike the global extinction time of the metapopulation). For an occupied site at the start of a run, \(\varepsilon(T_L)\) is the expected time it takes before the population will not return to that site.

**Fraction of occupied sites: \(m(t)\)**

To characterise the steady state of the metapopulation we focus on the long-term average value of \(m(t)\), the fraction of occupied sites. In the presence of density dependence (i.e. \(c_F > 0\)) the metapopulation is expected to converge either to global extinction \((m(t) = 0)\) or to a steady state distribution with \(0 < m(t) < 1\). To distinguish between extinction and persistence we compute the long-term average of \(m(t)\) denoted \(E(m(t))\), discarding transient dynamics. We estimate \(E(m(t))\) as the average of the last 200 values of \(m(t)\) in 100 model runs with \(T = 1000\) and \(k = 400\).

**Local population growth rate: \(\log \lambda_s\)**

We measure population growth at two levels: the local population growth rate (at site level) and the metapopulation growth rate. The former is defined by analogy with the theory of stochastic matrix models. If we simplify the metapopulation model by ignoring immigration of seeds from either dispersal or seed spillage (i.e. setting \(l_{ix} = 0\)) and density dependence (i.e., \(c_F = 0\)), then the metapopulation reduces to a set of \(k\) independent sites. The dynamics of each population can then be described with a stochastic matrix population model. The link with stochastic matrix models provides us with a well-studied measure of population growth: the stochastic population growth rate \(\log \lambda_s\). Although this quantity was originally defined for simple stochastic matrices, we use it to characterise the local dynamics in the metapopulation.

The long-term, stochastic population growth rate of \(n_i(t)\) is denoted \(\log \lambda_s\) and is computed as

\[
\log \lambda_s = \frac{1}{kT} \left( \frac{1}{T} \sum_{t=1}^{T} \frac{n_i(t+1)}{n_i(t)} - 1 \right)
\]

with \(kT\) sufficiently large (Tuljapurkar 1990, Caswell 2001). We normally use \(kT = 10^4\) and \(T > 200\) which gives an accurate estimate.

In the metapopulation model the local population growth rate \(\log \lambda_s\) (Eq. 5) is the expected growth rate that would normally be observed at any occupied site.

**Metapopulation growth rate: \(\log \lambda_m\)**

In analogy with the classical metapopulation models, which describe the dynamics of the fraction of occupied sites (Levins 1969, 1970), we measure the metapopulation growth rate \(\log \lambda_m\):

\[
\log \lambda_m = \frac{1}{T} \sum_{t=1}^{T} \frac{m(t+1)}{m(t)}
\]

which is the growth rate of the fraction of occupied sites. We estimate \(\log \lambda_m\) from the dynamics during the initial 100 years (i.e. long before the steady state \(m(t)\) has been reached), and averaged over 100 simulations.

**Elasticity of population growth and persistence time**

Elasticity is a popular measure of sensitivity of population-level quantities such as the population growth rate to individual-level parameters (de Kroon et al. 1986). The elasticity of the stochastic population growth rate \(\lambda_s\) to a life history parameter, denoted \(x\), is defined as the proportional change in \(\lambda_s\) in response to a proportional change in \(x\). It is computed as

\[
e_x = \frac{\partial \log \lambda_s}{\partial \log x}
\]

(Caswell 2001). Elasticities of \(\lambda_m\), \(\varepsilon(T_S)\) and \(\varepsilon(T_L)\) are defined analogously.

We calculate the elasticities of \(\lambda_m\), \(\varepsilon(T_S)\) and \(\varepsilon(T_L)\) numerically by determining their relative change in response to a relative change in each parameter separately. For each parameter, we run the model 100 times (i) with the default parameters as in Table 1; (ii) with the focus parameter perturbed by a 10% increase; and (iii) with the focus parameter perturbed by a 10% decrease. If we denote the perturbed focus parameter by \(x'\) and the resulting population growth rate by \(\log \lambda_s\) then an approximation to the elasticity of \(\lambda_s\) to parameter \(x\) is

\[
e_x = \frac{\log \lambda_s - \log \lambda_s'}{\log x - \log x'}
\]
For each parameter, we use the two different perturbations to obtain two estimates of elasticity and use the mean of the two results as our estimate of elasticity for that parameter. The elasticities of \( \lambda_m \), \( \varepsilon(T_S) \) and \( \varepsilon(T_L) \) are calculated analogously to Eq. 8, by replacing \( \lambda_s \) and \( \lambda_s' \) with the default and perturbed values of \( \lambda_m \), \( \varepsilon(T_S) \) or \( \varepsilon(T_L) \). We determine the elasticities for \( m = 0.01 \) with \( c_F = 0.001 \). The effect of density dependence on elasticity is checked by repeating the calculations with \( c_F = 0.01 \).

Results

For an impression of the dynamics of our metapopulation model and the typical patterns that arise, two space–time plots obtained with different parameter settings are given in Fig. 2. Following a 10-year spell of seed spillage, most established local populations go extinct quickly, while a number of sites remain occupied for a considerable time. Persistent sub-populations typically occur in groups, or 'patches', which remain more or less fixed in space, possibly spanning decades or even centuries (e.g. >140 years in Fig. 2b). The figure suggests that such patches persist longer with a high dispersal rate (\( \mu = 0.05 \)) than with a low one (\( \mu = 0.01 \)).

To study the effect of dispersal on persistence more rigorously we plot the cumulative frequency distribution of site-level persistence times (\( T_S \)) for a range of dispersal rates (Fig. 3). The persistence times \( T_S \) are roughly exponentially distributed, except for the initial decline (first 5–10 years) which is steeper than the tail (result not shown). Figure 3 shows that sites tend to persist longer with a high dispersal rate than with a low one, as was illustrated in Fig. 2. Yet the distribution of \( T_S \) for \( \mu = 0.05 \) does not readily suggest the occurrence of very long-lived patches (i.e. groups of persisting sites) such as the ones observed in Fig. 2. The reason \( T_S \) is not a good

Fig. 2. Space–time plots of the metapopulation model with \( k = 200 \) sites, \( T = 150 \) years, \( p = 0.15 \) and two different values of the dispersal rate \( \mu \). Pixel colour represents local seed bank abundance \( b_i(t) \); pixel co-ordinates represent location \( i \) (horizontal) and time \( t \) (vertical). Seed spillage (indicated by black pixels) lasts for 10 years with \( P_{\text{spill}} = 0.05 \). Horizontal grid lines mark 50 year intervals, starting from the end of seed spillage. Vertical grid lines are 50 sites apart. (a) Dispersal rate \( \mu = 0.01 \). (b) \( \mu = 0.05 \).

Fig. 3. (a) Cumulative frequency distributions of persistence time at site level, \( T_S \), for a range of dispersal rates. Each data point gives the frequency of \( T_S \) being in the represented 5-year range (0–5, 5–10, etc). For each value of \( \mu \), data points are computed from 200 simulations with \( k = 1000 \) sites and \( T = 500 \). (b) The effect of the dispersal rate \( \mu \) on the expected persistence time at site level \( \varepsilon(T_S) \) and on the expected time to last occupancy \( \varepsilon(T_L) \). Parameters are \( k = 1000 \) sites, \( P_{\text{spill}} = 0.05 \) during 5 years, disturbance rate \( p = 0.15 \), each point is the average of 50 runs with \( T = 2000 \).
predictor for the expected persistence times of such patches is that most of the individual sites that make up such a patch are much shorter lived than the patch as a whole.

The time until last occupancy, $T_L$, takes into account that sites can be recolonised, and hence is a better measure of the persistence of long-lived patches. The dependence on the dispersal rate of the expected time of last occupancy, $\varepsilon(T_L)$, is plotted in Fig. 3b, along with the expected persistence time $\varepsilon(T_S)$. In the absence of dispersal, empty sites cannot be recolonised which implies that for $\mu = 0$ the values of $T_S$ and $T_L$ should coincide (Fig. 3b).

Both quantities increase approximately exponentially with $\mu$, albeit at very different rates: $T_L$ is much more sensitive to changes in $\mu$ than $T_S$. For a wide range of $\mu$, $T_L$ is much larger than $T_S$ which implies that long-term persistence is possible despite relatively short local persistence times. Visual inspection of space–time plots suggests that the long persistence times ($T_L$) are due to long-lived, static patches such as depicted in Fig. 2.

We have so far looked at dynamics for parameter values typical for oilseed rape (low $p$ and $\mu$) for which the metapopulation tends to go extinct. We now turn to answering the questions: will sufficient dispersal enable the metapopulation to persist indefinitely? Or is this possible only with a high disturbance rate ($p$), as was the case in the matrix model? The latter predicted persistence only if $p > 0.28$ (Claessen et al. 2005). For a fixed disturbance rate ($p = 0.15$), Fig. 4 shows the steady-state, average fraction of occupied sites $m(t)$ without seed spillage plotted against the dispersal rate $\mu$, as well as the population growth rates $\log \lambda_s$ and $\log \lambda_m$. Long-term persistence ($m(t) > 0$) is found only for unrealistically large dispersal rates ($\mu > 0.2$). The metapopulation growth rate $\log \lambda_m$ reaches positive values only for $\mu > 0.25$, implying that with smaller dispersal rates the number of occupied sites gradually declines. Interestingly, the metapopulation growth rate is considerably higher than the local population growth rate (i.e. $\log \lambda_m > \log \lambda_s$) throughout the range of $\mu$. As in the classical metapopulation model, global persistence ($\log \lambda_m > 0$) is therefore possible despite sure local extinction ($\log \lambda_s < 0$), but it does require a very high dispersal rate.

Next, we determine the extinction boundary which separates the set of parameter values ($\mu$, $p$) for which the metapopulation is expected to go extinct in the long run, from the parameter values for which it may persist indefinitely. The extinction boundary is defined by the condition $\log \lambda_m = 0$. In Fig. 4b we saw that the condition is fulfilled for the parameter values $\mu \approx 0.25$ and $p = 0.15$. The condition can be fulfilled with a low dispersal rate $\mu$ only by choosing a high disturbance rate (Fig. 5). For small $\mu$, the critical $p$ for persistence decreases roughly linearly with the logarithm of $\mu$ (Fig. 5a) and reaches a minimum around $\mu = 0.8$. Close to the $\mu = 0$ limit the metapopulation is highly sensitive to changes in $\mu$; even small values of $\mu$ have a major influence of the population growth rate. Overall, Fig. 5 shows that a larger dispersal ability allows the population to persist with a much lower disturbance rate. Yet there is a critical disturbance rate $p^* \approx 0.13$ below which long-term persistence is impossible irrespective of the dispersal rate.

The role of environmental unpredictability: spatial correlation

Here we study the effect of spatial correlation in the occurrence of disturbances on the population growth rates $\log \lambda_s$ and $\log \lambda_m$. Fig. 6 shows a gradual decrease
of the population growth rates with the spatial autocorrelation, \( r \), up to \( r \approx 0.9 \), after which they decrease sharply with \( r \). The same pattern is observed for \( \mu = 0.01 \) (results not shown) and hence does not depend on the specific value of \( \mu \). The beneficial effect of spreading risk by dispersal is hence robust to a large amount of spatial autocorrelation (up to \( r \approx 0.9 \)), while negative autocorrelation between neighbouring sites enhances it.

The large difference in growth rate between the cases of complete correlation (\( r = 1 \)) and little to mild correlation (\( r < 0.9 \)) shows that the positive effect of dispersal on population growth depends on the asynchrony of disturbances in neighbouring sites. With \( r = 1 \) disturbances occur simultaneously throughout the metapopulation, while with \( r < 1 \) disturbances occur asynchronously. In the latter case, if a plant distributes its seeds over three sites, the probability that at least some of the seeds encounter favourable conditions (i.e. disturbed habitat) is larger than \( p \). Thus, distributing seeds to neighbouring sites provides a degree of insurance against unfavourable conditions (Wiener and Tuljapurkar 1994). In a spatially random environment (\( r < 1 \)), this insurance has a positive effect on population growth, as has been shown for a two-patch model by Wiener and Tuljapurkar (1994), who called the phenomenon the ‘insurance effect’. When disturbances occur synchronously (\( r = 1 \)), long-lasting patches (i.e. groups of occupied adjacent sites) are not observed. The existence of patches thus depends on the insurance effect of dispersal in a spatially random environment.

**Elasticity and the role of density dependence**

Figure 7 summarizes the results of the elasticity analysis for both the density dependent case (black bars) and the density independent case (grey bars). In all panels the parameters are ranked according to the elasticity of the local population growth rate \( \lambda_s \) (Fig. 7a). In the density dependent case the local population growth rate \( \lambda_s \) responds most sensitively to changes in the winter survival rate in the seed bank, \( \sigma_2 \), followed by the dormancy fraction \( d \) and spring seedling survival rate \( S_t \) (Fig. 7a, black bars). Seed production \( F_t \), summer seed bank survival \( \sigma_3 \) and germination \( g_t \) appear to be less important. Also the dispersal rate \( \mu \) turns out to have a low elasticity. The winter survival rate of early emerged seedlings, \( \sigma_1 \), has negligible impact on metapopulation growth. The coefficient for density dependence, \( c_F \), has a negative elasticity because increasing density dependence reduces seed production and hence population growth. The coefficient has a very low elasticity, though, suggesting that density dependence is not of major importance given these parameter values. This may not come as a surprise because the metapopulation is declining and heading for extinction, so densities are expected to be low.

The same Fig. 7a shows that removing density dependence by choosing \( c_F = 0 \) makes little difference to the ranking of the life history parameters in terms of their elasticity. The same three parameters (\( \sigma_2 \), \( d \) and \( S_t \)) top the elasticity table and the overall pattern of elasticity is fairly robust to the presence or absence of density dependence. The only differences are that seed production \( F_t \) and germination \( g_t \) become slightly more important without density dependence, while

\[
\begin{align*}
\log \lambda_s &= \log \lambda_m = 0 \\
\end{align*}
\]
dispersal \((m)\) becomes less influential. The effect on the elasticity of \(F_t\) reflects that in our model density dependence works by reducing fecundity at higher densities, thus reducing the influence of seed production on population growth.

In terms of the metapopulation growth rate \(\lambda_m\) the elasticities show a similar ranking (Fig. 7b). The main difference is that, in the density-dependent case, the elasticity of \(F_t\) is smaller than that of \(\sigma_3\). As in Fig. 7a, the main effect of density dependence is to reduce the elasticity of \(F_t\) and \(g_t\). Overall Fig. 7a and b show good correspondence between the elasticity of \(\lambda_m\) and \(\lambda_s\) in terms of the ranking of parameters and the small effect of density dependence on elasticity.

The response of the expected time to last occupancy, \(\varepsilon(T_L)\), to changes in life history parameters reveals roughly the same ranking (Fig. 7c). Again seed bank survival \((\sigma_2)\) and dormancy \((d)\) are most important. Three parameters have intermediate elasticity: \(F_t\), \(S_t\) and \(\sigma_3\), while the remaining parameters have little influence. The coefficient of density dependence \(c_F\) appears to have somewhat more influence on persistence time than on both population growth rates. This is also apparent from the large difference between the elasticities with and without density dependence.

**Discussion**

The aims of this article were: (i) to assess the importance of dispersal and metapopulation structure for the persistence and extinction of feral, roadside populations; (ii) to study the dynamics of a structured metapopulation subject to environmental stochasticity, with a focus on extinction; (iii) to evaluate the robustness of our previous approach of using a matrix population model by relaxing the assumptions of spatial homogeneity and density independence.

Concerning the importance of dispersal, we have found that including metapopulation structure can enhance persistence times considerably because adjacent sites can re-establish each other after local extinctions. This is manifested by the existence of small groups of occupied sites, or “patches”, which remain more or less fixed in space and which are likely to persist much longer than the expected, site-level persistence time. This conclusion is based on the result that with a positive dispersal rate, the time to last persistence is much larger than the site-level persistence time \((T_L < T_S)\), implying that despite short expected local persistence times (e.g. 5–10 years), persistence over medium long term (e.g. 150 years) is likely to occur, albeit in just a few localised patches.

We have also found that in the case of roadside oilseed rape, dispersal is unlikely to result in permanent, global persistence at the metapopulation level, because this requires unrealistically high dispersal rates \((\mu > 0.1)\), given that the disturbance rate is likely to be small \((p < 0.2)\).
Concerning environmental stochasticity, we have found that the emergence of patches (i.e. groups of occupied sites) depends on the spatial asynchrony of disturbances. The results confirm the analysis by Wiener and Tuljapurkar (1994), who used an analytic approximation of the stochastic population growth rate to show that dispersal in a system of two patches and two life stages increases population growth provided that the cost of migration and the correlation between the sites are sufficiently small. They compared the effect of dispersal to an insurance against unfavourable conditions. Distributing seeds over a number of sites reduces the net variability experienced in the next generation. Environmental variability has a negative effect on population growth (Lewontin and Cohen 1969), and by reducing variability the insurance effect increases population growth. The positive effect of spreading risk, here attained by distributing offspring over a number of sites, is akin to the success of ‘bet hedging’ life history strategies which are often favourable in random environments (Cooper and Kaplan 1982). We have shown that the insurance effect works in a more complicated, metapopulation system and that when the metapopulation is declining to extinction the effect manifests itself as the occurrence of static, relatively long lasting patches (Fig. 2).

The insurance effect is distinct from the ‘rescue effect’ which is defined as a reduced probability of extinction of small, local populations owing to immigration (Brown and Kodric-Brown 1977). The mechanism of the rescue effect is that (i) immigration increases local abundance and (ii) large populations are less likely to go extinct than small ones (Gyllenberg and Hanski 1997). The underlying mechanism may be deterministic or the relation between population size on the effect of within-site demographic stochasticity on extinction. In contrast to the insurance effect, the rescue effect does not require environmental variability. Thus, while the rescue effect works through the effect of immigrants on local population dynamics, the insurance effect works through the effect of distributing offspring over a number of sites which reduces the net variability. Both effects can be expected to be operational in metapopulations subject to environmental stochasticity.

Concerning spatial heterogeneity and density dependence, the elasticity analysis carried out in this study yields results very similar to the elasticity analysis of a spatially unstructured, density independent matrix model (Claessen et al. 2005). Both studies identify the survival rate of seeds in the seed bank as the life history trait with most impact on population growth, followed by the dormancy fraction \( d \), followed by spring seedling survival \( S_s \) and seed production \( F_t \). Both models predict that changes in germination fraction \( g_0 \), dispersal rate \( \mu \) and winter seedling survival \( \sigma_w \) have less impact on population growth. As in Claessen et al. (2005), we found that life history traits that are critical to the population growth rate are also critical to persistence times: the elasticities of \( \lambda_w \), \( \lambda_m \) and \( \varepsilon(T_1) \) display roughly the same ranking (cf. Fig. 7a–c). The added value of the present study is that we can assess the importance of the dispersal rate, which was impossible in the context of our previous model. We found that the elasticity of \( \mu \) is small, both in terms of the (meta-) population growth rate and in terms of persistence time. Yet our analysis shows that close to \( \mu = 0 \) there is a strong non-linear effect of dispersal on the extinction boundary (Fig. 5). Close to \( \mu = 0 \) the population growth rate responds most sensitively to changes in \( \mu \), while the response flattens with increasing \( \mu \) (Wiener and Tuljapurkar 1994). The non-linear effect of dispersal is also well illustrated by the effect of spatial correlation of disturbances. The case of \( r = 1 \) resembles the non-spatial matrix model, because there is no scope for spreading risk through the insurance effect. The drastic effect of even the slightest asynchrony on disturbances, and hence of dispersal in a spatially random environment, is apparent from Fig. 6. There is thus a large difference between \( \mu = 0 \) and \( \mu > 0 \), as well as between \( r = 1 \) and \( r < 1 \), but given that \( \mu > 0 \) and \( r < 1 \), the effect of changes in \( \mu \) and \( r \) are relatively small.

Models that exclude dispersal and metapopulation structure underestimate both the population growth rate and persistence times. However, for oilseed rape we do not expect dispersal to allow for long term persistence, but it seems likely that isolated patches (i.e. groups of adjacent occupied sites) can persist for decades or possibly even centuries, despite most sites going extinct within 5 or 10 years.

There is anecdotal evidence for the existence of long-lasting patches of roadside oilseed rape, such as described by Crawley and Brown (1995) who report the occurrence a small number of “apparently permanent, dense rape populations” along the M25 motorway. The authors speculate that in these patches local seed production may be so high that it provides resistance to invasion by perennial grasses. Our theoretical study suggests that such patches may simply be the consequence of the interplay between dispersal and spatially random disturbances. The possibility that adjacent sites rescue each other from local extinction can explain the existence of quasi-permanent populations without the need to invoke additional mechanisms such as resistance against invasion or positive density dependence.

### Implications of genetic modification

The context for this study is to assess the risks of transgenic crops spreading into the environment. Feral transgenic populations may serve as sources or stepping stones of transgene flow to wild relatives, or may be an ecological threat in themselves, if they...
constitute persistent weeds in arable fields or dominate wild vegetation. It is therefore important to assess the consequences of genetic modification on population growth and persistence of feral populations.

One of the tools we have used to this end is elasticity analysis. It should be noted that elasticities express the effect on the population growth rate (or another population level quantity) resulting from a proportional change in the life history trait. Thus, by identifying the importance of a trait with its elasticity, as we do below, it is implicitly assumed that the effects of GM on different life history traits will have similar relative magnitude. To make a definitive assessment of which transgenes will have the biggest impact, it is necessary to have quantitative estimates of these proportional changes in life history traits. As many authors study the changes merely in terms of increase or decrease under laboratory conditions, such estimates are unfortunately rare. The quantitative estimates of changes in d as found by Gruber et al. (2004) are an exception. Thus, the discussion below necessarily has a more qualitative character; a more precise risk analysis will require more quantitative measurements of the effect of transgenes on life history traits.

In a previous study we linked observed transgene-induced life history changes in oilseed rape to predicted consequences for population growth and persistence (Claessen et al. 2005). Our present study shows that the conclusions from that paper are robust to the extension to metapopulation structure and density dependence. That is, transgenes that improve survival in the seed bank or the dormancy fraction are most likely to improve persistence of feral populations.

Our recommendation are hence that (i) GM seeds are tested for these life history traits, and (ii) that cultivars with low seedbank survival and low dormancy are used as parental lines whenever new transgenic crops are being developed. An example in which this has been done (perhaps inadvertently) is revealed by a recently published comparison of two transgenic oil-modified cultivars with their parental lines (Walker et al. 2004). The transgenic high-laurate cultivar LA002 was based on parental line cv. 212/86. Walker et al. (2004) claim this parental line displays an unusually poor ability to enter secondary dormancy (as measured by unusually high levels of initial germination). Owing to this poor performance of the parental line, even the oil-modified cultivar has a low dormancy compared to conventional cultivars despite the positive influence of the oil-modification on dormancy.

As we have pointed out previously (Claessen et al. 2005), comparison of our modelling results with known effects of transgenes on life history in oilseed rape (Table 1), leads to predictions on the effects of particular transgenic traits on feral persistence. High-laurate transgenes have been reported to increase seed survival (Table 1). The role of dormancy in high-stearate transgenes is ambiguous, however, as both positive (Linder 1998) and negative (Walker et al. 2004) effects have been reported. For high-laurate cultivars the effect on seed survival is not known, but both known effects (increased dormancy and increased germination rate) promote persistence (Table 1). Although these effects of oil-modification have not been found under all experimental conditions (Linder 1998), we conclude that oil-modification at least has the potential to promote feral persistence. Bt transgenes increase performance through plant survival and fecundity (Table 1), which are predicted to have a relatively small impact on fitness. It will therefore depend to a large extent on the unintentional effects on seed survival and dormancy whether Bt transgenes increase or decrease persistence. To our knowledge, the effect of Bt transgenes on seed survival and dormancy is currently unknown, so the net effect on persistence cannot be assessed. A recent publication compares dormancy of transgenic, herbicide (glufosinate) tolerant cultivars with that of their near-isogenic conventional counterparts. It shows that the transgenic cultivars Lilly and Modull (d = 0.14, 0.009, resp) have lower dormancy than the conventional ones Liberator and Falcon (d = 0.58, 0.05, resp). In combination with a previously established negative effect on seed bank survival (Table 1) this confirms the expectation that, in absence of herbicide application, this transgene does not convey a fitness advantage.

Acknowledgements – Rothamsted Research is supported by the Biotechnology and Biological Sciences Research Council (BBSRC).

References


Subject Editor: Tim Benton
Alternative Life-History Pathways and the Elasticity of Stochastic Matrix Models

David Claessen1,2,*

1. Biomathematics Unit, Rothamsted Research, Harpenden AL5 2JQ, United Kingdom;
2. Department of Plant Sciences, University of Cambridge, Downing Street, Cambridge CB2 3EA, United Kingdom

Submitted May 20, 2004; Accepted October 22, 2004; Electronically published November 18, 2004

Abstract: Loop analysis is a powerful tool for analyzing matrix population models. This note shows that the results of loop analysis, which have been proved for constant matrices only, apply to stochastic matrices as well if elasticity is defined as the effect of a proportional perturbation of both mean and variance. Using the ideas of loop analysis, it is shown that the structure of the stochastic matrix in terms of alternative life-history pathways has important consequences for the effect of stochasticity on elasticities. If the life cycle contains nonoverlapping, alternative life-history pathways, the ranking in terms of elasticity of the most critical vital rates may be reversed in stochastic and the corresponding average environments. This has obvious and important consequences for population management because focusing on a deterministic model would lead to an ineffective or counterproductive management strategy.

Keywords: elasticity analysis, life cycle pathways, matrix population models, environmental stochasticity, loop analysis, annual plants.

Recently, Tuljapurkar et al. (2003) provided an example of a stochastic matrix model where elasticities of the stochastic matrix are very different from elasticities of the matrix of average transitions. This result contradicts the prevailing consensus that stochasticity has little effect on elasticity and hence on the relative importance of different vital rates for population growth, which was based on earlier examples showing striking similarities between elasticity analysis of stochastic matrices and their average equivalents (Benton and Grant 1996; Grant and Benton 2000; Caswell 2001; Caswell and Kaye 2001; Silvertown and Charlesworth 2001).

Demographic studies of stochastic matrix models often focus on the long-term stochastic population growth rate (denoted by $\lambda$; Caswell 2001) and its elasticity (Fieberg and Ellner 2001). The common definition of stochastic elasticity (denoted by $E^s$; Tuljapurkar et al. 2003) is the proportional change in $\log \lambda$, caused by a proportional perturbation of both the mean and standard deviation of a matrix element. The consensus that stochasticity has little effect on elasticity is based on comparisons of $E^s$ with the elasticity of the average matrix (denoted by $E^A$).

In their model of a hurricane-dependent shrub species, Tuljapurkar et al. (2003) find a poor fit between $E^s$ and $E^A$, which they attribute to two aspects of environmental stochasticity that are not taken into account in the average matrix and in $E^A$: the effects of habitat sequencing and the variance in life-history traits. It remains to be understood, however, how in the earlier examples cited above, which included both these aspects, stochasticity had such small effect on elasticity despite a considerable effect on population numbers. Solving the apparent contradiction between the early and recent findings will advance our understanding of the role of environmental randomness for population dynamics.

The aim of this note is to complement the observation of Tuljapurkar et al. (2003) with a more detailed understanding of what causes differences between stochastic and average matrix elasticities. One factor causing deviation between stochastic and average matrix elasticities is when the level of stochasticity becomes very large (Benton and Grant 1996; Caswell 2001). Tuljapurkar et al. (2003) identify two other factors: first, if there are many possible environmental states and transitions between them are correlated and second, if the demography of the mean matrix converges slowly and cyclically to a stable population structure.

Here it is shown that the structure of the matrix, in terms of life-history pathways or “loops,” is critical in determining the fit between stochastic and mean matrix elasticities. First, I show that loop analysis, which was
proved for constant matrices only (van Groenendael et al. 1994), is valid for stochastic matrix models as well. Second, I study the implications of alternative life-history pathways for the effect of environmental stochasticity on (prospective) elasticity.

Tuljapurkar et al. (2003) show that the dynamics of a stochastic matrix model can be characterized by many different types of population growth rates, sensitivities, and elasticities and that each measure has its own interpretation. In addition to \( E^S \), they introduced two other kinds of stochastic elasticity: one that assumes perturbations of the mean only (denoted \( E^{m} \)) and one that assumes perturbations of the variance only (denoted \( E^{v} \)). While my focus is on the original definition (\( E^S \)), the validity of the results for the other types is discussed briefly.

**Loop Analysis**

Consider stochastic matrix population models of the form

\[
    n(t+1) = X_n n(t),
\]

with population vector \( n(t) \), stochastic matrix \( X_n \) and initial condition \( n(0) = n_0 \). The long-term stochastic population growth rate is denoted \( \lambda \) (defined in the appendix). The most commonly used type of stochastic elasticity measures the effect on \( \log \lambda \) of a simultaneous and proportional perturbation of the mean and variance of an element of \( X_n \) (appendix, eq. [A4]). The results below apply to this type of stochastic elasticity, unless stated otherwise.

A life-history pathway or loop is a single, closed loop formed by a subset of the arrows (transitions) in the life cycle graph (van Groenendael et al. 1994; Wardle 1998). Each loop can be assigned a loop elasticity, which corresponds to the contribution of this loop to the total population growth rate. The ideas of “loop analysis” are based on two mathematical properties of matrices and are expressed by van Groenendael et al. (1994) and critically re-examined by Wardle (1998). The first property is that “for each stage \( i \) in the life cycle graph, the summed elasticity of incoming transitions equals the summed elasticity of outgoing transitions”: \( \sum e_{ij} = \sum e_{ji} \) (van Groenendael et al. 1994, p. 2,411). The second property is that all loop elasticities together sum to 1, just like normal elasticities. These properties were proved for deterministic matrices by van Groenendael et al. (1994). In the appendix, I show that the first property holds for a stochastic matrix model as well. Van Groenendael et al.’s (1994) proof of the second property requires that the first property is true and that the sum of all elasticities equals 1. Since the latter is the case for stochastic elasticities (Tuljapurkar 1990), the proof in the appendix ensures that the second property also holds for stochastic matrices.

A consequence of the first property is that within a loop all transitions have the same elasticity, which is termed the characteristic elasticity of that loop. It is denoted \( c_i \) where \( k \) is an index of the loop. The total loop elasticity equals \( c_i \) multiplied by the number of transitions in the loop. The elasticity of a transition rate \( a_{ij} \) in matrix \( A \) equals the sum of the characteristic elasticities of all loops that pass through element \( a_{ij} \). Loops link the elasticities of different matrix elements. Stochastic variation in a transition rate \( a_{ij} \) affects the characteristic elasticity of all loops passing through \( a_{ij} \). Similarly, \( c_i \) depends on stochastic variation in all transitions that are part of the \( k \)th loop.

Some ideas from loop analysis are illustrated by the following simple examples. Consider a 3 x 3 age-structured Leslie matrix \( A \), containing a single loop only, with \( p_1 \) and \( p_2 \) the survival rates at ages 1 and 2, respectively, and \( f_2 \) the fecundity of age 3 (fig. 1a). Because there is only a single loop, it follows from loop analysis that all nonzero elements of \( A \) have the same elasticity, equal to the characteristic elasticity \( c_i \). The transition matrix \( A_i \) and the corresponding elasticity matrix \( E_i \) are

\[
A_i = \begin{pmatrix} 0 & 0 & f \ \\
            p_1 & 0 & 0 \\
            0 & p_2 & 0 \end{pmatrix}, \quad E_i = \begin{pmatrix} 0 & 0 & c_i \ \\
            c_i & 0 & 0 \\
            0 & c_i & 0 \end{pmatrix}, \quad (2)
\]

with \( c_i = 1/3 \) irrespective of the values of \( p_1 \), \( p_2 \), and \( f \).

By allowing stage 3 individuals to survive annually with probability \( s \), the following (stage-structured) transition matrix and corresponding elasticity matrix is obtained:

\[
A_2 = \begin{pmatrix} 0 & 0 & f_3 \\
            p_1 & 0 & 0 \\
            0 & p_2 & s \end{pmatrix}, \quad E_2 = \begin{pmatrix} 0 & 0 & c_1 \ \\
            c_1 & 0 & 0 \\
            0 & c_1 & c_2 \end{pmatrix}. \quad (3)
\]

Matrix \( A_2 \) contains two loops: 1-2-3-1 and the 3-3 self loop. The two loops are nonoverlapping because they do not share any arrows (transitions) in the life cycle graph (fig. 1b). In consequence, the elasticity of any nonzero matrix element is either \( c_i \) or \( c_j \).

By contrast, by modifying the original matrix by allowing age-2 individuals to reproduce, matrix \( A \), with two overlapping loops (fig. 1c) is obtained. The elasticity of the element that belongs to both loops (element \( a_{ij} \)) is therefore \( c_1 + c_2 \) and necessarily the largest:

\[
A_3 = \begin{pmatrix} 0 & f & f_3 \\
            p_1 & 0 & 0 \\
            0 & p_2 & 0 \end{pmatrix}, \quad E_3 = \begin{pmatrix} 0 & c_2 & c_1 \\
            c_1 + c_2 & 0 & 0 \\
            0 & c_1 & 0 \end{pmatrix}. \quad (4)
\]
Here, $A_1$ is a $3 \times 3$ Leslie matrix with $f_i = 0$. Note that in any Leslie matrix of size $m \times m$ (assuming that the loops are indexed such that the loop that passes through the fertility of the last class is loop 1, of the previous class is loop 2, etc.), the elasticity $e_{11}$ equals $\sum_{i=1}^{m-1} c_i$, while $e_{12} = \sum_{i=1}^{m-1} c_i$, and so forth. This implies that for all the subdiagonal elasticities, $e_{i+1,i} > e_{i+1,i}$, and $e_{i+1,i} \geq e_{j+1,j}$ for all $j > i$. In other words, owing to the overlap of the loops, the elasticity of $p_i$ exceeds that of all $p_j$ and $f_j$ with $j > i$. Only the elasticity of the 1-1 self loop ($e_{11} = c_{11}$) can exceed $e_{11}$, provided that $f_i > 0$.

The Effect of Environmental Stochasticity

Excluding a number of special cases, a general result is that increasing variability in a matrix element has a negative effect on the population growth rate (Tuljapurkar 1990; Caswell 2001). Loop analysis tells us that increasing variability in one element will affect the elasticity of all elements that share loops with this element. Using the three examples above, I discuss the possible implications of stochasticity depending on the structure of the matrix.

The case of a single loop (eq. [2]) is almost trivial. From the first property, it follows that environmental stochasticity in any or all of the nonzero elements of $A_1$ simply cannot affect the elasticity matrix because all elasticities must equal $c_i$. This is true for single-loop matrices of any size. The size of the matrix (or the length of the loop) affects only the value of $c_i$, which is $c_i = 1/L$, where $L$ is the length of the loop.

In the case of two distinct loops as in equation (3), environmental stochasticity can affect elasticity. If only a single element is affected by stochasticity, it will generally decrease the elasticity of the loop it belongs to because the loop’s contribution to population growth decreases; it will also increase the elasticity of the other loop because its relative contribution to population growth increases. When elements in both loops are stochastic, the loop that experiences the lowest variability will increase in elasticity while the other one will lose elasticity. This is illustrated with some numerical examples below.

In the case of Leslie matrices with at least one nonproductive age class ($f_i = 0$) and at least two reproductive classes such as $A_1$ (eq. [4]), loop analysis shows that there exists a clear ranking of elasticities in which the survival rates $p_i$ generally have a high elasticity because these transitions belong to many loops. This inherent ranking due to the structure of the matrix implies that there is limited scope for stochasticity to result in rank reversals.

Numerical Examples

Consider the above model (eq. [1]), and assume that at each time step a matrix is selected, randomly depending on the state of the environment. Suppose the environmental conditions can be either “good” or “bad,” with probability $P$ and $1 - P$, respectively, and that

$$X_i = \begin{cases} g(v) & \text{with probability } P \\ b(v) & \text{otherwise} \end{cases}$$

(5)

where $g(v)$ and $b(v)$ are the matrices for good and bad years, respectively. The parameter $v \in (0, 1)$ is introduced to study the effect of variation. It determines the difference between the two matrices

$$g(v) = U + v(G - U),$$

(6)

$$b(v) = U + v(B - U),$$

(7)

where the average matrix $U$ is defined as

$$U = P G + (1 - P) B$$

(8)

and $G$ and $B$ are two distinct matrices. Choosing $v = 1$ gives maximum variation $g(1) = G$ and $b(1) = B$. In the absence of variability ($v = 0$), the average matrix is obtained, $g(0) = b(0) = U$, and the stochastic elasticity (eq. [A4]) then equals the average matrix elasticity ($E^S = E^S$).

Below, stochastic elasticities (eq. [A4]) are computed from observed population structure in simulations with $T = 50,000$ using the method of Tuljapurkar et al. (2003).
First, consider model (1) with the following matrices for good and bad years, respectively:

\[
G = \begin{pmatrix}
0 & 0 & 15 \\
0.3 & 0 & 0 \\
0 & 0.4 & 0.5
\end{pmatrix}, \quad B = \begin{pmatrix}
0 & 0 & 0 \\
0.2 & 0 & 0 \\
0 & 0.3 & 0.4
\end{pmatrix},
\]

with \( P = .5 \). The stochastic matrix \( X_s \) (eq. [5]) has the same structure as \( A_s \) (eq. [3]) and contains two loops, 1-2-3-1 and 3-3. In good years, adult plants (stage 3) have a high fecundity, but in bad years they cannot reproduce. Adults can survive bad years, however, creating the second loop of the life cycle. Figure 2A shows the relation between the stochastic elasticities of the nonzero elements of \( X_t \) and \( v \). First, despite elements \( x_{13}, x_{21}, \) and \( x_{32} \) being subject to different levels of variability, they always have the same elasticity, which corresponds to the characteristic elasticity \( c_1 \) of the loop they belong to. Second, note that as \( v \) increases, the larger variation in the first loop reduces its characteristic elasticity \( (c_1) \), while the relative contribution of the adult-stage self loop \( (c_2 = e_3) \) increases.

Next, consider a life cycle of the structure as in \( A_s \) (eq. [4]), with two loops that overlap in one transition \( (x_{32} = p_1) \). Based on loop analysis, I expect the elasticity of element \( x_{11} \) to be highest, irrespective of the level of stochasticity. To illustrate this, I deliberately choose a large variation in \( p_1 \):

\[
G = \begin{pmatrix}
0 & 10 & 5 \\
0.95 & 0 & 0 \\
0 & 0.4 & 0
\end{pmatrix}, \quad B = \begin{pmatrix}
0 & 0 & 5 \\
0.05 & 0 & 0 \\
0 & 0.3 & 0
\end{pmatrix}.
\]

Figure 2B confirms that the elasticity of element \( x_{11} \) is always highest (and equal to the sum of the other two values), as expected from loop analysis (eq. [4]). In addition, figure 2B shows that the larger variation in loop...
1-2-1 reduces its characteristic elasticity \( (c_1 = e_{11}) \) while that of loop 1-2-3-1 increases \( (c_1 = e_{11}) \).

I complement these hypothetical examples with a biologically more realistic one, based on a study of volunteer (weedy) populations of oilseed rape \( (Brassica napus) \). The life cycle consists of flowering plants (stage 1), seeds in the shallow seed bank (stage 2), and seeds in the deep seed bank (stage 3). The self loop within the flowering plants \( (1-1) \) corresponds to the annual life-history pathway, while self loops also exist in the two seed bank stages \( (2-2 \text{ and } 3-3) \). In addition, there are biennial and triennial loops involving exchange between seed bank layers or between flowering plants and the seed bank. The matrices for good and bad years are, respectively,

\[
G = \begin{pmatrix}
14.7 & 0.0029 & 0.042 \\
1.49 & 0.0012 & 0.017 \\
970 & 0.78 & 0.57
\end{pmatrix},
\]

\[
B = \begin{pmatrix}
0.019 & 0 & 0.0001 \\
0.89 & 0.0018 & 0.026 \\
388 & 0.78 & 0.57
\end{pmatrix} \quad (11)
\]

(Claessen et al. 2005). Note that there is no variation in survival in the deep seed bank \( (x_{13}) \), while there is considerable variation in the annual loop \( (x_{11}) \). For volunteer oilseed rape, the difference between good and bad years derives mainly from the probability of a small patch of plants to escape control measures, which I assume to be \( P = .1 \).

Figure 2C shows that for the average matrix \( (v = 0) \), the annual loop of flowering plants has the highest elasticity. For intermediate \( v \), the highest elasticity is found for elements \( x_{13} \) and \( x_{13} \), which make up the 1-3-1 loop between flowering plants and the deep seed bank. Figure 2C clearly shows a reversal in the contribution of the 1-1 and 3-3 self loops with increasing \( v \); for \( v = 1 \), the deep seed bank self loop, which itself is unaffected by stochasticity, contributes most to population growth.

In the numerical examples above \( (\text{eqq.} \: [9]-[11]) \), most of the covariances are positive. To check for the effect of covariance, simulations were repeated with negative covariances obtained by swapping matrix elements between \( G \) and \( B \). This had small quantitative but no qualitative effects on the curves in figure 2. Further analysis of the effect of covariances in more complicated matrices is left as a topic for future work.

**Discussion**

Starting from the observation of Tuljapurkar et al. (2003) that stochastic elasticities can be very different from average matrix elasticities, this note aimed at understanding in more detail the differences between these two kinds of elasticities. For the common definition of stochastic elasticity \( (E^{	ext{p}}) \), loop analysis yields the following results. First, environmental stochasticity can affect elasticities only if the life cycle graph contains alternative life-history pathways (loops). Second, if life-history pathways overlap, the overlapping transition(s) have a higher elasticity than the nonoverlapping transition(s), irrespective of the level of stochasticity or which elements are subject to stochasticity. This implies that rank reversals of the most important transitions in terms of their elasticity (such as observed in fig. 2A, 2C) are possible only if the life cycle graphs contain nonoverlapping loops. When loops are overlapping, rank reversals are possible only among less important transitions (such as observed in fig. 2B).

Further, the effect of stochasticity on the characteristic elasticity of a loop depends on the level of variability within the loop compared to that level in other loops. Unfortunately, I have not been able to quantify the relevant measure of loop-specific variability to arrive at a more rigorous condition. This remains open to future research. However, the effect of large versus small variability of matrix elements on the relation between elasticity and \( v \) is clear in the examples: large variation in loop 1-2-3-1 in equation (9) results in reduction of \( c_1 = e_{11} \) with \( v \) and an increase in \( c_2 = e_{12} \) (fig. 2A). The larger variation in loop 1-2-1 in equation (10) results in reduction of \( c_2 = e_{12} \) and an increase in \( c_1 = e_{11} \) (fig. 2B). The absence of variation in loop 3-3 in equation (11) eventually results in the deep seed bank being most critical in the life cycle of volunteer oilseed rape (fig. 2C).

Together, these findings suggest that the largest effect of stochasticity on elasticities, in particular in terms of their ranking, is to be expected when the life cycle graph contains alternative life-history pathways and when the loop with highest elasticity in the average matrix is subject to the largest variation. As illustrated by figure 2C, this configuration can be expected in models of annual plants with a seed bank because, first, the seed bank is a stable environment that buffers against environmental variability; second, the aboveground processes are likely to be subject to environmental variability; and third, the aboveground processes are essential to population growth. By contrast, life cycles that are abundant in overlapping loops, such as Leslie matrices with multiple adult stages \( (\text{e.g.,} \: \text{eq.} \: [3]) \), are less likely to be strongly affected by stochasticity because the overlapping transitions will have highest elasticity with or without stochasticity. Note, however, that as stochasticity increases, population numbers will become more variable even if elasticity is not affected (such as in the case of a single loop).

Life cycles in which transitions with high average matrix elasticity are buffered against environmental stochasticity
are predicted to be insensitive to the effect of stochasticity on elasticity because, at most, the effect will be that stochasticity lowers the low-ranking elasticities. Interestingly, Pfister (1998) found a negative correlation between the variance in life-history trait (i.e., matrix element) and its contribution to population growth (i.e., elasticity of the average matrix) in a survey of 17 populations (but see Morris and Doak 2004). If I can take this as a general pattern, then I expect that in many cases of natural populations stochasticity has indeed only a small effect on elasticity.

The question of why the earlier examples cited in the introduction showed a good fit between stochastic and average matrix elasticities can now be addressed with these new findings in mind. In the case of *Arisaema triphyllum* studied by Caswell (2001), the matrix element with highest average matrix elasticity \( (a_{ij}) \) has practically no variation, while elements with most variation are the ones with the lowest elasticity (cf. Pfister 1998). I hence do not expect stochasticity to affect elasticities significantly. In the model of (Caswell and Kaye 2001), the element with the highest average matrix elasticity \( (a_{ij}) \) is subject to small variation: the fecundities \( a_{i5} \) and \( a_{i6} \) display a >10-fold higher coefficient of variation (CV). However, the effect of stochasticity on the elasticity of these elements is diluted by the much smaller CV of other elements that belong to the same loops (e.g., survival rates up to stage 5). Benton and Grant (1996) studied life cycles of the Leslie type such as equation (3). Due to the large amount of overlap in such life cycles and the implicated ranking of elasticity, it is not surprising that they did not find large discrepancies between stochastic and deterministic elasticities.

**Other Kinds of Stochastic Elasticities**

The results presented above apply to one specific kind of stochastic elasticity \( (E^S) \), which assumes simultaneous perturbation of mean and variance of a matrix element. For the two recently introduced types (denoted by \( E^{sw} \) and \( E^{se} \); Tuljapurkar et al. 2003), loop analysis is not expected to be valid. First, these elasticities do not sum to 1 \( (\sum e_i \neq 1) \), and second, the proof in the appendix does not hold for these definitions. My results hence do not generally hold for \( E^{sw} \) and \( E^{se} \).

I have computed \( E^{sw} \) and \( E^{se} \) for the examples studied above. There is no apparent relation between \( E^{sw} \) and \( E^S \) (data not shown). There is more correspondence between \( E^{se} \) and \( E^S \). From their definitions (Tuljapurkar et al. 2003), it follows that in the limit of no variation in a matrix element, they are equal \( (E^{sw}_{ij} = E^S_{ij} \text{ if } CV_{ij} = 0) \). The results of \( E^{sw} \) for the oilseed rape model (eq. [11]) are depicted in figure 2D. Comparison of figure 2C and 2D indeed shows that \( E^{sw} \approx E^S \) for matrix elements with little variation, for example, for \( e_{s3} \) and \( e_{s1} \). Large discrepancies, however, are found for elements with large variation, for example, \( E^{sw} \approx E^S \) for \( e_{13} \) and \( e_{s1} \) (note that the CV of elements \( x_{s3} \) and \( x_{s1} \) is, respectively, 2.98 and 0.39 if \( v = 1 \)).

The invalidity of loop analysis for \( E^{sw} \) is most clearly illustrated by the case of a single loop, in which case the \( E^S \) values are necessarily all equal to the characteristic loop elasticity, but the \( E^{sw} \) values of matrix elements with non-zero variation deviate from this value (data not shown). A thorough analysis of the relation between the three types of stochastic elasticity is beyond the scope of this note. Here, I merely note that the results obtained in this note for \( E^S \) appear to carry over only to \( E^{sw} \) and then only in cases with little stochastic variation in all matrix elements.

**Conclusions**

Tuljapurkar et al. (2003) define and analyze three growth rates and five kinds of elasticities, concluding that, in general, they are different. In particular, their observation that average matrix elasticity is a poor predictor of stochastic elasticity contradicts prevailing ecological consensus. They stress that by using the average matrix, one discards all information on habitat sequencing, which explains the differences between \( E^S \) and \( E^S \). Complementary to their general perspective, this note aims at understanding in more detail the relation between two types of elasticity, \( E^S \) and \( E^S \), which are the most commonly used types. Using the theory of loop analysis, which I have shown to be valid for stochastic matrices (but only for \( E^S \)), I have shown that the structure of the stochastic matrix in terms of alternative life-history pathways has important consequences for the effect of environmental stochasticity on the pattern of elasticities.

**Acknowledgments**

I thank C. A. Gilligan and F. van den Bosch for their support and H. de Kroon and S. Kalisz for a constructive discussion on elasticity during their visit at Rothamsted. I thank the three anonymous reviewers for their thoughtful comments. The critical remarks of one reviewer in particular helped to greatly improve the manuscript. This work was supported by the Biotechnology and Biological Sciences Research Council.
APPENDIX

Extension of First Property of Loop Analysis to Stochastic Matrices

Consider model (1). Following Caswell (2001), normalizing the population vector gives us the population structure, \( \mathbf{w}(t) = \mathbf{n}(t)/\|\mathbf{n}(t)\| \). The dynamics of \( \mathbf{w}(t) \) are

\[
\mathbf{w}(t+1) = \frac{\mathbf{X}\mathbf{w}(t)}{\|\mathbf{X}\mathbf{w}(t)\|}.
\]  
(A1)

The growth factor in year \( t \) is defined as \( R_t = \|\mathbf{X}\mathbf{w}(t)\| \). The reproductive value vectors \( \mathbf{v}(t) \) follow

\[
\mathbf{v}^T(t-1) = \frac{\mathbf{v}^T(t)\mathbf{X}_{t-1}}{\|\mathbf{v}^T(t)\mathbf{X}_{t-1}\|}
\]  
(A2)

(Caswell 2001), where \( T \) denotes transpose. I define the growth factor of the reproductive values from year to year as \( \lambda_t \). The stochastic population growth rate is defined as

\[
\log \lambda_t = \lim_{T \to \infty} \frac{1}{T} \sum_{i=0}^{T} \log R_t
\]  
(A3)

(Tuljapurkar 1990). The elasticity of \( \log \lambda_t \) to matrix element \( x_{ij} \) is defined as

\[
e_{ij} = \frac{\partial \log \lambda_t}{\partial \log x_{ij}} = \lim_{T \to \infty} \frac{1}{T} \sum_{i=0}^{T-1} \frac{x_{ij}(t)v_i(t)w_j(t)}{R_t v^T(t+1)w(t+1)},
\]  
(A4)

which is stochastic elasticity of type \( E^S \) (Tuljapurkar et al. 2003). Below I will use the relation

\[
\mathbf{v}^T(t+1)\mathbf{w}(t+1) = \mathbf{v}^T(t)\mathbf{w}(t) \frac{Q_t}{R_t},
\]  
(A5)

which can be proved by substitution of expressions (eqq. [A1], [A2]):

\[
\mathbf{v}^T(t+1)\mathbf{w}(t+1)R_t = \mathbf{v}^T(t)\mathbf{w}(t)Q_t
\]  
(A6)

\[
\Leftrightarrow \mathbf{v}^T(t+1)\frac{\mathbf{X}\mathbf{w}(t)}{\|\mathbf{X}\mathbf{w}(t)\|}R_t = \frac{\mathbf{v}^T(t+1)\mathbf{X}}{\|\mathbf{v}^T(t+1)\mathbf{X}\|} \mathbf{w}(t)Q_t
\]  
(A7)

\[
\Leftrightarrow \mathbf{v}^T(t+1)\mathbf{X}\mathbf{w}(t) = \mathbf{v}^T(t+1)\mathbf{X}\mathbf{w}(t).
\]  
(A8)

**Property 1 of Loop Analysis**

To show that

\[
\sum_i e_{ij} = \sum_j e_{ij},
\]  
(A9)

write the left-hand side of equation (A9) as

\[
\sum_i e_{ij} = \lim_{T \to \infty} \frac{1}{T} \sum_{i=0}^{T-1} \sum_j \frac{x_{ij}(t)v_i(t+1)w_j(t)}{R_t v^T(t+1)w(t+1)}.
\]  
(A10)

Denote the innermost summation in equation (A10) by \( Z_i(t) \):
\[
\sum_j e_{ij} = \lim_{T \to \infty} \frac{1}{T} \sum_{t=0}^{T-1} Z(t), \quad (A11)
\]

and rewrite it as follows:

\[
Z(t) = \sum_j x_{ij}(t) v_j(t+1) w_i(t) = \frac{v_j(t+1)}{v(t+1) w(t+1)} \sum_j x_{ij}(t) w_i(t) R_i. \quad (A12)
\]

From equation (A1), it follows that \(\sum x_j w_i(t) / R_i = w_i(t+1)\), so

\[
Z_i(t) = \frac{v_j(t+1) w_i(t+1)}{v(t+1) w(t+1)}, \quad (A13)
\]

Similarly, for the expression of the summed outgoing elasticities \(\sum e_{ip}\)

\[
\sum_j e_{ip} = \lim_{T \to \infty} \frac{1}{T} \sum_{t=0}^{T-1} Y_i(t), \quad (A14)
\]

with \(Y_i(t)\) defined as

\[
Y_i(t) = \sum_j x_{ij}(t) v_i(t+1) w_j(t) = \frac{w_i(t)}{v(t+1) w(t+1)} \sum_j v_j(t+1) x_{ij}(t) R_i. \quad (A15)
\]

From equation (A2), it follows that \(\sum v_j(t+1) x_{ij}(t) = v_i(t) Q_i\), and hence,

\[
Y_i(t) = \frac{v_i(t) w_i(t)}{v(t+1) w(t+1) R_i} Q_i. \quad (A16)
\]

Using equation (A5), this can be rewritten as

\[
Y_i(t) = \frac{v_i(t) w_i(t)}{v(t) w(t)} = Z_i(t-1). \quad (A17)
\]

Because \(Y_i(t) = Z_i(t-1)\), the long-term averages of \(Y_i\) and \(Z_i\) (eqq. [A11], [A14]) are the same, and hence equation (A9) is true.

**Literature Cited**


Associate Editor: Daniel F. Doak
Bioenergetics, overcompensation, and the source–sink status of marine reserves

David Claessen, Anneke S. de Vos, and André M. de Roos

Abstract: One of the hypothesized functions of marine protected areas (MPAs) is to serve as sources of biomass, with biomass spilling over from the reserve into neighbouring, harvested areas. We argue that the net larval flow (from or to the marine reserve) depends on between-area differences in the population-level biomass production rate, whereas the direction of adult flow depends on differences in the biomass standing stock. Hence, an important question is whether population-level biomass production increases (overcompensation) or decreases (undercompensation) with increased per capita mortality. We show that in a consumer–resource context, the source–sink status of an MPA may depend on the details of the individual-level bioenergetics, as well as on the dispersal rates of larvae and adults. We compare two classic bioenergetic models (net-production vs. gross-production allocation). The net-production model predicts that population-level reproduction may increase with mortality (overcompensation), whereas gross-production allocation always results in undercompensation. We show that models often implicitly assume gross-production allocation, thus potentially overestimating the capacity of MPAs to source unprotected areas. We briefly discuss results of two other models (a simplified, logistic model and a size-structured model), suggesting that the relation between overcompensation and the larval sink status of MPAs is general.

Résumé : Une des fonctions présumées des zones de protection marine (« MPA ») est de servir de source de biomasse, cette biomasse débordant alors de la réserve vers les zones exploitées adjacentes. Nous soutenons que le flux net de larves (depuis ou vers la réserve marine) dépend des différences entre les deux zones du taux de production de la biomasse au niveau de la population, alors que la direction du flux des adultes dépend des différences de biomasse des stocks. Cela soulève une question importante à savoir si la production de biomasse au niveau de la population augmente (surcompensation) ou diminue (sous-compensation) en fonction d’une augmentation de la mortalité par individu. Nous montrons que, dans un contexte de consommateurs et de ressources, le statut de source ou de piège d’une MPA peut dépendre des détails de la bioénergétique au niveau individuel, mais aussi des taux de dispersion des larves et des adultes. Nous comparons deux modèles bioénergétiques classiques (allocation de la production brute ou de la production nette). Le modèle de production nette prédit que la reproduction au niveau de la population peut augmenter en fonction de la mortalité (surcompensation), alors que l’allocation de la production brute entraîne toujours une sous-compensation. Nous montrons que les modèles présupposent souvent une allocation de la production brute, surestimant ainsi la capacité des MPA d’alimenter les zones non protégées. Nous discutons brièvement des résultats de deux autres modèles (un modèle logistique simple et un modèle structuré en fonction de la taille) et nous croyons que la relation entre la surcompensation et le statut des MPA comme pièges pour les larves est une relation générale.

[Traduit par la Rédaction]

Introduction

Marine reserves (or marine protected areas, MPAs hereafter) introduce a spatial heterogeneity into ecosystems that is caused by spatial variation in mortality rates of harvested populations. This results in an imbalance between protected and unprotected areas not only in terms of standing biomass of populations in the food web (Halpern and Warner 2002; Micheli et al. 2004b) but also, by consequence, in terms of ecological interactions, such as competition for resources, predation, etc. This imbalance is the driving force behind the potential of marine reserves to serve as biomass sources (through spillover) for the harvested areas, possibly leading to equal or even enhanced yield compared with conventional, effort-based fisheries management (Mangel 1998; Neubert 2003; Hart 2006). This simple vision of marine reserves suggests that two aspects are important for the dynamics of MPAs and surrounding areas: (i) the local ecological response of the food web to the presence or absence of harvesting, and (ii) the exchange of individuals through migration (Walters 2000; Mangel and Levin 2005). Mathematical models have been used to investigate the consequences of different assumptions on these two aspects for
both the economic and conservational implications of MPAs 
(reviewed in Gerber et al. 2003; Baskett et al. 2007; Pellet- 
tier et al. 2008), including the roles of predation, competi-
tion, and mutualism (Baskett et al. 2007), spatial processes 
(Botsford et al. 2001; Guichard et al. 2004), juvenile–adult 
stage structure (St. Mary et al. 2000), and density-dependent 
growth (Gårdmark et al. 2006).

In this paper, we show that a fundamental but usually im-
licit assumption concerning individual-level energy alloca-
tion affects both aspects (i) and (ii) in a potentially important 
way, which has been ignored in most previous modelling 
work. We study two alternative, well-known models for 
individual-level bioenergetics. A bioenergetics model answers 
the question as to how much energy is allocated to growth, re-
production, and maintenance, given a quantity of food in the 
environment. In the literature, different models of energy allo-

cation exist, which can be divided into two classes: gross-
production allocation and net-production allocation. The 
distinction between the two classes is that reproduction is 
supposed to be proportional to the energy intake rate either 
before or after maintenance costs have been subtracted. By 
consequence, the allocation models assume different be-
vaviour at low food conditions: in the gross-production model, 
individuals reproduce as long as the food concentration is 
positive, whereas in the net-production model, there is a 
threshold food concentration below which individuals stop 
reproducing (Gurney et al. 1996). Simple MPA models for-
mulated in terms of ordinary or partial differential equations 
(ODEs or PDEs, respectively) are often based on Lotka– 
Volterra type models (Neubert 2003; Steele and Beet 2003; 
Baskett et al. 2006), without explicitly considering bioener-

gics. We show that such models are equivalent to an im-
licit assumption of gross-production allocation.

In the context of size-structured population models, Gur-
ney et al. (1996) have shown that the individual-level ener-
getics influence by which demographic process populations 
are regulated (i.e., reproduction, maturation, or survival lim-
itation). The relevance of such individual-level assumptions 
in the context of MPAs is their implications for the local 
ecological response to the presence or absence of harvesting 
(aspect (i) above); the food web response will depend on 
how individuals react to (changes in) their environment. To 
illustrate this point, in this paper we study the consequences 
of different bioenergetic assumptions embedded in a simple 
food web consisting of a harvested consumer population 
(without size structure) and its resource population, based 
on the model of Yodzis and Innes (1992). Space is divided 
into a protected area and a harvested area, between which 
consumers (but not the resource) are assumed to migrate. 
Our model allows the dispersal rates of larvae and adults to 
tune independently. Density dependence results from 
competition for, and depletion of, the resource. The resource 
density inside and outside the MPA influences the energy 
budget and hence the respective rates of biomass growth 
and reproduction of the consumers in the two areas.

We thus address the question of how density-dependent 
food limitation, and its consequences for growth and repro-
duction, will affect projected effects of spillover from ma-

rmed reserves. One of the hypothesized functions of marine 
protected areas (MPAs) is to serve as sources of biomass, 
with biomass spilling over from the MPA into neighbouring, 
harvested areas. We show that in a consumer–resource con-
text, the source–sink status of the MPA may depend on 
the details of the individual-level bioenergetics, as well as on 
the dispersal rates of larvae and adults. We investigate the 
robustness of our model results by comparing the results 
with two alternative models: a simpler one (based on the lo-
gistic model) and a more complex one (a size-structured 
model). The latter is of relevance as the origin of the energy 
allocation models lies in the context of size-structured mod-
els. In particular, size-structured models allow for different 
kinds of population regulation (maturation vs. reproduction 
limitation; de Roos et al. 2007), which may have consequen-
ces for the success of marine reserves.

The model

We model the biomass dynamics of a harvested fish pop-
ulation and its forage base, taking into account the 
consumer–resource interaction between these two popula-
tions, as well as a simple representation of the bioenergetics 
underlying the fish biomass dynamics. To introduce the no-
tation, we first present a spatially undivided version of the 
model, based on Yodzis and Innes (1992). The biomass dy-
namics of the fished stock (C for consumer) and its forage 
base (R for resource) are modelled as follows:

\[
\frac{dC}{dt} = g(R)C + b(R)C - \delta C
\]

\[
\frac{dR}{dt} = \rho(R_{\text{max}} - R) - j(R)C
\]

where C and R are biomass per unit of sea surface area. The 
fish dynamics are governed by biomass production though 
somatic growth g(R) and birth b(R), which depend on the 
current food density R, and mass-specific fish mortality, 
which is denoted by \(\delta\). The forage base R follows semi-
chemostat dynamics with dilution rate \(\rho\) and maximum re-
source density \(R_{\text{max}}\). The function \(j(R)\) is the mass-specific 
functional response of fish.

Note that \(g(R)\) and \(b(R)\) are production rates per unit of 
standing stock biomass. Below we make frequent use of the 
population-level production rates, which are defined as \(G = g(R)C\) and \(B = b(R)C\), respectively.

Energy budget: biomass growth, reproduction, and 
maintenance

The birth and somatic growth terms \((b(R)\) and \(g(R)\)) are 
based on simple bioenergetic considerations, based on 
the model of Yodzis and Innes (1992). Their model assumes 
that the production rate of biomass depends on the mass-
specific ingestion rate \((j(R))\), the loss rate due to mainte-
nance costs \((m)\), and the loss rate due to mortality \((\delta)\):

\[
\frac{dC}{dt} = C[\sigma j(R) - m - \delta]
\]

where \(1 - \sigma\) is the fraction lost as feces and urine between 
\(\text{ingestion and the metabolizable energy level. We assume that the consumer intake rate, } j(R), \text{ follows either a type-I or a type-II functional response, i.e., respectively,}\)
\[ j(R) = aR \quad \text{or} \quad j(R) = \frac{R}{R_h + R} j_{\text{max}} \]

where \( a \) is an attack rate, \( R_h \) is the half-saturation density, and \( j_{\text{max}} \) is the maximum intake rate. The Yodzis and Innes model (eq. 3) may be a sufficient description of the bioenergetics for a single population (in spite of its simplicity in terms of the absence of size or stage structure (de Roos et al. 2007) or energy storage (Kooijman 2000)). Yet for spatially subdivided populations with larval dispersal, such as in the case of marine reserves, it is necessary to at least specify the allocation of resources between reproduction, maintenance, and growth, because we need to know which part of the biomass production will be redistributed in the form of larval dispersal over the spatial range of the population.

**Net-production allocation vs. gross-production allocation**

Maintenance, reproduction, and somatic growth all derive from the assimilation rate, \( \sigma j(R) \) (Kooijman 2000). Two major types of energy allocation models have been proposed, which are referred to as net-production allocation and gross-production allocation (Gurney et al. 1996; Nisbet et al. 2004). The rate of net production per unit of biomass equals the difference between the energy intake rate and the loss rate due to maintenance, \( \sigma j(R) - m \), whereas gross production equals \( \sigma j(R) \). Under net-production allocation, it is assumed that a fraction \( \kappa \) of the net production rate is allocated to somatic growth, and the fraction \( 1 - \kappa \) is used for reproduction. For the net-production allocation model, the rates of biomass production through somatic growth (\( g \)) and reproduction (\( b \)) then equal

\[
\begin{align*}
g(R) &= \sigma j(R) \kappa - m \\
b(R) &= \sigma j(R) (1 - \kappa)
\end{align*}
\]

For this model, it is necessary to specify what happens in case of starvation. In the MPA model specified below, it is possible that biomass flow increases local competition to such an extent that net production becomes negative in one of the areas. In this case, we assume that the birth rate \( b \) becomes zero, and the somatic growth rate \( g \) becomes equal to the (negative) net production rate. This amounts to assuming that in case of starvation, standing biomass is converted to cover maintenance.

Under gross-production allocation, it is assumed that a fraction \( \kappa \) of the gross production rate is used for both maintenance and somatic growth, and the fraction \( 1 - \kappa \) is allocated to reproduction, which is also referred to as the “kappa rule” model (Kooijman 2000). So for the gross-production allocation model, the rates of biomass production through somatic growth (\( g \)) and reproduction (\( b \)) are

\[
\begin{align*}
g(R) &= \sigma j(R) \kappa - m \\
b(R) &= \sigma j(R) (1 - \kappa)
\end{align*}
\]

Note that a special rule for starvation is unnecessary in the gross-allocation model: in case of starvation, the birth rate is still positive, and standing biomass is used to cover maintenance. Although a special rule for starvation may be more realistic for any particular fish species, we choose to use this simple assumption.

An important difference between the net- and gross-production models is hence their behaviour at low food density: in the former model (eq. 5), reproduction and somatic growth stop simultaneously at a critical low food density, whereas in the latter model (eq. 5), growth stops but reproduction continues.

Note that for a spatially undivided population, the two models result in exactly the same total biomass dynamics, as all that matters is the sum of \( b(R) \) and \( g(R) \) (eq. 1). For both allocation models, we then obtain eq. 3.

We point out that in the gross-production allocation model, maintenance is equivalent to a local rate of biomass loss, similar to a mortality rate. At the population level, maintenance can hence be incorporated into a general loss term \( \delta = m + \delta \). MPA models that do not explicitly consider bioenergetics simply redistribute the population-level reproduction rate \( B = b(R)C \) over protected and harvested areas, without discounting the maintenance costs. This amounts to the implicit assumption that maintenance costs are either absent or incorporated in the mortality loss term. In other words, ignoring bioenergetics corresponds to assuming implicitly gross-production allocation. Note also that assuming the absence of maintenance costs (\( m = 0 \)) is equivalent to the gross-production model.

**Spatial structure: marine reserve and fished area**

Next we subdivide the consumer–resource system into a marine reserve and a harvested area. Adults (i.e., standing biomass) suffer a per-unit biomass harvesting mortality in the fished area, denoted by \( H \). The protected area is assumed to cover a fraction \( x \) of the total area \( A \). The fraction \( \alpha \) of produced consumer larvae is assumed to migrate and to be equally redistributed between the areas. Adults migrate with rate \( \beta \). Note that larval migration amounts to a redistribution of the birth rate, whereas adult migration amounts to a redistribution of standing stock biomass. The so-called larval pool equal redistribution (LPER) assumption is equivalent to setting \( \alpha = 1 \) and \( \beta = 0 \).

Variables and population-level rates pertaining to the protected and the harvested areas are denoted by indices 1 and 2, respectively. For example, the population-level birth rates are now defined as \( B_1 = b(R_1)C_1 \) and \( B_2 = b(R_2)C_2 \), respectively. Because all variables are expressed as densities per unit of sea surface area, the total biomass in the protected and fished areas equals \( C_1A\alpha x \) and \( C_2A(1 - x) \), respectively.

**Biomass flow between areas: source and sink**

The dynamics in the two areas depend on the biomass flow of larval and adult biomass. Assuming that from both areas, a fraction \( \alpha \) of produced larvae enters a common dispersal pool, the rate of larval settlement in each area equals the sum of local larval production, \( (1 - \alpha)B_2 \), and the inflow of larvae from the dispersal pool, \( \alpha x B_1 + (1 - x)B_2 \). This corresponds to the widely used LPER assumption. The larval settlement rate in the protected area is then, after simplification,

\[
S_1 = B_1 - \alpha(1 - x)(B_1 - B_2)
\]

For the fished area, the equivalent rate is

\[
S_2 = B_2 + \alpha x(B_1 - B_2)
\]

These equations show that the larval settlement rate exceeds the local birth rate in the area with the lowest local birth
rate. In other words, the net larval flow is always from the most productive to the least productive area. The net larval flow from the reserve to the harvested area (denoted by $f_L$) equals the difference between inflow and outflow in the harvested area, multiplied by the size of the harvested area:

$$f_L = \{a[xB_1 + (1-x)B_2] - aB_2\}(1-x)A = \alpha x(1-x)A(B_1 - B_2)$$

The flow is positive, i.e., from the reserve to the harvested area, if and only if the reserve is more productive (per unit of sea area) than the fished area ($B_1 > B_2$).

Similar to larval migration, adults are assumed to migrate with rate $\beta$ and to enter a common dispersal pool, which is then equally redistributed. Although this way to model adult migration is less standard than for larvae, the resulting equations is completely analogous to the most commonly used diffusive model.

We obtain expressions similar to larval migration. For example, in the reserve, the rate of departing biomass equals $\beta(C_1)$ and the rate of incoming biomass equals $\beta(x(C_1 - C_2))$. The migration balance thus equals $-\beta(1-x)(C_1 - C_2)$ in the reserve and $\beta(x(C_1 - C_2))$ in the harvested area. The net biomass flow due to adult migration is again found by multiplication of either of these by the corresponding surface area:

$$f_A = \beta x(1-x)A(C_1 - C_2)$$

The flow is positive, i.e., spillover from the reserve to the harvested area, if and only if the biomass density is highest in the reserve ($C_1 > C_2$).

Note that the direction of the larval flow depends on differences in the biomass production rate, whereas the direction of adult flow depends on differences in the biomass standing stock. The two flows do not necessarily point in the same direction. The net total biomass flow equals their sum $f_{tot} = f_L + f_A$.

The full MPA model

Extending eqs. 1–2 to two areas and substituting the migration terms, we obtain

$$\frac{dC_1}{dt} = G_1 + B_1 - a(1-x)(B_1 - B_2) - \beta(1-x)(C_1 - C_2) - C_1\delta$$

$$\frac{dC_2}{dt} = G_2 + B_2 + a\alpha(B_1 - B_2) + \beta\alpha(C_1 - C_2) - C_2(\delta + H)$$

$$\frac{dR_1}{dt} = \rho(R_{max} - R_1) - j(R_1)C_1$$

$$\frac{dR_2}{dt} = \rho(R_{max} - R_2) - j(R_2)C_2$$

The dynamics of the fish populations $(C_1$ and $C_2)$ are the sum of four terms: local growth of standing biomass, larval production and redistribution, adult migration, and mortality. Note that we assume that the food base $(R_1$ and $R_2)$ does not migrate.

Parameter values

The default parameter values of the consumer–resource model are based on the equivalent but stage-structured model of de Roos et al. (2007): $j_{max} = 6$, $m = 1$, $\sigma = 0.7$, $\rho = 0.5$, $R_0 = 1$. The natural mortality rate is assumed to be $\delta = 0.1$, which represents a typical value on a per-year basis for marine fish populations (Andersen and Ursin 1977). We have, however, tested the model behaviour over a range of $\delta$ up to 1. We obtain qualitatively similar results in all cases, although increasing $\delta$ decreases standing biomass and adult flow and makes the larval flow less negative. In our simulations presented below, we vary the harvesting rate $H$. We note, however, that rough estimates of harvesting mortality fall in the range of 2–10 times the natural mortality rate (Pope et al. 2006). With a mortality rate of $\delta = 0.1$, appropriate harvesting rates ($H$) thus fall in the range of 0.2 to 1. The larval migration parameter $\alpha$ is varied between 0 and 1 (note that $\alpha$ is a proportion). The adult migration parameter $\beta$ should be interpreted relative to the mortality parameter: on average, an unharvested individual is expected to migrate $\beta\delta$ times during its life time. We vary $\beta$ between 0 and 1.

Results

Consequences of choice of allocation model

To understand how the MPA system reacts to harvesting mortality, it is instructive to first analyse the how the equilibrium of the single-area system (eqs. 1–2) without dispersal depends on the level of mortality, $\delta$. Recall that for a single population, the allocation into growth and reproduction is irrelevant, as it is their sum that appears in eq. 1.

To simplify the analysis, we initially assume a linear functional response, which does not qualitatively influence the results (see below). For the single-area system (eqs. 1–2), the equilibrium equals

$$\bar{C} = \frac{\rho(\sigma aR_{max} - m - \delta)}{a(m + \delta)}$$

$$\bar{R} = \frac{m + \delta}{\sigma a}$$

These equations show that $\bar{C}$ decreases and $\bar{R}$ increases with mortality, because $\partial C/\partial \delta < 0$ and $\partial R/\partial \delta > 0$. The fish population goes extinct when mortality exceeds the value

$$\delta_{ext} = \sigma aR_{max} - m$$

The equilibrium (eqs. 15–16) is the same for the two allocation rules (eqs. 5–6). However, this is not true for the steady-state total reproduction rate, denoted by $\bar{B}$. With net-production allocation,

$$\bar{B} = \delta(1 - \kappa)\bar{C}$$

with $\bar{C}$ as defined in eq. 15, whereas with gross-production allocation,

$$\bar{B} = (m + \delta)(1 - \kappa)\bar{C}$$

An important difference between the allocation rules is the resulting relation between the total reproduction rate and (mass-specific) mortality. By substituting eq. 15 into eq. 19,
we find that with gross-production allocation, the reproduction rate necessarily decreases with mortality, i.e., \( \partial \hat{B}/\partial \delta < 0 \). Yet with net-production allocation, the curve of \( \hat{B} \) over mortality is dome-shaped; it first increases between \( \delta = 0 \) and a critical mortality rate \( \delta_{\text{crit}} \), after which it decreases to zero at \( \delta_{\text{ext}} \). The critical mortality rate is as follows:

\[
(20) \quad \delta_{\text{crit}} = -m + \sqrt{\alpha a R_{\text{max}} m}
\]

This equation shows that with \( m > 0 \), the population-level birth rate can increase with mortality, even though the equilibrium biomass \( \hat{C} \) always decreases with mortality. We refer to such a positive relation between total reproduction and mortality as “overcompensation”.

The explanation of the overcompensation lies in (i) the resource dependence of the reproduction rate and (ii) the balancing of the birth and mortality rates in equilibrium. Increasing mortality decreases competition for the resource, which increases the reproduction rate per unit of biomass. Yet the total (population) reproduction rate may still decrease if the standing biomass decreases faster than the fecundity increases. This is the case when reproduction is proportional to the intake rate and hence to the resource density, as in the gross-production model. However, when reproduction is proportional to the net-production rate, by contrast, fecundity can increase much faster than the resource density itself. This becomes obvious in the limit of zero mortality. The equilibrium condition \( d\hat{C}/dt = 0 \) requires that net production balances the death rate, i.e., \( \alpha f(\hat{R}) - m = \delta \), which means that for \( \delta = 0 \), the net-production rate is zero. This, in turn, means that the total reproduction rate \( \hat{B} \) is zero for \( \delta = 0 \). \( \hat{B} \) then necessarily increases with \( \delta \).

In summary, overcompensation is never found in the gross-production model, whereas it occurs in the net-production model whenever the background mortality is relatively low (\( \delta < \delta_{\text{eq}} \)). This result is true also for a type-II functional response, but the equation of \( \delta_{\text{crit}} \) is very ugly in that case. A graph of \( \delta_{\text{crit}} \) for a type-II functional response, plotted against \( m \) (Fig. 1), shows that overcompensation is likely to occur with plausible parameter values; across a range of taxa, the mortality rate appears to be of the order of 10% of the maintenance rate (de Roos et al. 2007). Figure 1 illustrates that for this estimation, most combinations of \( \delta \) and \( m \) fall in the region of overcompensation. Very similar results are obtained when varying the parameters \( R_{\text{max}} \), \( a \), and \( R_{h} \) (data not shown).

**Implications for the MPA model**

The analysis of the single-area model gives an idea of how the MPA model will behave. As a simple thought experiment, compare the two areas of the MPA model, without any migration and with a low harvesting rate. The analysis tells us that the standing biomass will be lower in the harvested area, for either allocation model. Assuming gross-production allocation, we also know that the total birth rate will be lower in the harvested area. So we expect that allowing either larval migration between the two areas or adult migration will result in a biomass flow from the reserve into the harvested area (i.e., \( f_{L} > 0 \) and \( f_{A} > 0 \)).

Next, consider the net-allocation model. Because of the overcompensation, the total birth rate is then likely to be higher in the harvested area than in the MPA. Larval migration is hence likely to result in a biomass flow from the harvested area into the reserve (\( f_{L} < 0 \)). With net-production allocation, we therefore expect the MPA to be a biomass sink for larvae.

However, these expectations should be checked with analysis of the full MPA model, as the steady state of the full model with migration does not necessarily conform to our intuition based on the limiting case of no migration.

**The MPA model: source–sink status of the reserve**

Here we study the dynamics of the full MPA system (eqs. 11–14) assuming a type-II functional response. We compute the steady state of the MPA model over a range of the harvesting rate \( H \) (similar to Fig. 2), with the MPA being 30% of the total area (\( x = 0.3 \)).

Model analysis confirms the intuition on the gross-production allocation model (Fig. 3). Assuming larval migration only (\( \alpha = 1, \beta = 0 \)), the standing biomass decreases with \( H \) in both the protected and harvested areas, but is consistently higher in the protected area (\( C_{1} > C_{2} \)). The same is true for the population birth rates (i.e., undercompensation and \( B_{1} > B_{2} \)), despite increasing resource density in both areas. The resulting biomass flow is positive; the MPA is a source of larval production. Note also that the MPA prevents the harvested population from going extinct at high harvesting rates (cf. Fig. 2).

Model analysis also confirms the intuition on the net-allocation model (Fig. 4). The population birth rate in the harvested area displays the typical pattern of overcompensation (cf. Fig. 2). By contrast, the birth rate in the protected area decreases and is zero over a considerable range of \( H \). In this range, the consumers in the MPA are
starving due to severe resource competition. Throughout the range where $B_1 < B_2$, the larval flow is negative (Fig. 4). The reserve is thus a larval sink for harvesting rates up to the point at which the harvested population would go extinct in the absence of an MPA (cf. Fig. 2). Beyond this point, the fished stock is practically extinct, and the MPA becomes a source of larvae, saving the harvested population from true extinction.

In the marine reserve, the biomass initially increases with $H$, mirroring the pattern of larval production in the harvested area (Fig. 4). Although the resource in the exploited area increases gradually with $H$, converging to its carrying capacity at very high $H$, in the reserve, the resource first decreases with $H$, showing that competition for food intensifies in the MPA. In fact, the strong inflow of larvae causes an unsustainably high biomass density in the MPA: severe resource competition leads to starvation, most notably resulting in the complete absence of reproduction inside the MPA.

The idea is thus that overcompensation in the harvested area causes the MPA to be a larval sink. We test this idea by drawing the curve for which the larval flow is zero (Fig. 1), which delimits the region in which the MPA is a sink (i.e., below the broken or dotted curves). The figure shows that with decreasing $H$, the sink region approximates the region of overcompensation, thus confirming the idea. Note that with increasing $H$, the sink region becomes smaller, which reflects the fact that the sink status of the MPA depends on $H$ and is obtained for small and intermediate $H$ only (Fig. 4).

The effect of adult migration

Allowing adults to migrate ($\beta > 0$) results in an additional biomass flow, which is always directed from the MPA to the harvested area, because $C_1 > C_2$ (cf. eq. 10). Here we study the effect of adult migration on the larval flow ($f_l$) and total flow ($f_{tot}$).

Consider the example with $\alpha = 1$ and $\beta = 0.5$ (Fig. 5). (Note that $\beta/\delta = 5$, meaning that unharvested adults migrate on average five times in a life time.) The flow of standing biomass changes the pattern of consumer biomass in the reserve; $C_1$ now decreases monotonically with $H$. The adult flow releases the resource competition in the MPA, allowing for reproduction in that area. Initially, however, reproduction is still higher in the harvested area ($B_1 < B_2$), resulting in negative larval flow up to $H \approx 3$. In this range, the MPA is hence a sink for larvae, but a source of adults. Overall, the MPA is a source of biomass ($f_{tot} > 0$).

Next we study the effect of adult migration on the source–sink status of the MPA systematically (Fig. 6a). Figure 6 depicts the regions in $H, \beta$-parameter space where the MPA is a sink of total biomass and (or) larvae. These zones are delimited by the curves of $f_l = 0$ and $f_{tot} = 0$. The curve of $f_l = 0$ is almost vertical, indicating that the rate of adult migration has little influence on the sign of the larval flow. The MPA is a larval sink even for very high values of $\beta$. By contrast, the total biomass flow depends strongly on $\beta$: increasing adult migration reduces the range of $H$ with negative total flow. The region marked “S” shows that starvation in the MPA, and hence zero reproduction, is restricted to low adult migration.

The effect of larval migration

The effect of changing the proportion of dispersing larvae ($\alpha$) mirrors the effect of changing adult migration. For fixed adult migration ($\beta = 0.1$), reducing $\alpha$ has no influence on the sign of the larval flow (Fig. 6b). As expected, the total biomass flow becomes positive when larval migration becomes very small. Starvation is limited to very high larval migration.

The effect of the size of the MPA

The relative size of the reserve ($x$) determines the relative contribution of the two areas to the biomass flows; increasing $x$ hence decreases the potential effect of overcompensation, which occurs in the harvested area only, on the larval flow. Changing $x$ may influence the direction of the larval flow ($f_l$); if the MPA is larger, the larval flow is negative.
over a wider range of $H$ (Fig. 7). At high $H$, small reserve size accentuates the “source” potential of marine reserves, as most of the locally produced larvae are lost to migration, thus reducing resource competition in the reserve and increasing the birth rate $\sim B_1$. However, at low and intermediate $H$, when overcompensation is strong in the harvested area (Fig. 4), small reserve size accentuates the “sink” potential of an MPA. With small $x$, the larval flow is largely determined by the harvested area such that overcompensation has a large impact on the dynamics in the MPA. By consequence, the starvation range is limited to small MPA size (Fig. 7).

Changing $x$ does not, however, influence the sign of the total biomass flow (Fig. 7). This “inertia” is independent of the values of the other parameters (data not shown). The inertia can be understood as follows: when the total biomass flow is zero, each unit of sea area has a zero net flow. Given that the biomass inflow (larvae + adults) is the same for each unit of sea surface area, the biomass outflow per unit area (larvae + adults) must be the same in the MPA and the harvested area. Changing the relative contribution ($x$) to the common dispersal pool then has no effect whatsoever.

**Alternative models**

**Size-structured population model**

The allocation models studied in this paper were originally designed for size-structured population models (Gurney et al. 1996). In size-structured population models, density dependence may affect not only fecundity, settlement, and survival, but also maturation and individual growth. To illustrate the consequences of size structure, we analyse a well-studied size-structured population model in the context of our findings above.

The gross-production allocation model (eq. 6) first appeared as the kappa rule in a size-structured population model (Kooijman and Metz 1984). The Kooijman–Metz model has since been studied in various forms, and here we use the model published by de Roos and Persson (2002), but without predators. We extend their model by including an MPA and larval dispersal (analogous to eqs. (11–14) with $a = 1$ and $b = 0$). In this model, individuals are born with size $b$ and mature at size $j$. The gross-production model results in von Bertalanffy growth with asymptotic size $\frac{mR}{F(R_\infty + R)}$. A model description (without MPA) can be found in de Roos and Persson (2002).

We do not present a full analysis of the size-structured model, but merely illustrate the implications of a juvenile life stage. In the size-structured model (without MPA), the consumer and resource biomass (Fig. 8a) follow the same pattern as in the unstructured model (cf. Fig. 2). Surprisingly, however, we find overcompensation (Fig. 8b), despite the gross-production allocation model. Based on the overcompensation, we thus expect a negative biomass flow (from the harvested area to the MPA) in the case of larval migration. Including an MPA in the size-structured model (with $x = 0.3$) confirms this expectation (Fig. 8c).
Which difference between the unstructured and structured models explains the found overcompensation? An important difference between unstructured and size-structured models is that the latter allows for different kinds of population regulation, depending on which demographic process (juvenile growth-maturation or adult reproduction) is most affected by intraspecific competition and hence limiting population growth. If a population is maturation-limited (as in Figs. 8a–8b), then increasing the mortality rate releases the competition among juveniles, and the adult biomass increases due to increased maturation (de Roos et al. 2007). In this case, we thus observe an overcompensation of the adult standing biomass: adult biomass increases with mortality (Fig. 8a, thin solid line), resulting in an overcompensation of the population-level reproduction rate (Fig. 8b). To demonstrate the crucial role of the juvenile life stage, we have rerun the model with \( \beta < \beta_N \), such that individuals are born mature (Figs. 8d–8f). In this case, the population is necessarily reproduction-limited. Without a juvenile stage, the size-structured model predicts undercompensation (Fig. 8e). The corresponding MPA model displays a positive biomass flow (Fig. 8e), as expected from the undercompensation.

This simple example illustrates that maturation limitation in structured populations may lead to overcompensation in both adult biomass and the population reproduction rate, as shown by de Roos et al. (2007). The examples demonstrate that (i) overcompensation is not restricted to net-production allocation models and (ii) overcompensation in a model without MPA implies a negative larval biomass flow in the corresponding MPA model, whatever mechanism is causing the overcompensation. The size-structured model thus suggests that the association of overcompensation and negative biomass flow is general.

**Logistic MPA model**

Now that we have identified the role of overcompensation, we can check that it works in the simplest model that could produce such a result, the logistic growth model:

\[
\frac{dN}{dr} = rN \left(1 - \frac{N}{K}\right)
\]

To be able to use the death rate as an independent parameter and to use the total birth rate as a model output, we have to decompose eq. 21 by making assumptions about how it reflects underlying per capita birth and death rates. As in the previous two models, we assume that density dependence affects the birth rate, whereas the death rate remains constant. By choosing \( b - \beta N \) for the per capita birth rate and \( d \) for the death rate, we obtain

\[
\frac{dN}{dr} = N(b - \beta N - d)
\]

which is equivalent to eq. 21 with \( r = b - d \) and \( K = (b - d)/\beta \). The equilibrium population of eq. 22 equals \( \hat{N} = (b - d)/\beta \). The population-level birth rate, defined as \( \dot{B} = \hat{N}(b - \beta N) \), thus equals...
Because $\bar{B}$ goes to zero when $d$ goes to zero, the logistic growth model clearly displays overcompensation for any value of $b$ and $\beta$. Based on this observation, we expect that an MPA model that connects two logistic equations with larval dispersal will predict that the MPA is a larval sink. To check this prediction, we have extended eq. 22 by including a second harvested population and assuming LPER. We have computed its steady state and the corresponding direction of the larval biomass flow. The results are analogous to the ones obtained with the consumer-resource model above and confirm that the logistic model indeed predicts the MPA to be a larval sink for $d$ below a critical background mortality rate (data not shown).

If, however, we assume that density dependence does not influence the birth rate but the death rate (by choosing $d + \beta N$ for the death rate and $b$ for the birth rate), we obtain $\bar{B} = b(b - d)/\beta$, which corresponds to undercompensation. Thus, overcompensation requires that density dependence affects the birth rate.

**Discussion**

Our main result is that the source–sink status of marine reserves depends on the effect of the per capita mortality rate, including harvesting mortality, on the population-level reproduction rate ($B$). If this relation displays overcompensation (i.e., $B$ initially increases with mortality), then any degree of larval migration results in a net biomass flow of larvae from the harvested area into the MPA, and the MPA is thus a sink of larval biomass. In the case of undercompensation, by contrast, the MPA is a source of larval biomass.

Why do MPAs become larval sinks? The explanation resides in intraspecific competition. Competition is more severe inside the MPA due to the reduced mortality rate (e.g., see the lower resource density $R_1$ in Fig. 3). When increased competition reduces the population-level reproductive output (i.e., overcompensation of $B$), then total reproduction is higher outside the MPA than inside. By consequence, the net larval flow will be towards the MPA. This, however, is not true for populations that display undercompensation of $B$ (such as the gross-production model (eq. 6)).

For migration of standing biomass (as opposed to larval migration), our results suggest that marine reserves are usually a source of biomass, if it is assumed (as done here) that net migration goes from high-biomass to low-biomass areas. Larval and adult flow may thus be in opposite directions, and the net effect obviously depends on the relative rates of larval and adult migration. It should be noted, however, that in size-structured populations, adult biomass may itself display overcompensation, i.e., increase with mortality (de Roos et al. 2007). This may hence cause an MPA to be a sink of adult fish.

The main work presented here concerns the effect of alternative bioenergetic models on the source–sink status of MPAs. The conclusion is that gross-production allocation results in undercompensation of the population reproduction.
rate (and hence the MPA being a larval source), whereas net-production allocation is likely to result in overcompensation (and hence the MPA being a larval sink). However, our complementary analyses of a size-structured model, in which overcompensation is caused by a juvenile growth and maturation despite gross-production allocation, and a logistic model show that the connection between overcompensation and the larval-sink status of MPAs is more general and not limited to net-production models. In other words, overcompensation of the total reproduction rate causes the MPA to be a larval sink, whatever underlying mechanism causes the overcompensation.

Our main result depends on the combination of two factors: (i) overcompensation and (ii) larval dispersal. When both factors are present, the MPA is predicted to be a larval sink. Our study of three models shows that different mechanisms can underly the first of these factors. Common between all of them is a strong, density-dependent response of the population reproduction rate with increasing mortality. This response is stronger (at high population density) for net-production allocation than for gross-production allocation, explaining the different results for these two types of models. The logistic MPA model shows that overcompensation is found only when the logistic density dependence affects the birth rate (instead of the death rate). In the size-structured model, the birth rate depends not only on the fecundity (determined by gross-production allocation), but also on the age at maturation and survival to maturation, which are both density-dependent themselves. By consequence, the presence of a juvenile life stage accentuates the density dependence in the birth rate to such an extend that it results in overcompensation, despite gross-production allocation.

With respect to the second factor (larval dispersal), our results stress the need to distinguish between migration of newborns and of older individuals. Whereas the former amounts to a redistribution of biomass production, the latter amounts to a redistribution of biomass standing stock. Our main result depends critically on this distinction; only the redistribution of biomass production can result in the MPA being a sink, because the production can be higher in the harvested area than in the MPA. By contrast, standing biomass is generally higher in the MPA than in the harvested area (Halpern and Warner 2002; Micheli et al. 2004b) and therefore is likely to induce spillover into harvested areas. Although the distinction between larval and adult migration is common in MPA models, its implications for the source–sink status of marine reserves has not been identified before.

Here we note that the strictly negative relation between standing (adult) biomass and mortality found for unstructured models (eq. 15) does not necessarily apply to size-structured populations. Size-structured populations can be limited by different demographic processes, depending on which one of them is most affected by intraspecific compe-
When a population is regulated by juvenile growth and maturation, then increasing mortality tends to increase the adult standing biomass. Although such overcompensation of adult biomass may in turn cause overcompensation of the population reproduction rate, in itself, it may cause a net migration of adults into the MPA. Hence, for maturation-limited populations, we predict that MPAs are likely to be general sinks of biomass, irrespective of the rates of larval and adult migration.

The review of Gerber et al. (2003) shows that previous models have often assumed either migration of larvae only (for example, models assuming LPER) or of adults only. Our work shows that it is in the case of LPER that overcompensation has the largest potential to cause MPAs to be biomass sinks. This result has not previously been found for the simple reason that the models used do not allow for factors (i) and (ii) to occur simultaneously. Many models in the literature do not explicitly take into account maintenance costs when computing the birth rate and hence implicitly correspond to the gross-production model (Neubert 2003; Baskett et al. 2006).

For example, the Lotka–Volterra competition models of Baskett et al. (2007) have the potential to display overcompensation, due to their equivalence to the logistic growth
model (see subsection Logistic MPA model). However, they assume that diffusion migration, i.e., migration of standing stock, results in a flow from high to low density and hence that the MPA is a source. The same is true for the models of Neubert (2003) and Steele and Beet (2003), which are equally based on the logistic growth model. The trophic models of Baskett (2006, 2007) and Baskett et al. (2006, 2007) also assume diffusion migration and hence do not need to distinguish between net- or gross-production allocation. However, we argue that in all of these models, larval flow is likely to be from the MPA to the harvested areas if larval dispersal and, in the trophic model, net-production allocation were assumed.

A second important result of our model is that the relative size of an MPA does not affect the sign of the total biomass flow between the MPA and surrounding areas. How can we understand this? This is a consequence of our assumption of a well-mixed, common dispersal pool. This assumption implies that for each unit of sea surface, the net biomass flow reflects the difference in local production of migrants and the global average production of migrants. Changing the MPA size ($x$) changes the relative contribution of areas with high and low migrant production and thus influences the average production of migrants. For example, if reserve size $x$ is small and the reserve has a lower migrant production than the fished area, then increasing $x$ will reduce the global average migrant production and hence reduce the influx of migrants into the MPA. Changing $x$ thus has a quantitative effect on steady-state biomass flux (and standing biomass). This is not true, however, for the special case of zero net biomass flow. In this case, each unit of sea surface produces exactly the same amount of migrants. The local production and the global average are hence the same, irrespective of the size of the MPA. Changing the reserve size $x$ has absolutely no effect on biomass flow or on the standing stock biomass.

Increasing reserve size is hence not likely to qualitatively improve the functioning of MPAs (in terms of the source-sink status of the MPA). Figure 7 illustrates that in the range of realistic harvesting rates (i.e., $H \approx 0.2$ to $1$), the MPA is a biomass sink for both larvae and adults, no matter the size of the reserve. Only for heavily overexploited fisheries ($H > 2$) does the MPA function as a biomass source.

All of the models that we have discussed are continuous-time models. But what about discrete-time models? Many fisheries models are formulated in discrete time, based on the Ricker or Beverton–Holt models, so the question seems relevant. There is no reason to presume that for discrete-time models, the relation between overcompensation and the source-sink status of MPAs should not hold, in combination with the redistribution of larvae. For example, in the discrete time, predator–prey model of Micheli et al. (2004a), it is assumed that larvae enter a common dispersal pool. We have simulated their model, simplified by excluding the MPA, and found that the steady-state value of the variable $L$ (equivalent to our $B$), increases with fishing mortality for high values of the parameter $a$. Based on this observation, our results thus predict that in this model, the MPA can be a larval sink. Micheli et al. (2004a) did not, however, compute the direction of the larval flow.

More generally, for discrete-time models, whenever the recruitment rate displays overcompensation, larval redistribution is expected to result in larval flow from the harvested area to the MPA. This should be the case of models based on the Ricker model, which is the classical example of overcompensation, in contrast to the Beverton–Holt model, which displays undercompensation. However, the actual dynamics will depend on how the density dependence in such models is assumed to depend on the underlying vital rates (birth, death, growth, maturation).

**Acknowledgments**

D.C. and A.M.dR. acknowledge financial support from the Netherlands Organisation for Scientific Research (NWO). The manuscript has benefitted greatly from constructive reviews from Marc Mangel and Roger Nisbet.

**References**


