INDIVIDUALS ON THE MOVE

BODY CONDITION DEPENDENT DISPERsal
AND
QUASI-LOCAL COMPETITION
IN METAPOPULATIONS

Margarete Utz

Department of Mathematics and Statistics
Faculty of Science
University of Helsinki
Finland

Academic Dissertation

To be presented,
by permission of the Faculty of Science of the University of Helsinki,
for public examination in Auditorium CK112 in Exactum
on November 25, 2010 at 12 o’clock noon.

Helsinki 2010
Author’s present address:

Ludwig-Maximilians-University Munich
Department Biology II
Evolutionary Ecology
Großhaderner Str. 2
82152 Martinsried-Planegg
Germany

Email: utz@bio.lmu.de

Cover design by Georg Utz

ISBN 978-952-10-6669-6 (Paperback)

http://ethesis.helsinki.fi

Yliopistopaino
Helsinki 2010
Author
Margarete Utz
Department of Mathematics and Statistics
University of Helsinki
Finland

Supervisors
Professor Mats Gyllenberg
Department of Mathematics and Statistics
University of Helsinki
Finland

Dr Éva Kisdi
Department of Mathematics and Statistics
University of Helsinki
Finland

Reviewers
Professor Otso Ovaskainen
Department of Biosciences
University of Helsinki
Finland

Dr Andrew White
Department of Mathematics
and the Maxwell Institute for Mathematical Sciences
Heriot-Watt University, Edinburgh
United Kingdom

Opponent
Professor Yoh Iwasa
Department of Biology
Kyushu University, Fukuoka
Japan
List of Publications

This thesis is based on the following articles. They are referred to by their Roman numerals.


II Mats Gyllenberg, Éva Kisdi and Margarete Utz. *Variability within families and the evolution of body condition dependent dispersal.* (accepted for publication in a special issue devoted to Adaptive Dynamics of the Journal of Biological Dynamics)

III Mats Gyllenberg, Éva Kisdi and Margarete Utz. *Body condition dependent dispersal in a heterogeneous environment.* (submitted manuscript)


Author’s contribution

According to the tradition in mathematical literature, the authors are listed alphabetically in articles I, II and III. I had the leading role in the development and analysis of the models and in writing the manuscripts of all articles.
Abstract

Individual movement is very versatile and inevitable in ecology. In this thesis, I investigate two kinds of movement — body condition dependent dispersal and small-range foraging movements resulting in quasi-local competition — and their causes and consequences on the individual, population and metapopulation level.

Body condition dependent dispersal is a widely evident but barely understood phenomenon. In nature, diverse relationships between body condition and dispersal are observed. I develop the first models that study the evolution of dispersal strategies that depend on individual body condition. In a patchy environment where patches differ in environmental conditions, individuals born in rich (e.g. nutritious) patches are on average stronger than their conspecifics that are born in poorer patches. Body condition (strength) determines competitive ability such that stronger individuals win competition with higher probability than weak individuals. Individuals compete for patches such that kin competition selects for dispersal. I determine the evolutionarily stable strategy (ESS) for different ecological scenarios. My models offer explanations for both dispersal of strong individuals and dispersal of weak individuals. Moreover, I find that within-family dispersal behaviour is not always reflected on the population level. This supports the fact that no consistent pattern is detected in data on body condition dependent dispersal. It also encourages the refining of empirical investigations.

Quasi-local competition defines interactions between adjacent populations where one population negatively affects the growth of the other population. I model a metapopulation in a homogeneous environment where adults of different subpopulations compete for resources by spending part of their foraging time in the neighbouring patches, while their juveniles only feed on the resource in their natal patch. I show that spatial patterns (different population densities in the patches) are stable only if one age class depletes the resource very much but mainly the other age group depends on it.
# Contents

1 Individual Movement  

2 Body Condition Dependent Dispersal  
  2.1 Empirical Data  
  2.2 Prior Models  
  2.3 Model Assumptions  
  2.4 Methods and Analysis  
  2.5 Results  

3 Quasi-Local Competition and Pattern Formation  
  3.1 Motivation  
  3.2 The Model  

4 Further Perspectives  

Acknowledgements  

References  

Article I  
Evolution of Condition-Dependent Dispersal under Kin Competition  

Article II  
Variability within Families  
and the Evolution of Body Condition Dependent Dispersal  

Article III  
Body Condition Dependent Dispersal in a Heterogeneous Environment  

Article IV  
Quasi-Local Competition in Stage-Structured Metapopulations:  
A New Mechanism of Pattern Formation
Species and individual movement is one of the most important processes in nature. On a daily basis, individuals need to move around in order to forage, avoid predators, defend their territories, or care for their offspring. These frequent movements occur on a relatively small spatial scale and are necessary for daily survival of individuals. They are mostly observed in motile animals, whereas plants, which are mainly immobile, have different mechanisms to carry out these performances (e.g., through root growth towards higher nutrient concentration).

At much larger scales, species undertake migration journeys. Often, groups of animals migrate together over large distances from one seasonal habitat to another. Extreme examples are migratory birds that fly between winter and summer habitats twice a year, and salmon that swim from their natal river into the sea and return to their place of birth years later when mature to spawn; both involve travels over thousands of miles.

Dispersal characterizes a movement that lies spatially and temporally in between small-scale daily activities and large-scale migrations. It is the movement away from an existing population or from the natal habitat. Natal dispersal, i.e., emigration of juveniles from their parents’ habitat in order to find a territory of their own, is prevalent in most animal and plant species. Breeding dispersal occurs between breeding events and serves to encounter mating partners and find suitable nesting sites.

All movements are population dynamically, genetically and ecologically important and are governed by complex interactions of many individual and environmental factors. The huge body of both empirical and theoretical literature that deals with different aspects and consequences of movement reflects the significance of these processes.

In this thesis, I investigate two aspects of individual movement: body condition dependent dispersal and quasi-local competition due to small-scale foraging movements.

**Body Condition Dependent Dispersal.** The focus of this thesis is on body condition dependent dispersal (articles I, II and III). Empirical data display diverse relationships between individual body condition and dispersal between as well as within species. Inspired by this dissonance, I address questions like ‘If body condition determines competitive ability, should stronger or weaker individuals disperse more?’, ‘If body conditions of siblings vary, which individuals from a family should disperse?’ or ‘How does the natal environment influence dispersal behaviour?’ I develop a metapopulation model where dispersal propensity is a function of individual body condition. Within the framework of Adaptive Dynamics, I find evolutionarily stable dispersal strategies for different population dynamical and environmental assumptions. My findings shed some light on the puzzling phenomenon of body condition dependent dispersal from a theoretical point of view. The general set-up of the model as well as the examples include both mechanisms that yield dispersal of strong individuals and that lead to dispersal of weak individuals. The most striking and biologically most relevant results I find when investigating the model under the assumption that body conditions vary within families. Then the evolu-
tionarily stable strategy (ESS) can be an equivalence class that includes infinitely many selectively neutral dispersal strategies that have the same fitness. This may explain why, for example, two populations of the same species that live in similar habitats show seemingly contradictory dispersal behaviour. Furthermore, if the ESS is no equivalence class of strategies but gives a clear rule for individual dispersal behaviour depending on the individual’s and its siblings’ body conditions, then this underlying rule can often not be detected in the population-wide behaviour. The population-wide distribution of body conditions among dispersers often has a shape close to the body condition distribution of all individuals in the whole population. Further empirical research may therefore benefit from more detailed studies of individual within-family dispersal behaviour.

**Quasi-Local Competition.** In article **IV**, I investigate how daily foraging movement that goes beyond the scope of the own population — resulting in so-called quasi-local competition — influences local population sizes in a metapopulation. What determines the presence and abundance of a species is one of the most important ecological questions. Not necessarily is a species evenly distributed over suitable habitat but patterns of different densities are observed. The model in **IV** emanates from two recent apparently similar metapopulation models but that exhibit contradicting dynamics. With quasi-local competition as the only interaction between populations, pattern formation occurs in one model but not in the other. I explicitly model the resource–consumer dynamics where the consumer population has two active age stages, juveniles and adults. Both feed on the resource in their home patch, but adults also forage in the neighbouring patches. I show that spatial patterns of different densities in the patches are stable only when one age class depletes the resource very much but mainly the other class depends on it.
2 Body Condition Dependent Dispersal

2.1 Empirical Data

Many empirical studies find that dispersers are not a random subset of the population but that they differ in body condition from philopatrics. Variable relationships between body condition and dispersal propensity are observed across species. For instance, in [47], dispersing males of the red-cockaded woodpecker (Picoides borealis) are on average lower in body mass than their philopatric brothers. In several rodent species, dispersers are found to be smaller or lighter [17, 23, 28]. The authors of these studies explain their findings to some extent by the social dominance hypothesis, which was introduced by Fretwell [18] as the ‘ideal despotic distribution’. It states that socially dominant and more aggressive individuals exclude (socially) weaker individuals from resources or mates and expel them from the local territory. But there are also examples of weak dispersers that are not explained by social interactions (e.g., in [24, 29], individuals of poor phenotypic condition and smaller body size disperse in a bird and an insect species).

At the same time, dispersal of strong individuals is very common in many species. For instance, roe deer (Capreolus capreolus) dispersers are found to be heavier on average than philopatrics in [62], larger individuals of the common lizard (Lacerta vivipara) disperse more in [34], heavier damselflies (Enallagma boreale) disperse in [1], and in [55], larger and heavier females containing more fat and glycogen preferentially disperse, and male dispersal correlates positively to larger size and higher levels of glycogen in the ant species Formica truncorum.

Yet, there are examples where no relationship between body condition and dispersal behaviour is found (e.g. in roe deer Capreolus capreolus in [61], in the side-blotched lizard Uta stansburiana in [15], in prairie voles Microtus ochrogaster in [38], and in the collared flycatcher Ficedula albicollis in [45]).

Note that some of these studies look at the same species (e.g. [61] and [62]) or species of the same taxonomic family (e.g. [15] and [34]) but find different relationships between body condition and dispersal.

While most animals actively disperse and also actively decide whether to disperse, in plants, dispersal is a passive process that often uses vectors such as wind, water or animals. Depending on the dispersal mechanism, the influence of body condition (e.g. seed size or weight) can be observed as well. In the common wood sorrel (Oxalis acetosella), explosive seed dispersal leads to a significant positive relationship between seed weight and dispersal distance in [4]. Among the wind-dispersed seeds of the rain forest tree Lophopetalum wightianum, lighter seeds reach larger distances in [51].

Intraspecific plasticity in dispersal is poorly understood in general and especially concerning the influence of body condition [3, 7, 43, 47, 49]. Strong individuals that are more competitive or more aggressive could easily retain their natal territory. So why do they often take on the risk of dying during dispersal when they could instead kick out the weak individuals? On the other hand, although the social dominance hypothesis attempts to explain dispersal of weak, it assumes no or limited kinship between individuals and
can thus only limited be applied to natal dispersal. However, a big part of all dispersal is natal dispersal (almost all above cited articles study natal dispersal). The phenomenon of body condition dependent dispersal remains thus puzzling and requires more research, both empirical and theoretical.

2.2 Prior Models

In fact, there has been no theoretical investigation of body condition dependent dispersal until recently. To the best of my knowledge, my models in I – III are the first that describe dispersal as a function of individual body conditions. I model natal dispersal that is driven by offspring competition for sites. Individual body condition is determined by the environmental condition of the natal patch, and competitive ability increases with body condition.

Hamilton and May [27] set up the first model for dispersal under kin competition. It is a simple deterministic metapopulation model for an annual semelparous species. Patches can be occupied by at most one individual each (i.e. after reproduction and before competition, many offspring live in a patch, but only one establishes itself through competition). Patches are connected through global dispersal. In each patch, a fair lottery chooses one offspring individual that establishes itself and becomes the new adult in the next year. With $\Pi$ being the probability to survive dispersal, the evolutionarily stable dispersal probability is

$$\hat{p} = \frac{1}{2 - \Pi},$$

which is always greater than one half. Even if dispersal almost surely leads to death ($\Pi \to 0$), half of the offspring disperse. But the fact that also almost all philopatrics die during competition (as every patch can sustain only one individual) selects for dispersal. Because dispersers are distributed over all patches, hence, families avoid kin competition through dispersal.

Among the many extensions and generalizations of the Hamilton–May model, the following two come closest to the models in this thesis and a comparison is worthwhile. Ezoe and Iwasa [16] and Kisdi [31] both introduced variation of offspring number between sites. Offspring number indicates the quality (e.g. resource level) of the local site. Moreover, having in mind plants or insects that produce dimorphic seeds or offspring [14, 67], in [16] a mother can decide how many dispersers and non-dispersers she produces. Other model assumptions are the same as in the Hamilton–May model. Both models predict threshold dispersal strategies for the ESS: a fixed number of non-dispersers stays in the patch and the rest disperse (if the total number of offspring is less than the threshold then all stay in the patch).

Recently, Bonte and de la Peña [5] simulated the evolution of body condition dependent dispersal in metapopulations. They related body condition to fecundity and investigated natal and adult dispersal under several scenarios that varied e.g. in environmental stochasticity or dispersal mortality. They found that dispersal probability increases with environmental stochasticity and decreasing dispersal mortality. With low environmental
stochasticity, their model yields a negative relationship between body condition and dispersal probability that shifts towards a strongly positive relationship when environmental stochasticity increases and dispersal mortality decreases.

2.3 Model Assumptions

The models in I – III are based on the Hamilton–May model [27]. Individual body condition is introduced via varying patch qualities that affect body conditions of local offspring. The evolving trait is the dispersal probability as a function of body condition such that the whole function is under selection.

Environmental Assumptions. The model area is a heterogeneous environment divided into small patches that may differ in several ecological and population dynamical respects. The number of patches tends to infinity. Every patch is labelled by a vector $y = (y_1, \ldots, y_n)^T \in Y \subseteq \mathbb{R}^n$ that contains all patch characteristics where $Y$ is the set of all feasible $y$. The probability density of patches $y$ is $\phi(y)$ such that $\int_Y \phi(y) \, dy = 1$. In this thesis, patches always differ at least in mean body condition of offspring produced in the patch. In I and II, this is the only variation between patches such that there is a one-dimensional continuum of patch qualities $y \in \mathbb{R}$ with average patch quality zero. In III, the general analysis of the model is carried out for $n$-dimensional $y$, but in all numerical examples $Y$ is reduced to $\mathbb{R}$. It is straightforward to extend also the general development and analysis of the model in II to vector-valued $y$; this would not change the results developed in II. However, in I, all offspring born in a patch have the same body condition as the natal patch quality such that the offspring body condition distribution in a patch is a point mass concentrated at the patch quality (a delta peak). This special property equates patch quality with offspring body condition such that it eliminates one of the two variables from the formulas and hence simplifies the analysis essentially. The general results in I (e.g. a property of the slope of the dispersal probability $p$) could not be derived with vector-valued $y$.

I analysed the model under two contrasting assumptions concerning the heterogeneity of the environment. Naturally, environmental conditions fluctuate spatially and temporally. In all models in this thesis, spatial structure is ignored. Temporal fluctuations are random in I and II (fluctuating environment), but in III, patch conditions do not change in time (fixed environment). These are two extremes on the scale where reality lies somewhere in between. However, because any scenario with temporal autocorrelation between zero and one would be very difficult to model, I restricted myself to these two cases. Conclusions for real cases can be drawn from the results of these two extreme cases.

Every patch can sustain at most one individual. The life cycle of the model species during one year is depicted in Figure 1.

Survival. In the beginning of a year, every patch is inhabited by one juvenile individual. An individual in a patch $y$ survives until maturation with probability $s(y)$. In I and II, $s$
is simply a constant. In the model in III, patches differ in safety and $s(y)$ is a non-constant function of $y$. For instance, the presence of predators, the availability of hiding places, or the resource abundance in a patch can determine the shape of $s$.

**Reproduction.** An individual that survives until maturation has on average $B$ offspring. Because there are infinitely many patches, $B$ will tend to infinity so that the resulting model is deterministic. Offspring have different body conditions $z \in Z \subseteq \mathbb{R}$ that depend on the environmental condition of the patch. $Z$ is the set of body conditions. $\beta(z, y)$ is the probability density of offspring with body condition $z$ that are born in a patch $y$. In I and II, where $y \in \mathbb{R}$, $y$ determines the mean of $\beta$, and, specifically in I, $\beta(z, y) = \delta(z - y)$ (where $\delta$ is the Dirac delta function) such that all offspring in a patch have the same body condition as the patch quality (see above). Patches are simply referred to as being 'good' patches (where the mean offspring body condition is positive) and 'bad' patches (where offspring have on average a negative body condition). However, in III, the notation is refined to ‘rich’ and ‘poor’ patches, with regard to the fact that patches with much and nutritious resource yield stronger offspring on average. Body condition is directly related to competitive ability such that ‘strong’ individuals have a positive value of $z$ and win competition with a high probability, and ‘weak’ individuals have negative...
2.3 Model Assumptions

$z$ and are competitively less successful. I do not take maternal effects into consideration, i.e., the body condition of an offspring individual depends solely on the environmental quality of the natal patch and not e.g. on nurturing impacts by the mother (for empirical evidence that natal habitat quality affects body condition and dispersal, see e.g. [3, 7]).

The species is semelparous, i.e., if an individual survives until maturation, it dies immediately after reproduction.

**Dispersal.** Offspring compete for patches and a family is interested in occupying as many patches as possible in the next year. Thus, part of the offspring disperse and the rest stay to defend the natal patch. $p(z, y)$ denotes the probability that an offspring individual with body condition $z$ that is born in a patch $y$ disperses. Dispersal is costly, and $\Pi(z)$ denotes the probability that a disperser with body condition $z$ survives dispersal. For this survival probability $\Pi$ I have mainly assumed a constant or monotonically increasing or decreasing function (except the general analysis in article I was performed for an arbitrary function $\Pi$). The assumption of constant $\Pi$ neglects performance differences during dispersal of different individuals. With increasing $\Pi$, stronger individuals are assumed to survive dispersal better than their weak conspecifics, e.g. because they have advantages during starving periods or fights that occur during dispersal [59]. Decreasing $\Pi$ models the assumption that weak, e.g. small and light, individuals have a higher probability to survive dispersal, e.g. if they are more agile and can thus easier escape predators [33, 66]. Philopatrics do not pay any cost. Patches are connected through global dispersal such that every patch receives an equal sample of immigrants. The number of immigrants with body condition in a short interval $dz$ is $B u_i(z) dz$ where

$$u_i(z) = \int_Y \phi(y) s(y) \beta(z, y) p(z, y) \Pi(z) dy.$$  \hspace{1cm} (2)

In article I, with $\beta(z, y) = \delta(z - y)$ and constant $s$, this simplifies to formula (2) therein. In articles II and III, with exclusively weighted lottery competition, I used instead the weighted sum of immigrants,

$$BI = B \int_Z g(z) u_i(z) dz.$$ \hspace{1cm} (3)

(formulas (6) in II and (2) in III) where $g$ is a weight function (see below).

Dispersal probability $p(z, y)$ is the evolving trait, and I aimed to find functions $\hat{p}$ that are evolutionarily stable. I do not make any assumptions about the function $p$, but all properties of the evolutionarily stable strategy derive from underlying population dynamical and environmental assumptions.

**Competition.** After dispersal, in each patch, local non-dispersers (if present) and immigrants compete and only one individual establishes itself in the patch whereas all others die. There are two types of patches, 1) with and 2) without local non-dispersers, depending on whether the individual inhabiting the patch in the beginning of the year survived
until reproduction or not. Competitive ability depends on body condition such that stronger individuals are better competitors. Competition is mostly modelled by weighted lottery, which is the most common way to model asymmetric competition [6, 22]. But article I develops the general model framework for arbitrary competition and includes numerical examples for weighted lottery and other competition mechanisms. In a weighted lottery, every individual is assigned a weight $g(z)$ according to its body condition, where the function $g$ increases with body condition.

In a patch $y$ of type 1 where local non-dispersers and immigrants compete with one another, the number of local non-dispersers with body condition in a small interval $dz$ is

$$B u_n(z, y, p) dz$$

where

$$u_n(z, y, p) = \beta(z, y) (1 - p(z, y)).$$

(Without specified competition mechanism, $B(u_n(z, y, p) + u_i(z)) dz$ is the number of all competitors with body condition $dz$ in a patch $y$ of type 1, see formula (i) in I for the special case that $\beta(z, y) = \delta(z - y)$.) With weighted lottery, the weighted sum of non-dispersers in a patch $y$ is

$$B n(p, y) = B \int_Z g(z) u_n(z, y, p) dz.$$  

(5)

In a patch $y$ of type 1, a focal individual with body condition $z$ thus wins competition and establishes itself with probability $P_1(z, y, p)$, which assumes the form

$$P_1(z, y, p) = \frac{g(z)}{B(n(p, y) + I)}$$

(6)

in a weighted lottery (see formulas (13) in I, (7) in II and (3) in III).

In type 2 patches, only immigrants compete, and the probability that a focal individual with body condition $z$ wins competition is

$$P_2(z) = \frac{g(z)}{BI}$$

(7)

with weighted lottery competition (formulas (14) in I, (8) in II and (4) in III). For the formulas for other competition scenarios, the reader is referred to Sections 3.2 - 3.4 in article I.

With global dispersal, the probability that a disperser with body condition $z$ that has survived dispersal wins a patch is then

$$R(z) = \int_Y \phi(y) \left[ s(y) P_1(z, y, p) + (1 - s(y)) P_2(z) \right] dy$$

(8)

(formulas (4) in I and (9) in II; in III this quantity appears in formula (7) but is not explicitly denoted by $R$).
2.4 Methods and Analysis

**Adaptive Dynamics.** The models in I – III investigate the evolution of the probability of dispersal \( p(z, y) \) as a function of individual body condition \( z \) and patch quality \( y \). The mathematical theory that has been developed specifically for studying the evolution of phenotypic traits is Adaptive Dynamics (Geritz et al. [21], Metz et al. [39]). It links short-term population dynamics to long-term evolution by explicitly respecting the different time scales of these two processes. One models first the dynamics on the population level that take place on a short time scale, say one year or one life cycle, and involve the focal trait. A basic assumption of Adaptive Dynamics is that only after the population has reached its dynamical attractor, evolution is addressed. Evolution is considered as successive events of mutation and establishment of a mutated trait and is modelled by introducing a rare mutant who differs in the focal trait from the established population (the resident). Only one mutation can occur at a time, and a new mutation does not occur before the previous mutated trait has either been established in the population or has vanished. Here, another essential assumption of Adaptive Dynamics is that an initially rare mutant does not affect the population dynamics of the resident. Whether a mutant can establish itself in the population depends on whether its invasion fitness is greater than the fitness of the resident. From the mutant’s initial invasion fitness, it is then assumed that one can infer the eventual fate (establishment or doom) of the mutant.

The resident populations in I – III are instantaneously at dynamical equilibrium. Because offspring body conditions in a patch depend only on the environmental condition of the patch and not on the body condition of the parent individual, at the time of reproduction, the probability density of offspring with a certain body condition born in patches of certain quality is the same every year.

Dispersal probability as the evolving trait is denoted by \( p \) for the resident and by \( p_m \) for the mutant. Resident and mutant do not differ in any other respect. The invasion fitness of a mutant depends on the number of patches its offspring can occupy in the future, but how exactly this invasion fitness is expressed depends on the properties of the environment (see below).

The number of patches and the number of offspring per individual tend simultaneously to infinity. Taking these limits eliminates stochasticity and the resulting models are deterministic and analytically easier to treat than the corresponding stochastic models. If \( N \) is the number of patches and \( M \) is the number of mutants in the whole population, then \( M/N \) is the number of mutants per patch. After dispersal, the number of mutant immigrants in a patch follows a binomial distribution, \( \text{Bin}(\int Y \int Z M \phi(y) s(y) B \beta(z, y) p_m(z, y) \Pi(z) dz dy, 1/N) \). When \( N \to \infty, M \to \infty \) and \( B \to \infty \) such that \( M/N \to 0 \) and \( BM/N \to 0 \), then the binomial distribution converges towards a distribution where most of the probability mass is concentrated at zero. The probability that two or more mutants immigrate into the same patch can then be neglected, and it is assumed that mutant immigrants compete only against resident offspring in patches that were inhabited by a resident individual in the beginning of the year.
Invasion Fitness in a Temporally Fluctuating Environment. In a fluctuating environment as in I and II where patch qualities are randomly reassigned every year, the qualities of all patches and hence the body conditions of the offspring in the next year are unknown. The reproductive value of each established individual is thus the same, and the number of mutant offspring that establish themselves after dispersal and competition can simply be counted. With the quantities derived in the previous Section, the mutant fitness is then

\[ W(p_m) = \int_Y \int_Z \phi(y) s(y) B \beta(z, y) \left[ p_m(z, y) \Pi(z) R(z) + (1 - p_m(z, y)) P_1(z, y, p_m) \right] dz \, dy. \]  

(9)

It is a functional of the mutant strategy \( p_m \), where the resident strategy \( p \) is included in \( R(z) \) (via \( n \) and \( I \) in \( P_1 \) and \( P_2 \)) and in \( P_1(z, y, p_m) \) (via \( I \)). The square bracket in formula (9) is the probability that one mutant offspring with body condition \( z \) that is born in a patch \( y \) and disperses with probability \( p_m(z, y) \) will win either a patch as a disperser (first summand) or retain the home patch (second summand). Multiplying this quantity with \( B \beta(z, y) \) gives the expected number of patches all mutants with body condition \( z \) born in a patch \( y \) can win. Because a mutation can happen in any patch, the average over \( y \) is taken. For generality, the survival probability until maturity, \( s(y) \), is denoted as a function of patch quality, although in I and II, \( s \) is always constant. (In I respectively II, \( W \) is found in formula (5) respectively formulas (1) and (2); for notational differences between these formulas and formula (9) above, refer to the derivations in the previous Section.)

Basic Reproduction Number in a Fixed Environment. In a fixed environment as in III where patch qualities stay constant in time, a family knows what quality their natal patch will have in the future and thus what body conditions their descendants will have that will be born in this patch if the family can retain the patch. Therefore, the invasion fitness of the mutant is not an annual quantity as in a fluctuating environment, but takes into account all descendants as long as the family can retain the patch. The probability that an established individual has offspring and one of the non-dispersing offspring wins competition and thereby the family retains the natal patch \( y \) is

\[ s(y) \int_Z B \beta(z, y) (1 - p_m(z, y)) P_1(z, y, p_m) \, dz. \]

(10)

The number of years a patch \( y \) can be retained by a descendant of the local family follows thus a geometric distribution with expectation

\[ T(y, n(p, y)) = \frac{1}{1 - s(y) \frac{B_n(p, y)}{B(n(p, y) + I)}}. \]

(11)
(formula (6) in III), which can conveniently be referred to as the expected lifetime of a family in a patch $y$.

According to Gyllenberg and Metz [26] and Metz and Gyllenberg [40], fitness in structured metapopulations is formulated via the next generation operator $W$ that defines one ‘generation’ as the lifetime of a local entity (e.g. a local population or the ‘population’ of dispersers (the dispersal pool)). The next generation operator gives the number of new local entities of a certain state that are produced by a collection of local entities of a certain state during their entire lives. In the present model for fixed environment, the weighted sum of mutants that have dispersed, survived dispersal and are at the point of leaving the dispersal pool, $\int_Z M(z) g(z) dz$, gives such a local entity. The next generation operator maps then the present weight of immigrating mutants onto the weighted sum $\int_Z M'(z) g(z) dz$ of their descendants that disperse and survive dispersal during the lifetime $T(y, n(p_m, y))$ of their families,

$$\int_Z M'(z) g(z) dz = W \left( \int_Z M(z) g(z) dz \right)$$

$$= R_0(p_m) \int_Z M(z) g(z) dz \quad (12)$$

where

$$R_0(p_m) = \int_Y \phi(y) \left( \frac{s(y)}{B(n(p, y) + I)} + \frac{1 - s(y)}{BI} \right) s(y) \cdot$$

$$\cdot \left( B \int_Z \beta(z, y) g(z) p_m(z, y) \Pi(z) dz \right) T(y, n(p_m, y)) dy \quad (13)$$

is the spectral radius of the next generation operator $W$, i.e., the basic reproduction number of the gross mutant competitive weight. A mutant disperser immigrates into a patch $y$ with probability $\phi(y)$; the term within the first set of parentheses in (13) is the probability that the mutant immigrant wins competition there; the mutant survives until maturation with probability $s(y)$; if it survives then the weighted sum of its offspring that again disperse and survive dispersal is the expression in the second set of parentheses; this cycle is repeated during the entire lifetime of the mutant family ($T(y, n(p_m, y))$).

In the case that patch qualities fluctuate with autocorrelation coefficient between zero and one, the fitness measure of a mutant would complicate very much such that the model would be essentially more difficult to analyze.

**Evolutionarily Stable Strategy.** The concept of evolutionarily stable strategies was introduced by Maynard Smith and Price [37]. They defined an evolutionarily stable strategy (ESS) as a strategy $\hat{p}$ that satisfies one of the following conditions,

1. $E(\hat{p}, \hat{p}) > E(p, \hat{p})$, \hspace{2cm} (14)
2. $E(\hat{p}, \hat{p}) = E(p, \hat{p})$ and $E(\hat{p}, \hat{p}) > E(p, p)$ \hspace{2cm} (15)


for all \( p \neq \hat{p} \), where \( E(p, q) \) denotes the payoff (or, in an ecological context, the fitness) of playing strategy \( p \) against strategy \( q \). An ESS, if played by the whole population, is thus a strategy that cannot be invaded by any other (mutant) strategy. In the ecological models in I and II, \( E \) corresponds to \( W \), in III it is \( R_0 \). Because the resident population is in equilibrium, \( W(p) = 1 \) or \( R_0(p) = 1 \). If the resident is at the ESS then \( W(p_m) \leq 1 \) or \( R_0(p_m) \leq 1 \) for all \( p_m \neq p \). A mutant’s best reply strategy \( \hat{p}_m \) to a given resident strategy \( p \) is one that maximizes \( W(p_m) \) or \( R_0(p_m) \). An ESS is a strategy \( \hat{p} \) such that \( \hat{p}_m = \hat{p} \), i.e., a strategy that is the best reply to itself. At an ESS evolution comes to a halt as no other strategies can invade any more.

In the classic framework of Adaptive Dynamics, only small mutation steps are allowed, and an ESS is only locally uninvadable but invasion by traits that are sufficiently different from the ESS is not considered. (This is, the above ESS conditions need to be valid only for strategies \( q \) within a small neighbourhood of a given strategy \( p \).) The assumption of small mutation steps is realistic when studying the evolution of genetically inherited phenotypical traits. However, in behavioural traits such as the probability of dispersal in the models in this thesis, mutations of any size can realistically be taken into account. In the model species as in many natural species, every individual has in principle the ability to disperse and can make use of this option or not. The analysis of the models does not need the assumption of small mutation steps. However, it does not always assure that there is a unique ESS, i.e., in some cases there may exist several strategies such that each is the best reply to itself. Because finding the ESS for any specific example involved numerical calculations (see below), I was content with finding one ESS in each example. In some cases it is though possible to prove analytically that the ESS is unique.

Adaptive Dynamics for Function-Valued Traits. The theory of Adaptive Dynamics is far developed for scalar-valued and vector-valued traits (in addition to the above cited introductory references, see e.g. [10, 11, 20, 22]). However, function-valued traits have not been considered very much so far. For the first time, Adaptive Dynamics of function-valued traits was formalized by Dieckmann et al. [12] and Parvinen et al. [46]. They displayed the calculus of variations as an appropriate tool to find evolutionarily stable strategies for function-valued traits.

**Calculus of Variations.** The calculus of variations generally finds a function \( \hat{p} \) that yields an extremal (a maximum or a minimum) of the functional

\[
J(p) = \int_{z_{\text{min}}}^{z_{\text{max}}} F(z, p(z), p'(z))dz
\]  

and satisfies the boundary conditions

\[
\hat{p}(z_{\text{min}}) = Z_{\text{min}}, \quad \hat{p}(z_{\text{max}}) = Z_{\text{max}}.
\]  

It can thus be used to find the mutant’s best reply strategy, which maximizes the fitness for the mutant, to a given resident strategy. The function \( F \) is assumed to be differentiable with respect to \( p \) and \( p' \), and the function \( p \) is assumed to be piecewise smooth. A
necessary condition for the functional $J$ to have an extremal at $\hat{p}$ is that Euler’s equation is satisfied,

$$\frac{d}{dz} \frac{\partial}{\partial p} F(z, \hat{p}(z), \hat{p}'(z)) = \frac{\partial}{\partial p} F(z, \hat{p}(z), \hat{p}'(z)) \quad \text{for all } z \in (z_{\min}, z_{\max}). \quad (18)$$

For a maximum, a sufficient condition is that $F$ is concave with respect to $p$ and $p'$,

$$\frac{\partial^2}{\partial p^2} F(z, \hat{p}(z), \hat{p}'(z)) + 2 \frac{\partial^2}{\partial p \partial p'} F(z, \hat{p}(z), \hat{p}'(z)) + \frac{\partial^2}{\partial p'^2} F(z, \hat{p}(z), \hat{p}'(z)) < 0. \quad (19)$$

In the models in I – III, the quantities that are to maximize (the invasion fitness $W$ or the basic reproduction number $R_0$ of the mutant) are functionals of the dispersal probability $p$. Although $p$ is generally a function of two variables $z$ and $y$, the functionals are only maximized with respect to $z$ for fixed $y$.

The derivative of the strategy, $p'$, is not involved in the models. Therefore, Euler’s equation simplifies to

$$0 = \frac{\partial}{\partial p(z)} F(z, \hat{p}(z)) \quad \text{for all } z \in (z_{\min}, z_{\max}) \quad (20)$$

(Euler’s equation for the specific models is given in eqs (9) in I, (16) in II and (28) in III.) The condition for a maximum reduces to the first term on the left hand side of the inequality in (19).

The ecological framework does neither request boundary conditions for the strategy $p$ to satisfy. In general, if boundary values are free, Euler’s boundary conditions have to be satisfied,

$$\frac{\partial}{\partial p'(z)} F(z_{\min}, \hat{p}(z_{\min}), \hat{p}'(z_{\min})) = 0, \quad (21)$$

$$\frac{\partial}{\partial p'(z)} F(z_{\max}, \hat{p}(z_{\max}), \hat{p}'(z_{\max})) = 0. \quad (22)$$

Because of the independence of $W$ or $R_0$ on $p'$, these conditions are automatically satisfied.

Although ecological realism demands finite boundaries of the integration interval $[z_{\min}, z_{\max}]$, the models in I – III are generally derived for infinite boundaries. Because the number of patches as well as the number of offspring per individual tend to infinity, the set of body conditions $Z = \mathbb{R}$, but is restricted to a finite interval only in the numerical examples. The general derivation of the conditions for maximals of functionals with infinite integration boundaries is analogous to the theory for functionals with finite integration boundaries. (For a textbook on the calculus of variations see e.g. [19, 60].)

Maximizing the functional $W$ or $R_0$ by the calculus of variations does generally not yield an explicit solution for the ESS dispersal probability $\hat{p}$. Nevertheless, the general
properties of the ESS can be derived analytically in all models. Because the ESS is the 
best reply to itself, every property that a mutant’s best reply strategy possesses is also 
inherent in the ESS itself. In I, for given competition mechanism, Euler’s equation can 
be solved for the mutant’s best reply strategy \( \hat{p}_m \) to a given resident strategy. The ESS can 
then be found numerically through a best reply series: I first assumed a given strategy for 
the resident and calculated the mutant’s best reply to this strategy. Then, I assumed this 
reply strategy as the new resident strategy and calculated the best reply again, et cetera, 
until the resident and mutant strategy coincide.

In II and III, with weighted lottery competition, the shape of the survival proba-
bility during dispersal, II, essentially characterizes the ESS. If II is constant, then there 
exist equivalence classes of dispersal strategies that are determined by the non-dispersing 
weight in a patch. This feature derives from the fact that, with constant II, the whole 
model dynamics can be written in terms of the non-dispersing weight such that the actual 
dispersal strategies \( p \) and \( p_m \) drop from the formulas. For instance, the second set of 
parentheses in (13) simplifies to \( B \Pi (A(y) - n_m(y)) \) where \( A(y) = \int_{Z} \beta(z,y)g(z)dz \) is the 
total competitive weight produced in a patch and \( n_m(y) \) denotes the mutant’s non-
dispersing weight in a patch \( y \) (cp. formula (5); see also formulas (21) and (22) in II 
and (11) – (13) in III). In II, the mutant’s best reply non-dispersing weight to a given resident 
strategy is

\[
\hat{n}_m = -I + \sqrt{\frac{I}{BHR}},
\]

(formula (24) in II), where \( \hat{R} \) is \( R(z)/g(z) \) which does not depend on \( z \). \( \hat{n}_m \) is constant 
and thus the mutant’s best reply (and hence the ESS, too) in II is to keep this constant 
weight in every patch and disperse the excess weight (unless the total competitive weight 
produced in a patch is less than this threshold weight, then all offspring stay in the patch). 
In order to find the ESS non-dispersing weight, resident and mutant strategy have to be 
equalled and equation (23) has to be solved for \( \hat{n} = n = n_m \). Because \( I \) and \( \hat{R} \) are both 
integrals that depend on \( n \) or \( n_m \), I solved equation (23) numerically, by calculating the 
best reply dynamics as mentioned above. In III, the non-dispersing weight in a patch 
depends on patch quality (formula (14) in III). Likewise, from the expression for the 
mutant’s best reply strategy, the ESS can be found numerically.

If II is a non-constant function of body condition then Euler’s equation cannot be 
satisfied for any open interval of \( z \) values if competition is modelled by weighted lottery 
as in II and III. With weighted lottery, the weight function \( g(z) \) cancels from the formu-
as and II is the only function that depends on \( z \). Hence in Euler’s equation (e.g. eq. (16) 
in II), one side depends on \( z \) but the other one does not. This implies that \( W \) or \( R_0 \) is 
maximized at the boundary. Because the dispersal strategy \( p \) is a probability it hence must 
be a step function with respect to \( z \) that assumes values zero and one only. I proved that, 
with monotone II, the ESS \( \hat{p} \) has at most one jump upwards respectively downwards if 
II is increasing respectively decreasing (formulas (44) in II and (29) in III). The equation 
that has to be satisfied at the position of the jump \( \hat{z} \) is an implicit expression for \( \hat{z} \) that 
has to be solved numerically (formulas (45) in II and (32) in III).
2.5 Results

Numerical Analysis. To illustrate the general structure of the ESS that was found analytically, I calculated numerically the actual dispersal probability $\hat{p}$ or the non-dispersing weight $\hat{n}$ for several examples. In almost all examples, the numerical analysis amounted to calculating a best reply series, which always converged. However, this need not be the case, e.g. if the ESS is a repellor of the population dynamics.

For all numerical calculations, I assumed that patch qualities are ordered along a one-dimensional continuum and that they follow a standard normal distribution, $\phi(y) = \mathcal{N}(0, 1)$. If necessary for numerical reasons, I truncated $\phi$ at three standard deviations. Similarly, body condition distribution (if no delta peak) is a (truncated) normal distribution with patch quality as mean, $\beta(z, y) = \mathcal{N}(y, 1)$. For the functions $s$ and $\Pi$, I assumed a sigmoid shape with inflection point at zero (the mean patch quality and mean body condition in the whole population) of the form $h(x) = h_1 + \frac{h_2}{1 + e^{-h_3x}}$ (cf. formulas (42) and (43) in II and (21), (34) and (35) in III). The weight function was assumed to be $g(z) = e^z$.

2.5 Results

Evolutionarily Stable Strategy in a Weighted Lottery. A special property of competition by weighted lottery is the simple exchange between number and competitive weight. A few strong individuals are as competitive as a large number of weak individuals. Therefore the competitive weight plays an essential role in the models.

Fixed versus Fluctuating Environment. The model species behaves fundamentally differently in a fixed and in a temporally fluctuating environment. In a fixed environment, a family knows that its natal patch quality will be the same in the future. It decides upon patch productivity (the total competitive weight $A$ produced) and patch safety (the probability $s$ that an established individual survives until maturation) whether it is worth to try to retain the patch. Patches that have too low productivity or are too unsafe are abandoned such that from these patches all offspring disperse. Families in other patches keep part of their offspring in the patch. Figure 2 (a) shows the ESS non-dispersing weight $\hat{n}$ as a function of patch quality (thick line) for the case that all patches are equally safe ($s = 0.5$) and all individuals are equally good dispersers ($\Pi = 0.5$). The thin dashed line gives the total competitive weight $A$ produced in a patch. The ESS $\hat{n}$ is zero in very poor patches (small values of $y$) and increases with patch quality in richer patches.

A family that tries to retain the natal patch keeps as much weight in the patch as is beneficial. The ESS balances the fitness benefit from keeping more weight at home and the benefit from dispersing more weight. The non-dispersing weight increases with patch quality in Figure 2 (a) because productivity increases with patch quality whereas all patches are equally safe.

If patches decrease in safety while they increase in productivity such that $s(y)$ is a decreasing function of patch quality but $A(y)$ is an increasing function, then both very poor patches (low value of $A$) and very unsafe patches (low value of $s$) are abandoned and $\hat{n}$ is a non-monotone function of body condition. Figure 3 shows such a case.
Contrarily, in a randomly fluctuating environment, the future quality of all patches is unknown and hence all patches are equally valuable to retain. Consequently, every family keeps a certain weight in the patch and disperses offspring only if the benefit from dispersing some offspring is higher than the benefit from keeping more weight in the patch. If all offspring are equally good dispersers (constant $\Pi$), then every family keeps the same weight in the patch and disperses the excess weight (except in patches where the total competitive weight is less than this threshold weight, then all offspring stay in the patch). Figure 2 (b) shows $\hat{n}$ for the case with $s = 0.5$ and $\Pi = 0.5$.

**Monotone versus Constant Survival During Dispersal.** How the non-dispersing weight in a patch is composed, i.e., which individuals of a family disperse, depends on the shape of the function $\Pi$, the probability to survive dispersal. In both environments, if $\Pi$ is a monotone function of body condition then, from each family, these individuals disperse who have the highest survival probabilities. In each patch, the dispersal probability $\hat{p}$ is a step function with respect to body condition $z$ that assumes only values zero and one and has at most one jump from zero to one (from one to zero) if $\Pi$ is increasing (decreasing). From patches with low productivity, all offspring disperse in a fixed environment and hence $\hat{p}(z, y) = 1$ for all $z$, but no offspring disperses in a fluctuating environment and hence $\hat{p}(z, y) = 0$ for all $z$.

If the probability to survive dispersal does not depend on the body condition of the disperser, then the ESS is characterized entirely by the non-dispersing weight in a patch rather than by an actual dispersal strategy. Dispersal strategies form equivalence classes that are determined by the same non-dispersing weight. Within an equivalence class, there are infinitely many strategies that are selectively neutral. The ESS is the equivalence class of strategies that yield the ESS non-dispersing weight $\hat{n}$. This involves that very diverse strategies can be equally evolutionarily stable and that it is not possible to predict...
any relationship between body condition and dispersal behaviour, neither for different families within a metapopulation nor for different populations of the same species that inhabit similar habitats. This is consistent with empirical data that do not find a clear dependence of dispersal on body condition.

**Within-Family versus Population-Wide Dispersal Behaviour.** The existence of equivalence classes of dispersal strategies if body condition does not influence survival during dispersal is one explanation why it can be very difficult to detect an underlying dispersal rule from empirical data. (The underlying rule in the case of equivalence classes is that a certain weight stays in a patch, but families can decide arbitrarily how they implement this rule.) Another explanation for this difficulty emerges when studying the body condition distribution of dispersers and non-dispersers on the population level. Figure 4 displays the body condition distribution $d$ of all dispersers in the population at the time of leaving the patches (left panels), and the body condition distribution $f$ of non-dispersers (middle panels) for the four possible combinations of fixed and fluctuating environment and increasing and decreasing survival probability during dispersal, $\Pi$. In each case, patches are equally safe; $s = 0.5$ with fixed environment, and $s = 0.9$ with fluctuating environment. The right panels in Figure 4 give the expected dispersal probability $E(\hat{p}(z, y)|z)$ of individuals as a function of body condition, averaged over the whole population.

In both environments, with increasing $\Pi$, the strongest offspring from each family disperse, and with decreasing $\Pi$, the weakest individuals disperse. However, this behaviour is not always reflected on the population level. In cases (a) and (d), both $d$ and $f$ are rather symmetric and their means (indicated by the vertical line in the plots) are near zero, which suggests no relationship between body condition and dispersal despite the clear underlying rule in each family. In case (a), the strongest from families in rich and intermediate patches disperse (due to increasing $\Pi$), but the weakest in the whole popu-
lation abandon their poor patches (due to the fixed environment). Because the strongest and the weakest in the whole population are rare, intermediate body conditions dominate among the dispersers. In case (d), the weakest from families in rich and intermediate patches disperse (due to decreasing $\Pi$), but the weakest in the whole population do not disperse because offspring born in the poorest patches all stay in their patches (due to the fluctuating environment).

In the other two cases (b) and (c), population-wide behaviour resembles within-family behaviour: Decreasing $\Pi$ in a fixed environment (case (b)) yields dispersal of the weakest in each family (because they are better dispersers than their stronger siblings) and in the population as a whole (because they abandon their very poor patches). $f$ as well as $E(\hat{p}(z,y)|z)$ reveals that all weak and intermediately strong individuals always disperse but the strongest never disperse. In case (c), dispersal probability is practically a step function of body condition and both $d$ and $f$ have a clearly positive respectively negative mean, as the strongest within a family and in the population as a whole always disperse (due to increasing $\Pi$) and the weakest never do (due to the fluctuating environment).

Measuring the population-wide body condition distribution among dispersers and non-dispersers is often the first endeavour in the field. However, Figure 4 suggests that this may not suffice to detect the actual dispersal rule exercised by the individuals. Within-family behaviour need not be reflected on the population level. If no apparent relationship between body condition and dispersal is found from $d$ or $f$, it is advisable to look into individual patches and study which individuals from a family disperse. Although patch quality may be very difficult to measure, it could be determined through the body conditions of the inhabiting individuals. Then, comparing families in different patches with each other may for instance disclose if very poor patches are abandoned or if families in richer patches disperse more offspring than families in poorer patches.

**Lottery Competition versus Strongest Wins Scenario.** The general model derivation in II and III assumes a weighted lottery competition. This assumption permits that a dispersal strategy is identified by the competitive weight that stays in the patch. Other competition mechanisms lack this simplifying but also constraining feature. The model in I is for arbitrary competition in a fluctuating environment for the special case that all offspring in a patch have the same body condition as the patch quality. All numerical examples in I assume constant survival during dispersal. Among the examples, the first assumes a weighted lottery and is hence a special case of the model in II; at the ESS, every family keeps a fixed weight in the patch and disperses the rest, except if the total competitive weight in a patch is less than this threshold weight, then all offspring stay in the patch (Figure 1 in I). The models by Ezoe and Iwasa [16] and Kisdi [31] also exhibit this feature: In their models, patches differ in productivity via the number of offspring the local individual can produce. Dispersal at the ESS depends on the number of offspring such that families with more offspring disperse more, but every family keeps a fixed number of offspring in the patch.

In two examples in I, the strongest competitor in a patch wins; because with infinitely many offspring, there is no strongest individual, the number of competitors is first re-
2.5 Results

(a) Fixed environment and increasing $\Pi$

![Graph a]

(b) Fixed environment and decreasing $\Pi$

![Graph b]

(c) Fluctuating environment and increasing $\Pi$

![Graph c]

(d) Fluctuating environment and decreasing $\Pi$

![Graph d]

Figure 4: The body condition distribution of dispersers, $d$ (left panels), and of non-dispersers, $f$ (middle panels), and the population-wide dispersal probability as a function of body condition, $E[p(z, y)|z]$ (right panels) at the ESS for different combinations of fixed and fluctuating environment and increasing and decreasing survival probability during dispersal $\Pi$. (The vertical lines indicate the means of $d$ and $f$). In each case, all patches are equally safe ($s = 0.5$ with fixed environment, $s = 0.9$ with fluctuating environment).
duced to a finite number $k$ in each patch. I calculated the ESS dispersal probability for $k = 2$ in every patch (Figure 2 in I), and for several cases where $k$ is a Poisson distributed number (Figure 3 in I). If the fraction of patches where $k = 0$ or $k = 1$ is sufficiently large, i.e., body condition does not play a role in competition in these patches, then weak individuals disperse with a high probability. This is because selection is weak on weak individuals due to their small competitive strength; they have a small probability to win competition as dispersers as well as philopatrics. Therefore, any mechanism that increases the fraction of patches with body condition independent competition, increases the dispersal probability of weak individuals. So does also the fourth example in I (Figure 4 therein), where in a small fraction of patches a fair lottery applies but in all other patches competition is by weighted lottery. Then the dispersal probability of weak individuals is high but individuals with intermediate and large body conditions behave as in a pure weighted lottery, such that also strong individuals disperse with high probability but individuals with intermediate body condition do not disperse. As long as competition is body condition dependent, different competition mechanisms do thus not induce any qualitative change of the ESS under the assumptions of I. However, the impact of body condition independent competition boosts dispersal of weak individuals.

**Dispersal of Strong Individuals.** The models in I – III provide different mechanisms that explain both dispersal of strong individuals and dispersal of weak individuals. Dispersal of strong individuals is mainly governed by their survival probability during dispersal. When they survive dispersal better than weak individuals (increasing $\Pi$), then their dispersal probability increases with body condition (cases (a) and (c) in Figure 4). Contrarily, with decreasing $\Pi$, their dispersal probability decreases with body condition (cases (b) and (d)). This is independent of whether the environment is fixed or fluctuating. It can be thus expected that in an environment with moderate temporal fluctuations of patch qualities, strong individuals would disperse according to how fit they are for dispersal.

**Dispersal of Weak Individuals.** In contrast, the dispersal behaviour of weak individuals is affected by how much the environment fluctuates but also by their survival probability during dispersal. Because selection on them is weak, i.e., they have small chances to win any patch through body condition dependent competition, they capitalize on every possibility to improve their expected reproductive success by random events. For instance, the example mechanisms in I that are different from pure weighted lottery and that increase the fraction of patches where the winner is chosen randomly, increase the dispersal probability of weak individuals. Or, in a fluctuating environment, the probability that a very poor natal patch will have a better quality in the next year is very high, and therefore, all offspring try to retain their natal patch by combining their competitive strength as non-dispersers. However, in a fixed environment, the only chance to improve individual productivity is to leave the very poor natal patch and, with very small but positive probability, gain a better patch. In an environment with partly correlated temporal fluctuations, weak are expected to disperse with high probability if fluctuations are
strongly correlated, and with low probability if fluctuations are mildly correlated.

The models in I – III simplify to the Hamilton–May model [27] under the same assumptions as in [27] (all patches have equal quality, all individuals have equal body condition, competition is via fair lottery, all established individuals survive until reproduction ($s = 1$), and the survival probability during dispersal, $\Pi$, is constant) and then predict the ESS dispersal probability given by formula (1).

The simulation model by Bonte and de la Peña [5] results (for constant dispersal mortality) in a negative relationship between body condition and dispersal in a patchy habitat with low environmental stochasticity that shifts towards a positive relationship when environmental stochasticity increases. These findings harmonize at least with some conclusions from my models; in a fluctuating environment, weak individuals do not disperse and the probability to disperse increases with increasing patch quality (cf. Figure 2 in II where the non-dispersing weight is constant and hence the dispersing weight increases with patch quality). In a fixed environment, very poor patches are abandoned.
3 Quasi-Local Competition and Pattern Formation

Ecological pattern formation is an attractive phenomenon that includes striped or mosaic vegetation over a larger region as presumably the most prominent example. In arid and semi-arid regions, so-called tiger bush can be observed which is a pattern of alternating bands of vegetation and bare ground [9, 64]. Other apparent examples are mosaic grassland [2], striped mussel beds or patterns in coral reefs [48]. But any stable non-uniform distribution of one or several species over some habitat can be considered as spatial pattern formation. The question ‘What determines the presence and abundance of a species in an environment?’ addresses a topic at the intellectual frontier of ecology [36].

Activator–Inhibitor Systems. Turing [58] provided a pioneer model for pattern formation by describing morphogenesis by reaction-diffusion equations. Diffusive instability (now even called Turing instability) is meanwhile the best-known mechanism for pattern formation. In a two-component system (e.g. two interacting chemicals or species), stable spatial patterns can occur if one component plays the role of an activator (it has a positive effect on its own and the other component’s synthesis or growth) and the other is an inhibitor (it inhibits the formation or growth of both components). Inhibition must happen on a larger spatial scale than activation. In ecology, this principle can apply to e.g. predator–prey systems when the predator is more mobile than the prey [50].

Pattern Formation in Metapopulations. In theoretical models for ecological systems, pattern formation emerges through a number of processes that all include these two key elements local activation and lateral inhibition [8, 50]. With these, pattern formation is also possible in single-species systems [25, 35]. In metapopulations, spatial patterns amount to the stable coexistence of different population densities in neighbouring habitat patches although environmental conditions are identical in all patches (homogeneous environment). Lateral inhibition involves processes between adjacent populations that have a negative effect on population growth (e.g. competition for resources, predation). Local activation includes processes that enhance local population growth (e.g. Allee-effects).

Quasi-Local Competition. Quasi-local competition [13] defines competitive interactions between individuals of neighbouring populations. For instance, as in the models in article IV, it can describe competition for resources such that individuals from one habitat patch forage not only in their own patch but also visit neighbouring patches and consume resources there. Alternatively, the resource could be moving between patches. Predator–prey interactions or host–parasite relationships can form another way of quasi-local competition if predators hunt in several adjacent habitat patches or if a host can be infected by more than one parasite individual [30].
3.1 Motivation

Doebeli and Killingback [13] model a single-species metapopulation where individuals spend a fraction $p$ of their foraging time in adjacent patches and a fraction $1 - p$ in the home patch. Individuals reproduce only in the home patch. Population density in a patch changes from one year to the next according to a function of Beverton–Holt type. Due to visiting foragers from neighbouring patches, the reproductive output in a patch depends on the density in that patch as well as in neighbouring patches. In the case of a two-patch metapopulation, the equation for the density $x^{(m+1)}_i$ of individuals in patch $i$ in the next year $m + 1$ as a function of the present density is

$$x^{(m+1)}_i = \frac{\lambda x^{(m)}_i}{1 + A \left[ (1 - p)x^{(m)}_i + px^{(m)}_j \right]}$$

for $i, j \in \{1; 2\}, i \neq j$. (24)

The parameters are phenomenologically motivated: $\lambda$ is the maximal per capita reproductive output in the absence of competition, and $A$ measures the impact of individuals in patch $i$ on reproduction in patch $i$.

This system has four equilibria: the trivial equilibrium $(0, 0)$, two asymmetric equilibria $(\tilde{x}, 0)$ and $(0, \tilde{x})$, and a symmetric equilibrium $(\bar{x}, \bar{x})$. The symmetric equilibrium is unstable and the two asymmetric equilibria are stable, i.e., stable patterns occur, if and only if $p > \frac{1}{2}$.

Kisdi and Utz [32] model the same scenario as in [13], but derive the population dynamics from first principles such that the model parameters are interpretable on an individual level. Moreover, they model resource dynamics explicitly, but assume fast resource dynamics such that the consumer dynamics can be analyzed at the quasi equilibrium of the resource. An inert egg stage is also included into the model. During the year, adults lay eggs according to the amount of resource they feed, and at the end of the year, the adults die and new adults hatch from the survived eggs. Between-year dynamics in an isolated patch follow the Beverton–Holt model. With two patches, the between-year dynamics of the adult consumers are

$$x^{(m+1)}_i = C x^{(m)}_i \left( \frac{1 - p}{1 + A \left[ (1 - p)x^{(m)}_i + px^{(m)}_j \right]} + \frac{p}{1 + A \left[ (1 - p)x^{(m)}_j + px^{(m)}_i \right]} \right)$$

for $i, j \in \{1; 2\}, i \neq j$. (25)

where $p$ is again the fraction of foraging time spent in the neighbouring patch. $C = \frac{K \gamma \beta (1 - e^{\delta})}{\delta}$ and $A = \frac{K \beta}{\alpha}$, where all parameters are defined as in the model in the following Section; however, the conclusion drawn in this Section does not depend on their precise interpretations.

For $C > 1$, this system has four biologically meaningful fixed points: the trivial equilibrium, two inhomogeneous boundary equilibria and a homogeneous equilibrium. The homogeneous equilibrium is generically stable for $p \neq \frac{1}{2}$, and the other equilibria are unstable. Ecological pattern formation is hence not possible in this model.
Why these two apparently similar models exhibit very different dynamics, becomes clear when comparing their equations for the between-year dynamics. In equation (25), the first summand in the parentheses considers how individuals that forage in the home patch compete with each other and how they are affected by visiting foragers from the neighbouring patch. Analogously, the second summand describes competition between individuals that forage in the neighbouring patch with the local individuals there that do not leave the patch. Hence, quasi-local competition (foraging in the neighbouring patch) does not only affect negatively the neighbouring population but also the own population because individuals from one population continue to compete for resources also while jointly foraging outside their own patch. Quasi-local competition effects lateral inhibition, but at the same time no locally activating mechanism is working. Therefore, the model in [32] does not yield stable inhomogeneous patterns.

However, the population dynamics given by equation (24) lack the term that regards competition in the neighbouring patch. The inhibiting effect that quasi-local competition has on the neighbouring patch is hence accompanied by relaxed competition between individuals from the same patch while foraging in the other patch. This facilitates pattern formation.

3.2 The Model

Within-Year and Between-Year Dynamics. Motivated by the discrepancy between the two introduced models, I have extended the resource–consumer model in [32] to an age-structured model by introducing a second active age stage, the juveniles. In a patch \( i \), adults \( x_i \) consume the resource \( R_i \), but also spend a fraction \( p \) of their foraging time in the neighbouring patches (dividing the time evenly between the two adjacent patches) feeding on \( R_{i-1} \) and \( R_{i+1} \). Food is consumed at rate \( \beta \) and converted into eggs at rate \( \gamma \). Eggs \( E_i \) are laid only in the home patch during the year, and their mortality rate is \( \mu \). Juveniles \( y_i \) feed only in their home patch and consume the resource at rate \( \delta \). They die during the year according to the function \( \eta(R_i) = \frac{a}{b+R_i} + c \) that depends on the available resource. Adults are immortal during the year. The within-year dynamics for two patches \( i, j \in \{1; 2\}, i \neq j \), are given by the following ordinary differential equations where dots indicate time derivatives,

\[
\dot{R}^{(m)}_i(t) = \alpha \left[ 1 - \frac{R^{(m)}_i(t)}{K} - \beta R^{(m)}_i(t) \left( (1 - p)x^{(m)}_i(t) + p x^{(m)}_j(t) \right) - \delta R^{(m)}_i(t) y^{(m)}_i(t) \right] (26)
\]

\[
\dot{E}^{(m)}_i(t) = \gamma \beta x^{(m)}_i \left( (1 - p)R^{(m)}_i(t) + p R^{(m)}_j(t) \right) - \mu E^{(m)}_i(t) (27)
\]

\[
\dot{y}^{(m)}_i(t) = -\eta(R^{(m)}_i(t)) y^{(m)}_i(t) (28)
\]

\[
\dot{x}^{(m)}_i(t) = 0 (29)
\]

(see eqs (1) – (4) in IV). At the beginning of the next year \( m + 1 \), juveniles mature into adults (with probability \( \xi \)) and new juveniles hatch from the eggs that have survived the
3.2 The Model

winter (with probability $\sigma$). The winter survival probability of the resource and of the adults is $\varrho$ respectively $\theta$. The between-year dynamics are then

\begin{align}
R_i^{(m+1)}(0) &= \varrho R_i^{(m)}(1) \quad (30) \\
E_i^{(m+1)}(0) &= 0 \quad (31) \\
y_i^{(m+1)}(0) &= \sigma E_i^{(m)}(1) \quad (32) \\
x_i^{(m+1)} &= \xi y_i^{(m)}(1) + \theta x_i^{(m)} \quad (33)
\end{align}

(eqs (5) – (8) in IV). It is not possible to integrate the differential equations for the eggs (27) and for the juveniles (28) analytically. I have therefore analyzed this model entirely numerically. The resource dynamics are assumed to be much faster than the consumer population dynamics such that the resource is in quasi equilibrium. I performed a numerical analysis for changing parameter values of $a$ (how strongly juvenile survival depends on resource abundance), $\delta$ (the consumption rate of juveniles) and $p$ (the fraction of time adults spend foraging in the neighbouring patch), while keeping the other parameters fixed.

**Results.** The model exhibits intriguingly rich dynamics including stable inhomogeneous equilibria. Figure 5 shows the area where pattern formation occurs (shaded area) for a few selected cases. (For more examples, see Figure 1 in IV.) For parameter values within the shaded areas, the homogeneous equilibrium is unstable and two inhomogeneous boundary equilibria $(\tilde{x}, \tilde{y}, 0, 0)$ and $(0, 0, \tilde{x}, \tilde{y})$ are stable; outside the shaded areas stability is reversed, except for very high values of $\delta$ (bottom right panel) when cycles occur for values below the dashed line.

Stable patterns occur only when adults spend more time foraging in the other patch than at home ($p > \frac{1}{2}$). Only then the lateral inhibition effected by quasi-local competition is strong enough to induce pattern formation. Two different mechanisms are at work for different parameter regions. The first applies when juveniles feed very little ($\delta$ is very small) but their survival depends strongly on resource abundance ($a$ is large). If population densities are very different in the two patches, then many adults from the densely populated patch forage in the sparsely populated patch. They very much harm the juveniles there and induce high mortality among the juveniles. Hence, the already small population decreases further and tends towards extinction. At the same time, these same adults let much resource to their own juveniles in their home patch so that their juveniles enjoy resource in abundance. Although adults from the sparsely populated patch find abundant resource in their neighbouring patch (because juveniles do not feed much), this activating effect is weaker than the lateral inhibition due to quasi-local competition. Hence, stable patterns form.

The second mechanism occurs when the situation is reversed such that juveniles eat very much ($\delta$ is large) but are not much harmed by a scarce resource ($a$ is small). In this case, adults from the sparsely populated patch that try to forage in the neighbouring patch are not satisfied because of the resource depletion by the local juveniles there and
can hence not produce many eggs. Their population density decreases thus further until extinction. Adults and juveniles from the densely populated patch find much resource in the neighbouring patch respectively are not very sensitive to a low resource level in the own patch. Consequently, their population prospers, and the spatial pattern is stable.

Mechanisms analogous to lateral inhibition and local activation are both present in this model and enable spatial pattern formation. In the two scenarios described above, adults inhibit the growth of the neighbouring juvenile population (by foraging in the neighbouring patch) and the growth of their own adult population (by still competing with each other while foraging in the other patch). At the same time, they facilitate the growth of their own juvenile population by not harming their own juveniles too much. This is different from other classic activator-inhibitor systems where one component is not assumed to affect the dynamics at different places at the same time. Furthermore, in the classic activation mechanism, the growth rate of the activator increases with increasing density of the activator. Here, instead, absence of the adults from their natal patch increases the growth rate of their juveniles. Quasi-local competition is sufficient to induce pattern formation if it is supported by different levels of interaction with the resource in the two age classes.

Figure 5: Pattern formation in the two-patch model with quasi-local competition. The inhomogeneous equilibria are stable and the homogeneous equilibrium is unstable for parameter values within the shaded areas. Outside the shaded area, stability is reversed, except for the region below the dashed line in the bottom right panel where the model exhibits cycles.
4 Further Perspectives

Being pioneer models for body condition dependent dispersal, the models in articles I – III elucidate some aspects of this puzzling phenomenon. Not only do they provide several mechanisms that entail dispersal of weak individuals and dispersal of strong individuals, but they offer startling explanations why relationships between body condition and dispersal sometimes are not or cannot be detected in empirical data. Improving the models with regard to different aspects will certainly enhance understanding and refine further empirical investigations.

Competition Mechanisms. The prevailing assumption of competition by weighted lottery simplifies the analysis greatly but also constrains the results. The existence of equivalence classes of strategies is a consequence of this. Other competition mechanisms lack this feature and possibly yield different predictions. Although a competition scenario where the strongest individual in a patch wins does not qualitatively change the predictions compared to a weighted lottery in I, it may have a significant effect under different assumptions. Within-family behaviour is not considered in I. With variable body conditions among siblings as in II and III, individuals with small and intermediate body conditions may disperse more due to their low chance to retain their home patch, even when they are worse dispersers than their stronger siblings.

Sexual Populations. The models assume clonal inheritance of the focal trait. All offspring of a family are clones with respect to their dispersal behaviour. This does not mean that all offspring in a patch disperse with the same probability but that all apply the same dispersal rule $p$. With clonal inheritance there is no conflict of interest between an individual and the family it belongs to; both are interested in maximizing the fitness of the whole family. However, in sexual populations, offspring of a family are mainly not fully related to each other. Then, a parent is still interested in maximizing the fitness of the whole family, but an offspring cares most about itself and less about its kin. Models for sexual populations can therefore differ in their predictions depending on whether the parent or the offspring make the dispersal decision [41, 42, 56].

When offspring of a family are not all fully related, then the social dominance hypothesis takes effect, which assumes limited relatedness between individuals in a patch. With sexual reproduction, the models could thus be used to test the predictions of the social dominance hypothesis.

Within a sexual population, if sexes are explicitly distinguished, studying sex-specific dispersal behaviour may be of interest, as in nature, one of the sexes often disperses more actively [44, 59, 63].

Habitat Imprinting. Dispersal in general is considered intensely in the literature. On the contrary, small-range movement such as in article IV where individuals do not permanently leave but return to their natal territory, is given much less attention. However,
familiarity with habitat characteristics seems to be important. Immigrants can have a higher mortality risk [57] or lower reproductive success than local non-dispersers [65]; philopatric individuals need not spend time and energy to get acquainted with new habitat [52]. Further, the influence of natal habitat on habitat selection of dispersers is empirically evident [52, 53, 54]. The models in I–III could allow for these aspects such that non-dispersers or immigrants coming from similar natal patches have competitive advantages over other immigrants.

Propensity to Immigrate. Another worthwhile alteration of the models might be to implement the propensity to immigrate into a patch of certain quality in the sense that dispersers aim to improve their reproductive success by immigrating into a patch of better quality than their natal patch. An additional cost for choosiness could be added as well. The coevolution of dispersal probability and immigration propensity could then be studied. This would induce more competition for good patches and presumably yield different predictions than the present models.
Acknowledgements

I would like to express my deep gratitude to my two supervisors Éva Kisdi and Mats Gyllenberg for their excellent guidance and support during my PhD project. I have very much enjoyed working with and learning from them. I thank them for being an invaluable source of wisdom, expertise, and encouragement. I feel very honoured having been working with them.

The whole Biomathematics Research Group deserves my tribute for providing an outstanding and inspiring scientific environment with many talented researchers. I see it as a privilege that I could develop among them. I truly appreciate having been part of this group of strong scientific competence and diverse personal characters.

I thank Otso Ovaskainen and Andrew White for instructive comments while reviewing this thesis.

Innumerable dear friends have enriched my life in Finland essentially. I am grateful for their friendships that will accompany me forever.

I am deeply indebted to my parents and my brother for their immeasurable love and support in any possible way at any time.

This work has been financially supported by the Finnish Ministry of Education (through the graduate schools ComBi and FICS) and by the Finnish Centre of Excellence in Analysis and Dynamics Research of the Academy of Finland.

*Trust in the Lord with all your heart and lean not on your own understanding; in all your ways acknowledge Him, and He will direct your paths.*

Proverbs 3:5–6
References


REFERENCES


REFERENCES


Article I

With minor modifications adapted from

Evolution of condition-dependent dispersal under kin competition,
Mats Gyllenberg, Éva Kisdi, Margarete Utz.

Printed with kind permission from Springer Science + Business Media.
Evolution of Condition-Dependent Dispersal under Kin Competition

Mats Gyllenberg, Éva Kisdi, Margarete Utz

Department of Mathematics and Statistics, University of Helsinki, Finland

Abstract

Dispersers often differ in body condition from non-dispersers. The social dominance hypothesis explains dispersal of weak individuals, but it is not yet well understood why strong individuals, which could easily retain their natal site, are sometimes exposed to risky dispersal. Based on the model for dispersal under kin competition by Hamilton and May, we construct a model where dispersal propensity depends on body condition. We consider an annual species that inhabits a patchy environment with varying patch qualities. Offspring body condition corresponds to the quality of the natal patch and competitive ability increases with body condition.

Our main general result balances the fitness benefit from not dispersing and retaining the natal patch and the benefit from dispersing and establishing somewhere else. We present four different examples for competition, which all hint that dispersal of strong individuals may be a common outcome under the assumptions of the present model. In three of the examples, the evolutionarily stable dispersal probability is an increasing function of body condition. However, we found an example where, counterintuitively, the evolutionarily stable dispersal probability is a non-monotone function of body condition such that both very weak and very strong individuals disperse with high probability but individuals of intermediate body condition do not disperse at all.

Keywords: adaptive dynamics · condition-dependent dispersal · evolution · ESS · function-valued trait · kin competition · spatially structured population

Mathematics Subject Classification (2000): 92D15 · 92D40

1 Dispersal and kin competition

Dispersal plays a crucial role in the dynamics of populations and in species persistence and expansion. There is a huge and diverse body of literature exploring the evolution of this important trait, clustering around three main factors that promote dispersal. Firstly, dispersal alleviates competition among kin and thereby increases the inclusive fitness of individuals (Hamilton and May [21]; Taylor [43]; Gandon and Michalakis [13]; Rousset and Billiard [41]). This mechanism works when competition acts within a small area, which, in absence of dispersal, will be populated by relatives. Secondly, dispersal prevents inbreeding by removing relatives from the area where mating takes place (Bengtsson [4]; Perrin and Mazalov [39]). Thirdly, dispersal is favoured in stochastic environments and between non-equilibrium populations provided that environmental conditions and population densities at different locations are not fully correlated. Dispersal has then two advantages: it takes individuals from crowded locations to sites with low population density (e.g. to sites that have recently undergone a catastrophe) and hence helps avoiding competition with any
dispersal and kin competition

conspecifics (Levin et al. [30]; Olivieri et al. [36]; Holt and McPeek [24]), and it also avoids large fluctuations in the annual growth rate (which severely decrease the long-term geometric mean rate) by sampling the environment at different locations in each year (Metz et al. [32]; Wiener and Tuljapurkar [46]). In this paper, we concentrate on kin competition, specifically on competition among siblings, in an annual (semelparous) species.

On the other hand, there is a number of factors that disfavour dispersal. Firstly, dispersers are exposed to additional mortality, which reduces their fitness. Dispersal propensity often decreases with increasing dispersal cost (Hamilton and May [21]; Levin et al. [30]), although there are examples where very high dispersal cost selects for increasing dispersal via the indirect effect that the few successful dispersers have a high fitness because there are not many competitors (Gandon and Michalakis [13]; Kisdi [26]). This may prevent the extinction of a whole metapopulation. Secondly, being specifically adapted to the local habitat is a reason not to disperse and take the risk of landing in a disadvantageous environment (Balkau and Feldman [2]; Meszéna et al. [31]; Kisdi [27]). Thirdly, if a population experiences an Allee effect such that the population growth rate is negative if population size is below some critical value, dispersal into an empty patch is selected against (Gyllenberg et al. [17]; Gyllenberg et al. [20]). If, in addition, catastrophes destroy local populations, selection may drive a metapopulation to extinction (Gyllenberg and Parvinen [19]; Gyllenberg et al. [20]). This scenario, which is called evolutionary suicide, can also occur if the catastrophe rate increases with decreasing local population size, without any Allee effect (Gyllenberg et al. [20]).

An important individual aspect that affects dispersal propensity is body condition of the potential dispersers. In many instances, competitive ability, dispersal success and survival until maturation depend on body condition. More robust seeds do better in sustaining dry periods or times when they cannot germinate immediately and seedlings from larger seeds grow faster and therefore enjoy advantage in competition for light (see Geritz [14] or Mogie et al. [35] and references therein). On the other hand, among seeds that are carried by the wind, lighter individuals reach larger dispersal distances (Sinha and Davidar [42]). When animals compete for territories, social rank or mates, mainly body condition determines the winner; heavier individuals defeat lighter ones and demonstrative sexual traits ensure successful mating (Fisher and Cockburn [11]; Hoem et al. [23]). Individuals that have more fat reserves in their bodies may be prepared better against starvation, but cannot escape predators as easily (Kullberg et al. [28]; Witter et al. [47]). In this paper, we focus on the effect of condition-dependent competitive ability on dispersal behaviour and also consider condition-dependent dispersal cost whereas survival to maturity is taken to be independent of body condition.

The best known verbal hypothesis concerning condition-dependent dispersal is the social dominance hypothesis, also called ideal despotic distribution (Fretwell [12]). It states that stronger individuals that dominate socially suppress weaker individuals by e.g. defeating them in fights or denying them access to resources, whereupon the weaker individuals are forced to leave the local territory. But also without active suppression by stronger individuals, weaker individuals are often not very successful in foraging or reproducing (e.g. because strong individuals exploit the resources or get hold of all potential mating partners) and might disperse and try to find a better place somewhere else. For example Pasinelli and Walters [38] and Hanski et al. [22] observed dispersal of weak individuals, which they explained partly by social dominance. However, examples where strong individuals disperse rather than weak ones are frequently observed among different species (mammals: Wahlström and Liberg [45], birds: van der Jeugd [44], reptiles: Le Galliard et al. [29], insects: Anholt [1], plants: Berg [3]). Dispersal of strong individuals is also highlighted in
a recent review on natal habitat effects on dispersal behaviour (Benard and McCauley [3]).

The chances of obtaining new territories, social rank or reproduction mates are higher for stronger individuals, as well as dispersal cost may be smaller for strong individuals. However, because the former advantages hold also in the natal patch and there is at least some dispersal cost, the question why strong individuals disperse at all still remains. Because empirical studies show contrasting evidence both in favour and against the social dominance hypothesis and because there is no satisfying theory that explains dispersal of strong individuals, there is definitely a need for detailed mathematical models of condition-dependent dispersal.

Hamilton and May [21] were the first to develop analytical models for dispersal strategies under kin competition. They introduced a simple model for a population inhabiting a patchy environment where each site is occupied by one individual, the number of offspring per individual is constant and very large, parents die after reproduction, a fraction $p$ of the juveniles disperse, dispersal is global (dispersers are distributed evenly over all patches) and costly (probability of survival is $\Pi < 1$), and after dispersal, competition between immigrants and local non-dispersers is modelled by a fair lottery. The evolutionarily stable strategy (ESS) of dispersal, which is stable against the invasion of mutants with any different strategy, is then $p^* = 1/(2 - \Pi)$. Thus, the probability of dispersal is always greater than 1/2; even when dispersal is very costly ($\Pi \to 0$), still about half of the offspring disperse. Choosing almost sure death during dispersal for so many offspring may at first look surprising. However, note that almost all non-dispersers die as well, since only one individual establishes itself in each patch. Because dispersing kin are not all immigrating into one patch but are distributed over all patches and are thus not competing with each other, kin competition is avoided to a large extent by dispersal.

Comins et al. [8] generalized the model by Hamilton and May [21], assuming more than one individual per site, local extinction due to catastrophes, and stochastic reproduction, dispersal and competition. Gandon and Michalakis [13] investigated the dependency of the dispersal strategy on dispersal cost and relatedness between individuals within the population and showed that the ESS dispersal strategy is not always a decreasing function of dispersal cost or an increasing function of relatedness. Ronce et al. [40] studied the relation between natal dispersal and parental age by allowing for iteroparous individuals and thus adding kin competition between mother and offspring to the model. They verified the verbal hypothesis by Hamilton and May [21] that dispersal probability is decreasing with the age of the mother.

Ezoe and Iwasa [9] and Kisdi [26] developed extensions to the model of Hamilton and May [21] that investigated conditional dispersal. In both models the number of offspring varies between sites and dispersal strategy depends on the number of siblings. The ESS dispersal strategies are threshold strategies in both models: a certain number of offspring stay in the home patch while the rest disperse.

In this paper, we investigate dispersal under kin competition when dispersal cost and competitive ability depend on body condition. First we generalize the model of Hamilton and May [21] to condition-dependent dispersal and obtain a general analytic solution for the first order derivative of the ESS dispersal strategy with respect to body condition. It shows under which conditions dispersal is increasing with body condition. Then we find the evolutionarily stable dispersal strategy numerically in four examples of local competition within patches. All examples result in functions for dispersal probability such that strong individuals disperse with high probability. In three examples, the ESS dispersal strategy is an increasing function of body condition, but one example yields a non-monotone dispersal strategy, which is a highly counterintuitive result.
2 The model

We consider a single species, monomorphic population inhabiting a spatially structured environment with patches of varying quality. Let \( y \in (-\infty, +\infty) \) be the environmental quality of a patch and \( \phi(y) \) the probability density of patch qualities. In the beginning of the season each patch is occupied by one (juvenile, female) individual. (We do not consider male individuals here, merely assuming that there are sufficiently many males available to fertilize the females.) The life cycle is as follows:

1. **Survival.** A juvenile individual survives until maturity with probability \( s \).
2. **Reproduction.** The species is semelparous, i.e., if an individual survives until maturation, it dies immediately after reproduction. The average number of offspring of one individual is \( B \). Patches of good quality, such as patches with high resource abundance or suitable nesting conditions, naturally yield strong individuals. Thus, we assume that body condition \( z \in (-\infty, +\infty) \) of the offspring is the same as the patch quality \( y \) where they are born, or, retrospectively, we define the quality of the patch by the condition of offspring raised in that patch. We do not take maternal effects into consideration, i.e., the body condition of an offspring individual depends solely on the environmental quality of the natal patch and not e.g. on nurturing impacts by the mother. The offspring condition distribution in a patch of quality \( y \) is thus the point mass \( B\delta(\cdot - y) \) concentrated at \( y \).
3. **Dispersal.** Part of the offspring disperse and the rest stay in the natal patch. Let \( p(z) \) be the probability that an offspring individual of body condition \( z \) disperses. Dispersal is costly, whereas there is no cost to staying in the home patch. The probability of surviving dispersal is denoted by \( \Pi(z) \) and may also depend on the individual’s condition such that \( \Pi(z) \) increases with \( z \). Further more, we assume global dispersal, i.e., dispersers are distributed uniformly over patches.
4. **Competition.** After dispersal, non-dispersers and immigrants compete in each patch and only one individual establishes itself in the patch whereas all others are killed. Competitive ability depends on body condition such that stronger individuals establish themselves with higher probability.
5. **Random Reassignment of Patch Qualities.** After competition, all patches are occupied by one individual. We assume that there are environmental fluctuations within patches and that patch qualities are independent over years; e.g., resource abundance can depend on temporary local conditions and be high in one year but low in the next year. Thus, we randomly reassign the qualities of the patches at the end of the season, independently of the past and of the body conditions of the individuals that inhabit the patches. Then the population state of the monomorphic population is the same as in the previous year, i.e., the density of patches of a certain quality \( y \) that are inhabited by individuals of a certain condition \( z \) is constant over the years and the population is in dynamical equilibrium.

2.1 Mutant population dynamics

In the following we use the theory of adaptive dynamics to link short-term population dynamics with long-term evolutionary changes. (Geritz et al. [15] and Metz et al. [33] provide a general introduction to the theory of adaptive dynamics, Gyllenberg and Metz [18] and Metz and Gyllenberg [34] specialize to metapopulations).

We introduce a mutant, assuming that the resident population is at equilibrium. Mutants are initially rare, thus, the equilibrium dynamics of the resident population are at first not affected by
the occurrence of a mutation. The mutant differs from the resident in its propensity to disperse, which we denote by \(p_m(z)\); otherwise there is no difference to the resident. Unlike many applications of adaptive dynamics, which assume small mutational steps (see e.g. Geritz et al. [15] and Metz et al. [33]), we allow for mutational steps of any size in the dispersal strategy. This assumptions is realistic in our case, since every individual has in principle the ability to disperse and can make use of this option or not.

Let \(N\) be the number of patches, \(m\) the number of mutants in the population and \(M = m/N\) the number of mutants per patch. After dispersal, the number of mutant immigrants in a patch follows a binomial distribution, \(\text{Bin}(m \cdot B \cdot d_m, 1/N)\), where \(d_m = \int_{-\infty}^{+\infty} \phi(z) \cdot p_m(z) \cdot \Pi(z) \, dz\) is the average probability of successful dispersal of one mutant offspring. To be able to use deterministic models, we let \(N \to \infty\), \(m \to \infty\) and \(B \to \infty\), such that \(M \to 0\) and \(B,M \to 0\). The binomial distribution then converges towards a distribution where most of the probability mass is concentrated at zero, and we neglect the probability that two or more mutants immigrate into the same patch. Therefore, we can assume that dispersing mutant offspring competes only against resident offspring, i.e., there is at most one mutant immigrant in a patch that was inhabited by a resident individual in the previous year.

Denote the probability that a mutant offspring of body condition \(z\) wins competition and establishes itself in a patch of quality \(y\), when the number of mutant competitors in that patch is \(M\) and the number of resident competitors with body condition within a small interval \(dz\) is \(Bu(z,y)dz\) by \(P(z,M/B,u)\). The particular form of \(u\) respectively \(P(z,M/B,u)\) depends on the type of patch (see list below) respectively on the assumptions made about competition (see examples in Section 3). The mutant encounters three kinds of patches:

(i) resident patches where the individual in the previous year survived until reproduction and an immigrating mutant competes with resident immigrants as well as resident non-dispersers born in that patch. The number of mutants is \(M = 1\) and the number of residents with body condition in a small interval \(dz\) is \(Bu_1(z,y)dz\) where

\[
u_1(z,y) = \phi(z) \cdot s \cdot p(z) \cdot \Pi(z) + \delta(z-y) \cdot (1 - p(y)) .\]

The first term on the right hand side of (i) represents the immigrants and the second term represents the non-dispersers that were born in that patch. Hence the probability that an immigrating mutant of body condition \(z\) establishes itself in a patch of quality \(y\) is \(P(z,\varepsilon, u_1(\cdot, y))\), where \(\varepsilon := 1/B\).

(ii) resident patches where the individual in the previous year did not survive until reproduction and a mutant only competes with immigrating residents. \(M = 1\) and the number of resident individuals with body condition \(dz\) is \(Bu_2(z)dz\) where

\[
u_2(z) = \phi(z) \cdot s \cdot p(z) \cdot \Pi(z) \]

which is the first term of the right hand side of (i). Here, a mutant of body condition \(z\) wins competition with probability \(P(z,\varepsilon, u_2(\cdot))\).

(iii) mutant patches where non-dispersing mutant offspring compete against immigrating resident individuals. In these patches the number of mutants is \(M = B(1 - p_m(z))\) and the number of resident immigrants with body condition \(dz\) is \(Bu_2(z)dz\). Thus, the probability that one of the mutants retains the patch is \(P(z, 1 - p_m(z), u_2(\cdot))\).
Because the qualities of the patches are randomly reassigned at the end of the year, the reproductive value of each established individual is the same, and therefore we can simply count the mutants in the population. The number of mutants per patch in the beginning of the next year $t + 1, \mathcal{M}_{t+1}$, is

$$\mathcal{M}_{t+1} = \mathcal{M}_t \left( \int_{-\infty}^{+\infty} \phi(z) s p_m(z) \Pi(z) R(z) \, dz + \int_{-\infty}^{+\infty} \phi(z) s P(z, 1 - p_m(z), u_2(\cdot)) \, dz \right)$$

where $\mathcal{M}_t$ is the number of mutants per patch in the previous year $t$ and

$$R(z) = B \int_{-\infty}^{+\infty} \phi(y) s P(z, \varepsilon, u_1(\cdot, y)) \, dy + B (1 - s) P(z, \varepsilon, u_2(\cdot))$$

is the number of resident patches that would be won by the offspring of a mutant of body condition $z$ if all offspring ($B$) dispersed and survived dispersal. The first term in the parentheses on the right hand side of (3) is the number of resident patches that are obtained by dispersing mutants that survive dispersal, averaged over the quality distribution of the patches. Because immigrants in a patch are independent, the number of patches won by dispersing mutants increases linearly with the fraction of dispersers and with slope $\Pi(z)R(z)$. The second term in the parenthesis on the right hand side of (3) is the number of patches that have already been occupied by a mutant in the previous year and that are retained by a mutant offspring. Since $B$ is very large and $P(z, \varepsilon, u_1(\cdot, y))$ as well as $P(z, \varepsilon, u_2(\cdot))$ are very small, we have to assure that the products $BP(z, \varepsilon, u)$ are well defined as $B \to \infty$ and $\varepsilon = 1/B \to 0$. Therefore, we assume that $P(z, \varepsilon, u_1(\cdot, y))$ and $P(z, \varepsilon, u_2(\cdot))$ increase linearly with $\varepsilon$ for small $\varepsilon$ such that $0 < \lim_{\varepsilon \to 0} BP(z, \varepsilon, u) < \infty$.

From (3) the invasion fitness of the mutant (Gyllenberg and Metz [18]; Metz and Gyllenberg [34]), i.e., the basic reproduction ratio when mutants are rare, is

$$W(p_m) = \int_{-\infty}^{+\infty} \phi(z) s \left( p_m(z) \Pi(z) R(z) + P(z, 1 - p_m(z), u_2(\cdot)) \right) \, dz$$

If $W(p_m) > 1$, the mutant increases in number from one year to the next and — if population dynamics are deterministic — the mutant spreads within the resident population over the years. When the mutant population is so small that it is subject to demographic stochasticity, the probability of invasion is positive but less than 1 (see e.g. Feller [10] or Jagers [25]). If $W(p_m) < 1$, the mutant goes extinct with probability 1. Note that the resident’s fitness is $W(p) = 1$ (i.e., on average, every individual is replaced by one descending individual) as the population is in dynamical equilibrium.

### 2.2 Evolutionarily stable strategy

Our aim is to find an evolutionarily stable strategy (ESS), i.e., a strategy that cannot be invaded by any other strategy if the entire population plays this strategy. When the resident strategy is the ESS, the mutant’s fitness is maximal (and equal to the resident’s fitness) if the mutant also plays the ESS, i.e., an ESS is the best reply to itself. We will first search for the best reply $\hat{p}_m(z)$ to a given strategy $p(z)$ by maximizing $W(p_m)$ for given $p(z)$. Because an ESS has the properties
2.2 Evolutionarily stable strategy

common to all best replies, we can deduce some characteristics of the ESS already from $\hat{p}_m(z)$. In the next section we calculate numerically the ESS for four different mechanisms for competition.

Because $p_m$ is a function-valued strategy and $W$ is a functional, we use the calculus of variations to find the best reply $\hat{p}_m$ to a given resident strategy $p$. (See Parvinen et al. [37] for the application of calculus of variations to adaptive dynamics of function-valued traits.) Let $F(p_m, z)$ denote the integrand in (1):

$$F(p_m, z) = \phi(z) s\left(p_m(z) \Pi(z) R(z) + P(z, 1 - p_m(z), u_2(\cdot))\right)$$

(6)

A necessary condition for the functional $W$ to have an extremal at $p_m = \hat{p}_m$ is that Euler’s equation is satisfied,

$$\frac{d}{dz} \frac{\partial F}{\partial p_m} \bigg|_{p_m = \hat{p}_m} = \frac{\partial F}{\partial p_m} \bigg|_{p_m = \hat{p}_m} \quad \text{for all } z \text{ such that } 0 < \hat{p}_m(z) < 1$$

(7)

with $\hat{p}'_m = dp_m/dz$. In our case, $W$ (and thus $F$) depends only on the function $p_m$ itself but not on its derivative $p'_m$, and Euler’s equation simplifies to

$$\frac{\partial F}{\partial p_m} \bigg|_{p_m = \hat{p}_m} = 0 \quad \text{for all } z \text{ such that } 0 < \hat{p}_m(z) < 1.$$  

(8)

Applying eq. (8) to the function $F$ defined by (6) one obtains the following result.

Result 1. The best reply strategy $\hat{p}_m$ of a rare mutant to the dispersal strategy $p$ of a resident population satisfies

$$\Pi(z) R(z) = P_2(z, 1 - \hat{p}_m(z), u_2(\cdot)) \quad \text{for all } z \text{ such that } 0 < \hat{p}_m(z) < 1$$

(9)

where $R(z)$ is given by (4), $u_1(z, y)$ and $u_2(z)$ are given by (1) and (2). Here, and in the remainder of this paper, we denote derivatives of $P$ by subscripts indicating the arguments with respect to which $P$ is differentiated. Thus, $P_2$ denotes the derivative of $P$ with respect to its second argument, $P_{22}$ the second order derivative with respect to its second argument, and $P_{21}$ the derivative with respect to the first and second argument.

In addition, $\hat{p}_m$ must also satisfy the condition

$$\frac{\partial^2 F}{\partial p_m^2} \leq 0 \bigg|_{p_m = \hat{p}_m} \quad \text{for all } z \text{ such that } 0 < \hat{p}_m(z) < 1,$$

(10)

such that it maximizes the mutant’s fitness, given the resident’s dispersal strategy $p$, i.e., it is the best reply to $p$. Inequality (10) is satisfied whenever $P_{22}(z, 1 - \hat{p}_m(z), u_2(\cdot))$ is negative, i.e., whenever $P(z, 1 - \hat{p}_m(z), u_2(\cdot))$ is concave with respect to the fraction of non-dispersing mutants. This is often true in nature; $P(z, 1 - p_m(z), u_2(\cdot))$ must saturate at a value less than or equal to 1 as a function of its second argument and thus must at least partly be concave. On the boundaries, $\hat{p}_m(z) = 0$ can be the best reply only if $W(p_m)$ is decreasing at $p_m(z) = 0$, whereas $\hat{p}_m(z) = 1$ can be the best reply only if $W(p_m)$ is increasing at $p_m(z) = 1$. In case the fitness functional has several maxima at different values of $p_m(z)$ for fixed $z$, the $p_m(z)$ that maximizes fitness globally is regarded as the best reply $\hat{p}_m(z)$. 

In (9), $P_2$ is certainly non-negative: the more individuals stay in the natal patch, the higher is the probability that one of them wins competition. $\Pi(z)R(z)$ is the slope at which the number of resident patches taken over by a mutant increases with the fraction of mutant dispersers (cf. argumentation after (4)). Therefore, (9) is what is known as a marginal value theorem (see Bulmer [6], pp. 102–121, for an introduction to the marginal value theorem applied to foraging theory): at the best reply $\hat{p}_m$ and consequently also at the ESS, the benefit from dispersing one more offspring is the same as the benefit from keeping one more offspring at home. When the benefit from dispersing one more offspring is greater or less than the benefit from keeping one more offspring at home, then the dispersal strategy evolves towards higher or lower dispersal probabilities, respectively.

In the general framework, it is not possible to solve (9) for $\hat{p}_m(z)$ explicitly, nor can we generally obtain an explicit expression for the ESS as the best reply to itself, for which we would have to solve (9) for $\hat{p}_m(z) = p(z)$. However, we can differentiate (9) implicitly with respect to $z$ and then solve for $d\hat{p}_m/dz$ to see whether $\hat{p}_m(z)$ is increasing or decreasing with $z$, i.e., whether stronger or weaker individuals disperse more. In this way we obtain the following result.

**Result 2.** The best reply $\hat{p}_m$ to a given dispersal strategy $p$ has the first order derivative with respect to body condition $z$,

$$
\frac{d\hat{p}_m(z)}{dz} = \frac{P_{21}(z, 1 - \hat{p}_m(z), u_2(\cdot)) - \frac{d}{dz}(\Pi(z)R(z))}{P_{22}(z, 1 - \hat{p}_m(z), u_2(\cdot))}
$$

for all $z$ such that $0 < \hat{p}_m(z) < 1$ (11)

with $R(z)$, $u_1(z, y)$ and $u_2(z)$ as given in (4), (i) and (2).

By (10), the denominator in (11) is negative at the best reply, and thus $\hat{p}_m(z)$ is increasing (decreasing) if the numerator is negative (positive). Recall that $\Pi(z)R(z)$ is the slope at which the number of resident patches obtained by a mutant increases with the fraction of mutant dispersers. $d(\Pi(z)R(z))/dz$ is thus a second order derivative characterizing how the increase of the number of patches obtained by dispersing mutants, with respect to the fraction of dispersers, depends on $z$. $P_{21}(z, 1 - \hat{p}_m(z), u_2(\cdot))$ describes how the increase of the mutant’s probability of retaining its own patch with respect to the fraction of non-dispersing offspring depends on $z$. If $d(\Pi(z)R(z))/dz$ is greater than $P_{21}(z, 1 - \hat{p}_m(z), u_2(\cdot))$, then $\hat{p}_m(z)$ is an increasing function, i.e., stronger individuals disperse with higher probability than weaker ones. In simpler words, if the change in the probability of success elsewhere with respect to the fraction of dispersers increases more with $z$ than the change in the probability of success in the home patch with respect to the fraction of non-dispersers increases with $z$, then dispersal is favoured more and more the stronger the individuals are. Note that $d(\Pi(z)R(z))/dz$ and $P_{21}(z, 1 - \hat{p}_m(z), u_2(\cdot))$ are generally functions (and not constants) and that thus their relative behaviour might be different for different $z$, and $\hat{p}_m$ might be a non-monotone function of $z$.

The above argumentation for the best reply $\hat{p}_m$ to an arbitrary but given resident strategy $p$ holds also for the ESS since the ESS is itself a best reply, i.e., the best reply to itself. Thus, the ESS has the same properties as those described by Result 1 and Result 2.

The present model simplifies to the model by Hamilton and May [21] if we assume that all patches have the same quality ($\phi(y) = \delta(y)$), all established individuals survive until reproduction ($s = 1$), the probability of survival during dispersal is constant ($\Pi(z) = \Pi$) and competition after dispersal is modelled as a fair lottery. Then the probability of establishment of an immigrant is $P(z, \varepsilon, u_1(\cdot), y) = 1/(Bp\Pi + B(1 - p))$. (Note that because $s = 1$, there are local non-dispersers...
in every patch such that \( u_1 \) determines the number of competitors.) The probability that one of the non-dispersers establishes itself in the patch is \( P(z, 1 - p_m(z), u_2(\cdot)) = B(1 - p_m)/(Bp\Pi + B(1 - p_m)) \). Thus, \( R(z) = 1/(p\Pi + 1 - p) \) and \( P_2(z, 1 - p_m(z), u_2(\cdot)) = p\Pi/(p\Pi + 1 - p_m)^2 \). Now solving (9) for \( p_m = p \) yields the ESS \( \hat{p} = 1/(2 - \Pi) \), as given by Hamilton and May [21].

In the model by Hamilton and May [21], \( \hat{p} = 1 \) is an ESS if \( \Pi = 1 \), i.e., when dispersal is costless all offspring disperse. However, in our model this is only true if the probability of retaining the natal patch is concave as a function of the fraction of non-dispersers, as it is the case in the fair lottery competition used by Hamilton and May [21]. With the assumptions of Hamilton and May [21] but with arbitrary competition, (6) with \( p(z) = 1 \) and \( \Pi(z) = 1 \) simplifies to \( F(p_m, z) = \delta(z)(p_m(z)B P(z, z, u_2(\cdot)) + P(z, 1 - p_m(z), u_2(\cdot))). \) (Note that because \( p = 1 \), in every patch, there are only immigrants such that \( u_2 \) determines the number of competitors.) Eq. (8) is satisfied for \( p_m = 1 \), i.e., the fitness has an extremum at \( p_m(z) = 1 \) for all \( z \). The second order derivative of \( F \) simplifies to \( \partial^2 F/\partial p_m^2 \big|_{p_m=1} = \delta(z)P_{22}(z, 0, u_2(\cdot)) \), and condition (10) is satisfied only if \( P_{22} \) is negative. Thus, the constant function \( \hat{p}_m(z) = \hat{p}(z) = 1 \) is the best reply to itself only if \( P(z, 1 - p_m(z), u_2(\cdot)) \) is concave.

### 3 Examples

In this section we investigate the model under concrete assumptions for local competition, which specify the probability of establishment \( P(z, M/B, u) \).

#### 3.1 Example: Weighted lottery competition

Let us assume that, after dispersal, competition in a local patch happens via weighted lottery. All individuals in a patch (i.e., non-dispersers and immigrants in patches where the individual survived until reproduction in the year before, or only immigrants in patches where the individual did not survive) are given weights according to their body condition \( z \). This is the most common way of modelling asymmetric competition (see e.g. Chesson and Warner [7], Geritz [14] or Geritz et al. [16]). Let \( g(z) \) be an increasing weight function. The probability that a mutant of body condition \( z \) establishes itself in a patch where \( M \) mutants are present and where \( u \) determines the number of resident individuals, is

\[
P(z, M/B, u) = \frac{g(z)M/B}{g(z)M/B + \int_{-\infty}^{+\infty} g(z') u(z', y) dz'} \tag{12}
\]

Because \( B \) is very large we will neglect the term \( g(z)M/B \) in the denominator of \( P(z, M/B, u) \) in patches where a single mutant is competing against residents (\( M = 1 \)). Hence, the probabilities that a mutant establishes itself in a given patch are as follows.

(i) In a resident patch of quality \( y \) where the individual in the previous year survived until reproduction, an immigrating mutant wins competition with probability

\[
P(z, \varepsilon, u_1(\cdot, y)) = \frac{g(z)}{B \int_{-\infty}^{+\infty} g(z') u_1(z', y) dz'} \tag{13}
\]
(ii) In a resident patch where the individual did not survive until reproduction, the probability that a mutant establishes itself is

\[ P(z, \varepsilon, u_2(\cdot)) = \frac{g(z)}{B \int_{-\infty}^{+\infty} g(z') u_2(z') dz'} \tag{14} \]

(iii) One of the non-dispersing mutant offspring retains the natal patch with probability

\[ P(z, 1 - p_m(z), u_2(\cdot)) = \frac{g(z)(1 - p_m(z))}{g(z)(1 - p_m(z)) + \int_{-\infty}^{+\infty} g(z') u_2(z') dz'} \tag{15} \]

It follows that

\[ P_{22}(z, 1 - p_m(z), u_2(\cdot)) = -\frac{2g(z)^2 \int_{-\infty}^{+\infty} g(z') u_2(z') dz'}{(g(z)(1 - p_m(z)) + \int_{-\infty}^{+\infty} g(z') u_2(z') dz')^3} < 0 \tag{16} \]

which ensures that the solution of (9) is a fitness maximum (cf argumentation after (10)). Formula (11) simplifies to

\[ \frac{d\hat{p}_m(z)}{dz} = -\frac{1}{P_{22}} \left[ \frac{g'(z) g(z)(1 - \hat{p}_m(z)) \int_{-\infty}^{+\infty} g(z') u_2(z') dz'}{(g(z)(1 - \hat{p}_m(z)) + \int_{-\infty}^{+\infty} g(z') u_2(z') dz')^3} \right. \]

\[ \left. + \Pi(z) R(z) \left( \frac{d \ln \Pi(z)}{dz} + \frac{g'(z)(1 - \hat{p}_m(z))}{g(z)(1 - \hat{p}_m(z)) + \int_{-\infty}^{+\infty} g(z') u_2(z') dz'} \right) \right] \tag{17} \]

with \( g'(z) = dg/dz \) and

\[ R(z) = \int_{-\infty}^{+\infty} \phi(y) \left[ \frac{sg(z)}{\int_{-\infty}^{+\infty} g(z') u_1(z', y) dz'} + \frac{(1 - s) g(z)}{\int_{-\infty}^{+\infty} g(z') u_2(z') dz'} \right] dy \tag{18} \]

as defined in (4). Both terms in the square brackets of (17) are positive and thus \( d\hat{p}_m/dz \) is positive, i.e., the probability of dispersing from the natal patch increases with body condition. This holds for every best reply \( \hat{p}_m \) and thus also for the ESS.

To explain this result heuristically, recall that there are two mechanisms at work that favour dispersal of stronger individuals. Firstly, stronger individuals have higher chances to win competition. In the weighted lottery model, there is a simple exchange ratio between strong and weak individuals such that few strong individuals are equivalent to many weak individuals concerning the probability of establishment. The effective number of individuals of a given body condition shall be the number of individuals multiplied by the weight function \( g(z) \). In a patch of good quality (where strong individuals are born) less individuals are needed to retain the patch than in a patch of bad quality (with weak individuals) and members of a family of strong individuals have high chances to establish themselves in many other patches if they disperse. Secondly, stronger individuals survive dispersal with higher probability. Note that for \( p_m \) being an increasing function it is sufficient that either \( \Pi(z) \) or \( g(z) \) is increasing.

Solving (9) for \( \hat{p}_m \), one formally obtains

\[ p_m(z) = 1 + \frac{D \sqrt{\frac{D}{\Pi(z)C}}}{g(z)} \tag{19} \]
where \( C := R(z)/g(z) \) and \( D := \int_{-\infty}^{+\infty} g(z')u_2(z')dz' \) are positive numbers which depend only on the resident strategy \( p(z) \).

Because \( p_m \) is a probability, we consider only the minus sign in the numerator on the right hand side of (19) because then and only then is \( p_m(z) \leq 1 \) for all \( z \). Secondly, \( p_m(z) \) is an increasing function (assuming that \( \Pi(z) \) and/or \( g(z) \) are increasing), which may assume negative values for small \( z \). For these values of \( z \), the fitness \( W(p_m) \) is decreasing at \( p_m(z) = 0 \) and therefore the best reply equals zero. Let \( z_0 \) be such that \( p_m(z_0) = 0 \); the best reply is then

\[
\hat{p}_m(z) := \begin{cases} 0 & \text{if } z \leq z_0 \\ p_m(z) & \text{if } z > z_0 \end{cases}
\]  

(20)

Combining (19) and (20) we define

\[
E(z) := (1 - \hat{p}_m(z))g(z) = \begin{cases} g(z) & \text{if } \hat{p}(z) \leq \hat{p}(z) \\ (1 - \hat{p}_m(z))g(z) & \text{if } \hat{p}(z) > \hat{p}(z) \end{cases}
\]  

(21)

The effective number of non-dispersing individuals in a patch is then \( E(z)B \). Recall that the constants \( C \) and \( D \) in (19) depend on the resident dispersal strategy \( p(z) \). The ESS \( \hat{p}(z) \), which is the best reply to itself, hence satisfies

\[
E(z) = \begin{cases} g(z) & \text{if } \hat{p}(z) = 0, \ i.e., \ z \leq z_0 \\ (1 - \hat{p}(z))g(z) & \text{if } \hat{p}(z) > 0, \ i.e., \ z > z_0 \end{cases}
\]  

(22)

when \( \hat{p}(z) \) is substituted for the resident strategy in \( C \) and \( D \). Note that when \( \Pi(z) = \Pi \) is constant, then \( E(z) = E \) is constant whenever \( \hat{p}(z) > 0 \). This means that the same effective number \( EB \) of offspring of all patches in each patch and the rest disperse whenever the effective number of offspring in a patch exceeds \( EB \), and all offspring stays in the natal patch if the effective number in the patch is less than \( EB \).

As an example, assume that patch qualities follow the standard normal distribution with probability density \( \phi(y) = (1/\sqrt{2\pi})\exp(-y^2/2) \), that the probability that an established individual survives until maturation is \( s = 0.9 \), and that the probability of surviving dispersal is \( \Pi(z) = 0.6 \). Let the weight function be \( g(z) = e^z \). Solving (22) numerically for \( E \) and \( z_0 \) such that \( g(z_0) = E \), one obtains \( z_0 \approx -0.97 \), \( E \approx 0.38 \) and \( \hat{p}(z) \) as shown in Figure 1 (solid line). In patches of quality \( z < z_0 \) the effective number of offspring born is less than \( EB \) and all offspring are kept at home; in patches of better quality \( z > z_0 \), a fixed effective number \( EB \) of offspring do not disperse and the rest disperse.

One might expect that increasing dispersal cost, i.e., decreasing \( \Pi \), would cause dispersal propensity to reduce. However, this is not generally true as very high dispersal cost can select for higher dispersal probability (Gandon and Michalakis [13]; Kisdi [26]). In the present example, \( \hat{p}(z) \) assumes smaller values for \( \Pi = 0.1 \) (dashed line in Figure 1) but higher values for \( \Pi = 0.01 \) (dotted line) than for \( \Pi = 0.6 \) (solid line). When dispersal cost increases, less immigrants come into a patch, and thus less non-dispersers are needed to retain the home patch. For the same reason, the fitness of those dispersers that survived dispersal and immigrated into a patch increases as well. On the other hand, the fitness of dispersers decreases because of the increasing dispersal cost. The relation of these factors decides whether dispersing or not dispersing is more profitable (see references above).
Figure 1: The ESS $\hat{p}(z)$ if competition is modelled as a weighted lottery with weight function $g(z) = e^z$, patch quality distribution $N(0, 1)$ and probability of survival until maturation $s = 0.9$. Solid line: the probability of survival during dispersal is $\Pi(z) = 0.6$. If patch quality and thus offspring body condition is less than $z_0 \approx -0.97$, all offspring stay in the natal patch. Dashed line: $\Pi = 0.1$. Dotted line: $\Pi = 0.01$.

### 3.2 Example: Strongest of $k$ offspring wins

A natural way to model condition-dependent competition is to assume that the strongest individual among all competitors in a patch wins. However, in our model there is no strongest individual because $B \to \infty$. Therefore, we assume that after dispersal condition-independent mortality reduces the number of individuals in each patch to a finite number $k$. Among these, the strongest individual establishes itself in the patch and the rest die. In this section we investigate the case when $k$ is a fixed number and the same for every patch; the next section deals with the more realistic case when $k$ is stochastic.

The probability $P(z, M/B, u)$ that a mutant of body condition $z$ establishes itself in a given patch where $M$ mutants are present and where $u$ determines the number of resident individuals, is as follows.

(i) A mutant that immigrates into a resident patch where the individual in the previous year survived until reproduction competes with resident immigrants and non-dispersers born in that patch, and wins competition with probability

$$P(z, \varepsilon, u_1(\cdot, y)) = \frac{k}{B \int_{-\infty}^{+\infty} u_1(z', y) dz'} \left( \frac{\int_{-\infty}^{z} u_1(z', y) dz'}{\int_{-\infty}^{+\infty} u_1(z', y) dz'} \right)^{k-1}$$  \hspace{1cm} (23)

The first factor on the right hand side of (23) is the probability that the mutant is among the $k$ individuals that survive the condition-independent stage of competition; the second factor gives the probability that all of the other $k-1$ surviving individuals have worse body condition than the mutant.

(ii) In a resident patch where the individual did not survive until reproduction and an immigrating mutant competes only against other immigrants, the probability that the mutant establishes itself is

$$P(z, \varepsilon, u_2(\cdot)) = \frac{k}{B \int_{-\infty}^{+\infty} u_2(z') dz'} \left( \frac{\int_{-\infty}^{z} u_2(z') dz'}{\int_{-\infty}^{+\infty} u_2(z') dz'} \right)^{k-1}$$  \hspace{1cm} (24)
3.2 Example: Strongest of $k$ offspring wins

Figure 2: The ESS $\hat{p}(z)$ if in each patch $k = 2$ individuals survive and then the stronger one establishes itself in the patch. Other parameter values are $s = 0.9$ and $\Pi(z) = 0.6$ and the patch quality distribution is $N(0, 1)$. If patch quality and thus offspring body condition is less than $z_0 \approx -0.4$, all offspring stay in the natal patch.

(iii) One of the non-dispersing mutant offspring retains the natal patch if at most $k - 1$ immigrants survive first and they are all of worse body condition than the mutant. The probability of establishment of a mutant of condition $z$ is

$$P(z, 1 - p_m(z), u_2(\cdot)) = \sum_{i=0}^{k-1} \binom{k}{i} \left( \frac{\int_{-\infty}^{z} u_2(z')dz'}{1 - p_m(z) + \int_{-\infty}^{+\infty} u_2(z')dz'} \right)^i \left( \frac{1 - p_m(z)}{1 - p_m(z) + \int_{-\infty}^{+\infty} u_2(z')dz'} \right)^{k-i}$$

The resulting formulas for the fitness (5) and for the best reply $\hat{p}_m$ to a given resident strategy $p$ are complicated and bulky, so that we restrict ourselves to the case $k = 2$. We solved (9) analytically to obtain the best reply $\hat{p}_m$ to a given strategy $p$. We then used a simple numerical procedure to find the ESS: we calculated the best reply $\hat{p}_m^{(1)}(z)$ to the constant function $p^{(1)}(z) = 0.5$, then the best reply $\hat{p}_m^{(2)}(z)$ to $p^{(2)}(z) = \hat{p}_m^{(1)}(z)$, and so forth. In the example shown in Figure 2 (patch quality distribution $N(0, 1)$ and parameter values $s = 0.9$ and $\Pi(z) = 0.6$), this procedure yielded a converging series of functions $\hat{p}_m^{(n)}(z)$, $(n = 1, 2, 3, \ldots)$, and the limit function is the best reply to itself, the ESS $\hat{p}(z)$.

In this example, $\hat{p}(z) = 0$ for $z < z_0 \approx -0.4$, i.e., in low quality patches all individuals stay in their natal patch (Figure 2). If one of them dispersed successfully and were among the two surviving individuals in the patch into which it immigrated, the probability that it is of better body condition than the other surviving individual and thus establishes itself in the patch is very small. On the other hand, if all offspring stay in the natal patch, there is a good chance that two of them survive with no other competitor left, and the patch is retained by a family member. In contrast, strong individuals establish themselves with high probability if only one of them is among the two surviving individuals. Keeping one more individual at home increases the probability of retaining the home patch more for weak individuals than for strong individuals, and that is the reason why weak individuals disperse less than strong individuals (cf (11)).
3.3 Example: Strongest of a random finite number of offspring wins

Assume now the more realistic situation that after dispersal, \( k \) individuals survive and \( k \) is a random variable. The strongest among the \( k \) individuals then establishes itself in the patch. The probability that a mutant with body condition \( z \) establishes itself in a particular patch of type (i), (ii) or (iii) is the average of (23), (24) or (25), respectively, over the distribution of \( k \). The fraction of patches that will contain only immigrants after dispersal is \( 1 - s + s \Pr(k = 0) \) (in a fraction \( (1 - \Pr(k = 0))(1 - s) \) of the patches, the individual in the previous year did not survive until reproduction; in a fraction \( \Pr(k = 0) \) of the patches, zero individuals survive condition-independent mortality). The number of patches obtained by dispersing mutants if all mutants from one patch dispersed and survived dispersal would be (cf (4))

\[
R(z) = \left( \int_{-\infty}^{+\infty} \phi(y) s (1 - \Pr(k = 0)) P(z, \varepsilon, u_1(\cdot, y)) dy + (1 - s + s \Pr(k = 0)) P(z, \varepsilon, u_2(\cdot)) \right)^{k - 1}
\]

Let us first investigate the case in which \( k \) follows a truncated Poisson distribution with parameter \( \lambda = 0.5 \), so that the total probability mass for \( k \geq 3 \), \( \Pr(k \geq 3) \approx 0.0144 \), is split proportionally among the terms for \( k = 0, 1 \) and \( 2 \). The numerical algorithm used in the previous example converges also in this case (for patch quality distribution \( X(0, 1) \) and parameter values \( s = 0.9 \) and \( \Pi(z) = 0.6 \) towards the ESS \( \hat{p}(z) \) that is shown in Figure 3 (thick line). The ESS is an increasing function of body condition and non-zero for all \( z \). This is qualitatively different from the ESSs in the previous examples: even in patches of very low quality always a certain fraction of offspring disperse. The reason behind this is that there is a relatively high probability that only one individual survives the condition-independent stage of competition (\( \Pr(k = 1) \approx 0.31 \)), whereupon it establishes itself in the patch, regardless of its body condition. On the other hand, the probability that \( k > 2 \) is relatively small (\( \Pr(k = 2) \approx 0.08 \)). The probability of establishment is thus affected very little by body condition and is only marginally lower for weak individuals than for strong individuals.

This case can be interpreted as a perturbation of the situation when \( \Pr(k = 0) = 2/3 \), \( \Pr(k = 1) = 1/3 \) and \( \Pr(k \geq 2) = 0 \). Then body condition is irrelevant for establishment and the ESS is a constant function (thin horizontal line in Figure 3). Further perturbation such that \( \Pr(k = 1) \) decreases and \( \Pr(k = 2) \) increases leads to a sigmoid \( \hat{p}(z) \) of wider range (dotted line in Figure 3 for \( \Pr(k = 0) = 0.47 \), \( \Pr(k = 1) = 0.23 \), \( \Pr(k = 2) = 0.3 \) and \( \Pr(k \geq 3) = 0 \). If \( k \) follows a truncated Poisson distribution with increasing \( \lambda \) and taking more terms of the Poisson sum into consideration, the probabilities for \( k = 0 \) and \( k = 1 \) decrease and competition depends more on body condition. With higher \( \lambda \), the evolutionarily stable dispersal strategy decreases for small values of \( z \) and increases for large values of \( z \) such that it spans a larger interval between 0 and 1, and eventually it assumes a shape as in the previous two examples (Figures 1 and 2) such that \( \hat{p}(z) = 0 \) for \( z < z_0 \) and \( \hat{p}(z) > 0 \) for \( z > z_0 \). The dashed line in Figure 3 is the ESS \( \hat{p}(z) \) when \( k \) follows a truncated Poisson distribution with \( \lambda = 2.5 \) such that \( \Pr(k \geq 7) = 0 \).

3.4 Example: Mixture of weighted and fair lottery competition

Let us now assume that condition-independent mortality occurs before competition only in a fraction \( \mu \) of the patches. For simplicity, we assume that in these patches exactly one randomly
chose individual survives as in a fair lottery, which then establishes itself in the patch. In all other patches a weighted lottery determines competition as in Example 3.1.

The probability \( P(z, M/B, u) \) that a mutant of body condition \( z \) establishes itself in a patch where \( M \) mutants are present and where \( u \) determines the number of resident individuals is

\[
P(z, M/B, u) = (1 - \mu) \frac{g(z) M/B}{g(z) M/B + \int_{-\infty}^{+\infty} g(z') u(z', y) dz'} + \mu \frac{M/B}{M/B + \int_{-\infty}^{+\infty} u(z', y) dz'}
\]  

which is a combination of a weighted lottery with weight function \( g(z) \) (first term on the right hand side of (27), cf (12)) and a fair lottery (second term on the right hand side of (27)). As before, we neglect the terms with \( M/B \) in the denominators in (27) if \( M = 1 \). Because

\[
P_{22}(z, 1 - p_m(z), u_{2}(\cdot)) = -(1 - \mu) \frac{2g(z)^2 \int_{-\infty}^{+\infty} g(z') u_{2}(z') dz'}{(g(z)(1 - p_m(z)) + \int_{-\infty}^{+\infty} g(z') u_{2}(z') dz')^3} - \mu \frac{2 \int_{-\infty}^{+\infty} u_{2}(z') dz'}{(1 - p_m(z) + \int_{-\infty}^{+\infty} u_{2}(z') dz')^3}
\]

is negative, the solution of (9) is a fitness maximum (cf argumentation after (10)).

Figure 4 shows the ESS (obtained by the same numerical algorithm as in the previous examples) if patch quality distribution is \( \mathcal{N}(0, 1) \), the weight function is \( g(z) = e^z \) and the parameter values are \( \mu = 0.1, s = 0.9 \) and \( \Pi(z) = 0.6 \). Surprisingly, the ESS is a non-monotone function of body condition. High dispersal of very weak offspring derives from the fact that in a fraction \( \mu \) of the patches, body condition does not play a role for competition (we have seen a similar effect in Example 3.3). In a pure weighted lottery (\( \mu = 0 \), as in Example 3.1), very weak individuals have a minimal chance to obtain any patch, independently of their dispersal strategy, even if a family tries to retain the natal patch by keeping all its offspring at home. Therefore, selection is very weak on weak individuals. In contrast, a fair lottery (\( \mu = 1 \)) selects for high dispersal as in the model by Hamilton and May [21]. If competition is independent of body condition in a fraction

Figure 3: The ESS \( \hat{P}(z) \) if in each patch \( k \) individuals survive the condition-independent stage of competition and then the strongest wins the patch where \( k \) is random. Parameter values are \( s = 0.9 \) and \( \Pi(z) = 0.6 \) and patch quality distribution is \( \mathcal{N}(0, 1) \). Thick line: \( k \) follows a truncated Poisson distribution with \( \lambda = 0.5 \), such that \( \Pr(k \geq 3) = 0 \). Horizontal thin line: \( \Pr(k = 0) = 2/3, \Pr(k = 1) = 1/3 \) and \( \Pr(k \geq 2) = 0 \). Dotted line: \( \Pr(k = 0) = 0.47, \Pr(k = 1) = 0.23, \Pr(k = 2) = 0.3 \) and \( \Pr(k \geq 3) = 0 \). Dashed line: \( k \) follows a truncated Poisson distribution with \( \lambda = 2.5 \) such that \( \Pr(k \geq 7) = 0 \).
Figure 4: The ESS $\hat{p}(z)$ if in a fraction $\mu$ of the patches a fair lottery determines the winner and in the rest of the patches local competition happens via a weighted lottery. Parameter values are $\mu = 0.1$, $s = 0.9$ and $\Pi(z) = 0.6$ and patch quality distribution is $\mathcal{N}(0, 1)$.

$\mu$ of the patches, the probability of establishment in these patches increases much for weak individuals. A small but positive $\mu$ selects for high dispersal among the weakest individuals (where selection is weak if $\mu = 0$), but does not affect the dispersal of strong individuals (where selection is stronger). Therefore, $\hat{p}(z)$ is increasing for intermediate and high values of $z$ as in Example 3.1 (cf Figure 1) but is also high at very small values of $z$.

4 Discussion

We investigated dispersal behaviour under kin competition when competitive ability (and possibly survival during dispersal) depends on body condition. Our model explains dispersal of strong individuals, a puzzling phenomenon given that strong individuals could easily retain the natal patch and yet are exposed to risky dispersal. Our main general result (Result 1) is a marginal value theorem balancing the fitness benefit from not dispersing and retaining the natal patch and the benefit from dispersal and establishment somewhere else. By implicitly differentiating both sides of (9) with respect to $z$ we obtain an expression for the slope of the best reply $\hat{p}_m(z)$ (Result 2). $\hat{p}_m(z)$ can be increasing or decreasing, depending on the behaviour of the probabilities of establishment in the different kinds of patches. An ESS is the best reply to itself and thus all properties of the best reply hold especially for the ESS. A common outcome of the present model seems to be an increasing dispersal fraction (Examples 3.1, 3.2 and 3.3), but we also presented a mechanism for competition that yields a non-monotone ESS (Example 3.4).

It is tempting to reason that under our assumptions, strong individuals generally disperse more because fewer of them are enough to retain the natal patch with high probability and strong dispersers can obtain many other patches. The non-monotone ESS in Example 3.4 (Figure 4) demonstrates clearly that this reasoning is generally not sound. Only under weighted lottery competition (Example 3.1) this argument applies, because there is a simple exchange ratio between strong and weak individuals. It is important to realize that the shape of the best reply $\hat{p}_m$ is determined by the mixed second order derivatives of the probabilities of establishment (Result 2) and not by first order derivatives.

We investigated four different mechanisms for within-patch competition. In the first example,
where competition is modelled by a weighted lottery (Figure 1), a fixed effective number \( EB \) of individuals per patch stay in each patch and the rest disperse if dispersal cost \( \Pi \) does not depend on \( z \) (if the effective number in a patch is less than \( EB \) then all offspring stay at home). Recall that the effective number is the weighted sum of individuals, where the weight is proportional to the competitive ability and thus an increasing function of body condition. This result is in accordance with the threshold strategies found by Ezoe and Iwasa [9] and Kisdi [26]. In fact, these two models are rather similar to our Example 3.1: Both models assume that the number of offspring varies between patches and dispersal depends on brood size, whereas in our Example 3.1 the effective number of offspring varies between patches, because it is determined by body condition and thus by the quality of the patch, and dispersal depends on the effective number.

In our second Example (Figure 2) where in each patch \( k = 2 \) individuals survive and then the stronger one wins, the ESS is such that all offspring stay in the natal patch if their body condition is low (\( z < z_0 \)), and part of the offspring disperse if \( z > z_0 \). However, in the case when \( k \) is a random variable, the ESS may predict high dispersal for every \( z \), even in patches of very low quality (Example 3.3, Figure 3). There is thus a qualitative difference between fixed and random \( k \). In the former case, individuals born in low quality patches do not disperse because their probability of establishment after dispersal is very low, whereas they increase the probability that two of them survive the condition-independent stage of competition and thus one of them retains the natal patch if they all stay in the home patch. A probabilistic \( k \) offers the opportunity of dispersing into patches where \( k = 1 \) and thus where body condition does not affect competition. If patches with \( k = 0 \) and \( k = 1 \) dominate, body condition plays only a minor role in the process of competition and establishment. Consequently, every patch yields dispersers, similarly to the model by Hamilton and May [21].

Examples 3.1, 3.2 and 3.3 illustrate that an increasing dispersal strategy is readily explained by our model with several different mechanisms for competition. However, in Example 3.4 (Figure 4) the ESS dispersal fraction is a non-monotone function of body condition. In Example 3.4, too, competition is independent of body condition in a small part of the patches. Already a small fraction of patches where the winner is determined at random is enough to yield high dispersal of very weak offspring, which have a chance to win in only these patches. The shape of \( \hat{p}(z) \) for intermediate and high values of \( z \) is not affected much by the fact that competition is random in a small part of the patches and is thus very similar to the ESS in Example 3.1 (Figure 1). Note that experimenters may have difficulties to recognize this kind of non-monotone function in their empirical data. In a scenario as in Figure 4, a few low measurements of dispersal at intermediate values of \( z \) might be interpreted as noise, and a regression analysis might be misleading by showing no correlation between dispersal and body condition. In the appendix, we demonstrate that our model can also yield a constant ESS while it still satisfies the basic biological properties of competition. By perturbing the example in the appendix, one can obtain ESSs of any shape (sufficiently close to the constant function).

In Example 3.1, there exists a unique ESS. In all other examples, it is possible that there exist multiple ESSs, i.e., there exist two or more functions each of which is the best reply to itself. However, due to the complexity of our model, we cannot be sure that there are no other ESSs besides the ones we calculated. Therefore, in all of our examples we are content with finding one ESS.

Our model is based on a number of simplifying assumptions that can be modified in future research to make the model more realistic and gain more insight into the underlying mechanisms that shape the evolution of condition-dependent dispersal. In the present model, the distribution
of offspring body condition is solely determined by the quality of the natal patch. More realistically one could assume that offspring body condition follows some (e.g. Gaussian) distribution with mean at the quality of the natal patch. Then the question can be studied which individuals within a family disperse. Even more realistic would be to assume that body condition is partly influenced by the mother and thus is a function of both the mother’s body condition and the environmental quality of the natal patch.

Another modification of the present model could be to assume a trade-off between fecundity of the parent and body condition of the offspring. Ezoe and Iwasa [9] assume that in patches of better quality more offspring are born, but all offspring have the same body condition, whereas in the present model all patches yield the same number of offspring, but individuals born in better patches are stronger. Our model could be extended by assuming that a parent can decide the number of offspring such that the body condition of the offspring decreases with increasing number of offspring. This implies that a parent knows the quality of the patch at the time of reproduction. The present assumption of constant fecundity is realistic if a parent at reproduction is not aware of patch quality, e.g. because it has to decide the number of offspring before the random reassignment of patch qualities.

In this article we assumed that environmental qualities of patches in consecutive years are independent and hence patch qualities are randomly reassigned each year. The opposite case would be that patch quality remains constant over the years. It is particularly interesting how dispersal behaviour will change under this assumption. Weak individuals would want to disperse more in order to obtain better patches than their natal patches, but their chances of establishment are small. A family inhabiting a good quality patch, on the other hand, might put more effort in retaining this patch and thus strong individuals might disperse less than in the present model. A natural addition would be to introduce the propensity of immigrating into a patch of a certain quality, e.g. that every dispersing individual wants to immigrate into a patch of at least the same quality as its natal patch; being choosy can be penalized by a higher dispersal cost due to visiting more patches.

This research was supported by the Graduate School in Computational Biology, Bioinformatics and Biometry (ComBi) of the Ministry of Education in Finland and by the Academy of Finland. We thank Hans Metz for many illuminating discussions.

Appendix

In this appendix, we construct an example in which \( \hat{p}(z) \) is constant and thus prove that a constant \( \hat{p}(z) \) is consistent with the underlying biological framework of the model. By perturbing this example one can obtain models where the ESS dispersal fraction assumes any arbitrary function (sufficiently close to the constant function).

Let the ESS dispersal fraction be

\[
\hat{p}(z) = 1 - Q
\]  

and assume that the probability density of patch qualities \( \phi(y) \) is given, as well as the probability of survival until maturity, \( s \), the probability of dispersal success, \( \Pi \) (it shall be constant), and the fraction of non-dispersers, \( Q \). (We will later see that \( Q \) has to be chosen such that it satisfies inequality (35).) Let \( \psi(z) \) be an increasing function such that \( 0 < \psi(z) < 1 \) for all \( z \in \text{supp}(\phi) \) and let \( f(z,y) \) be an increasing function in \( z \) and decreasing in \( y \) with \( 0 < f(z,y) < 1 \). Further, define the function \( \gamma(z) := s \int_{-\infty}^{\infty} \phi(y) f(z,y) \, dy + 1 - s \), which is increasing and \( 0 < \gamma(z) < 1 \).
If the putative ESS in (29) is established in the population, the number of individuals with body condition in a small interval $dz$ after dispersal in patches where the individual did not survive in the year before is then $Bu_2(z)dz$ where (cf (2))

$$u_2(z) = \phi(z) s(1 - Q)\Pi$$

and in patches where the individual survived it is $Bu_1(z,y)dz$ where (cf (1))

$$u_1(z,y) = u_2(z) + \delta(z-y)Q$$

A single immigrant of body condition $z$ has smaller chances to establish itself in a patch where there are other immigrants as well as local non-migrants $(u_1(z,y))$ than in a patch with only immigrants $(u_2(z))$. Therefore, let

$$P(z, \varepsilon, u_1(\cdot, y)) = P(z, \varepsilon, u_2(\cdot)) f(z, y)$$

We linearize $P(z, \varepsilon, u)$ around $\varepsilon = 0$ and take into consideration that $P(z, 0, u) = 0$. Then (4) simplifies to

$$R(z) = s \int_{-\infty}^{+\infty} \phi(y) P_2(z, 0, u_1(\cdot, y)) dy + (1 - s) P_2(z, 0, u_2(\cdot))$$

(33)

(recall that $\varepsilon B = 1$).

We define the probability that one of the non-dispersing individuals retains the natal patch if the number of immigrants $\int_{-\infty}^{+\infty} Bu_2(z)dz$ is as at the ESS, as a function of body condition $z$ and of the fraction of non-dispersers $q$,

$$P(z, q, \phi(\cdot) s(1 - Q)\Pi) = \frac{1}{CQ} Q \psi(z) q \left[ \frac{1}{\sqrt{\Pi \gamma(z)}} - 1 \right] q$$

(34)

with scaling factor $C = sQ \int_{-\infty}^{+\infty} \phi(z) \psi(z) \left[ \sqrt{\Pi \gamma(z)} + \frac{1 - Q}{q} \Pi \gamma(z) \right] dz$. The right hand side of (34) is positive for all $z$ and all $0 \leq q \leq 1$ and increasing in both $z$ and $q$. Because $P(z, 0, u_2(\cdot)) = 0$ and $P(z, q, u_2(\cdot))$ is an increasing function in $q$, it is sufficient to ensure that $P(z, Q, u_2(\cdot)) < 1$ for $P$ to be a probability. At the putative ESS $q = Q$, $\lim_{z \to \infty} P(z, q, u_2(\cdot)) < 1$ if and only if we choose

$$Q < \frac{s \sqrt{\Pi} \int_{-\infty}^{+\infty} \phi(z) \psi(z) \sqrt{\gamma(z)} dz}{1 - s \int_{-\infty}^{+\infty} \phi(z) \psi(z) \sqrt{\gamma(z)} \left[ 1 - \sqrt{\Pi \gamma(z)} \right] dz}$$

(35)

with the right hand side of (35) being a positive number. If $P(z, q, u_2(\cdot))$ exceeds 1 for some higher values of $q$, $\hat{q} < q \leq 1$, we can truncate $P(z, q, u_2(\cdot))$ and define $P(z, q, u_2(\cdot)) = 1$ for $\hat{q} < q \leq 1$ (where $\hat{q} > Q$ such that $P(z, q, u_2(\cdot))$ remains differentiable at $q = Q$).

After having set up the example with all necessary functions, let us now verify that a population playing strategy $\hat{p}(z) = 1 - Q$ is in dynamical equilibrium as well as $\hat{p}(z) = 1 - Q$ is indeed an ESS. At the putative ESS, $P_2(z, 0, \phi(\cdot) s(1 - Q)\Pi) = \psi(z)/C$ and hence $R(z) = \psi(z) \gamma(z)/C$. The fitness of a population with strategy $\hat{p}(z) = 1 - Q$ is then (cf (5))

$$W(1 - Q) = \frac{sQ}{C} \int_{-\infty}^{+\infty} \phi(z) \psi(z) \left[ \Pi \gamma(z) \frac{1 - Q}{Q} + \sqrt{\Pi \gamma(z)} \right] dz$$

(36)
i.e., the population is in dynamical equilibrium.

\( \hat{p}(z) = 1 - Q \) satisfies our two ESS conditions (9) and (11),

\[
P_2(z, Q, u_2) = \frac{1}{C} \Pi \psi(z) \gamma(z) = \Pi R(z)
\]

and

\[
P_{22}(z, Q, u_2) = -\frac{2}{Q} \psi(z) \Pi \gamma(z) \left[ 1 - \sqrt{\Pi \gamma(z)} \right] < 0
\]

so that \( \hat{p}(z) \) maximizes fitness and is indeed an evolutionarily stable dispersal strategy.

References


REFERENCES


Article II

With minor modifications adapted from

Journal of Biological Dynamics, in press
Variability within families and the evolution of body condition dependent dispersal, Mats Gyllenberg, Éva Kisdi, Margarete Utz.

Variability within Families
and the Evolution of Body Condition Dependent Dispersal

Mats Gyllenberg, Éva Kisdi, Margarete Utz
Department of Mathematics and Statistics, University of Helsinki, Finland

Abstract

In a population where body condition varies between and within families, we investigate the evolution of dispersal as a function of body condition ("strength", e.g., body size). Strong individuals are better competitors in a weighted lottery. If body condition does not influence survival during dispersal, then there is no unique evolutionarily stable strategy: Instead, there are infinitely many dispersal strategies that all lead to the same non-dispersing weight in a patch. These strategies are all selectively neutral but determine wildly different relationships between body condition and dispersal probability. This may explain why there is no consistent pattern between body condition and dispersal found in empirical studies. If body condition influences survival during dispersal, then neutrality is removed and individuals with higher survival probability disperse. Dispersers may be the competitively weaker individuals if smaller body size helps to avoid dispersal risks.

Keywords: adaptive dynamics · body condition dependent dispersal · evolution · ESS · function-valued trait · kin competition · spatially structured population

Mathematics Subject Classification (2000): 92D15 · 92D40

1 Introduction

Dispersal is undisputedly a fundamental ecological process. A vast amount of empirical and theoretical research deals with many different aspects of dispersal. Not all individuals within a population are equally likely to disperse, but dispersal is a complex process that is governed by many factors such as individual morphological, physiological and behavioural traits, structure of the local population and environmental properties of the habitat. Numerous review articles give valuable surveys of the abundant literature on that topic, see for example [26] or [2] for recent ones.

One significant parameter that influences dispersal behaviour is individual body condition. Empirical studies give evidence that body condition can be relevant for an individual’s decision whether to stay in its natal habitat patch or to leave and seek for new territory. Differences in body condition between dispersers and non-dispersers are observed in many species, including examples in mammals [10, 34], birds [25, 32], reptiles [21, 24], insects [1, 15] and plants [3]. In some of the studied examples, dispersers are heavier, larger, fatter or faster-running than philopatrics, whereas in other examples dispersers are observed to be lighter, smaller, slower, less aggressive or have less reproductive success than their conspecifics.

Especially in animals, there is limited understanding of the mechanisms that lead to dispersal of either strong or weak individuals. Dispersal of weak individuals is sometimes explained by the
social dominance hypothesis [11], arguing that weak individuals that are suppressed by stronger individuals leave their natal area and try to find a better place to live (see e.g. [25, 10]). Why strong individuals disperse is not well-understood. Although strong individuals can have essential advantages during dispersal (e.g. higher sustainability during periods of low food availability, [7, 30]) and establishment in a new patch (e.g. higher chance to win fights for new territory or reproduction mates, [32]) compared with weak individuals, they still often pay a relatively high cost during dispersal since dispersal is a very risky process in itself ([25, 19]) and they would have the option of staying in the patch and kicking out the weak if they wanted.

Understanding dispersal in plants may be more straightforward than in animals, as seeds and fruits are often dispersed by external dispersal vectors like wind or animals. It depends on the dispersal mechanism whether larger or lighter seeds disperse further away from the mother plant. Among wind-dispersed seeds, lighter seeds are likely to reach larger dispersal distances than heavier seeds [27], but with explosive seed dispersal, heavier seeds are dispersed farther [3]. In a plant with heteromorphic (proximal and distal) seeds, distal seeds have been found to be heavier [6].

Despite the large amount of empirical studies dealing with body condition dependent dispersal, very little emphasis has been given to actually model body condition dependent dispersal mathematically. In [16] we set up a mathematical model where the focal trait is the probability of dispersal as a function of individual body condition. That model is based on the model for dispersal under kin competition by Hamilton and May [18] but assumes that patches have different environmental qualities and individuals have different body conditions such that the body condition of offspring corresponds to the environmental quality of the patch where they are born. Further, survival probability during dispersal and competitive ability both increase with body condition. We found evolutionarily stable strategies (ESSs) for body condition dependent dispersal for several mechanisms for local competition (i.e., mechanisms that govern the establishment of offspring in habitat patches in the new season), which mainly predict dispersal of strong individuals. That model with its simple ecological and population dynamical assumptions provides insight into how different mechanisms for local competition affect dispersal behaviour and therefore can be seen as a null-model for body condition dependent dispersal.

The most important oversimplification of our previous model [16] is the perfect correlation of offspring body condition with natal patch quality. In the present article, we therefore generalize the model such that offspring body condition in a patch follows a Gaussian distribution around the environmental quality of the patch. This modification allows us to answer the question how differences in body condition of offspring affect dispersal behaviour and which individuals within a family disperse if a family consists of a range of individuals from weak to strong.

We assume full information and flexibility; every offspring individual is fully aware of its own body condition and of the quality of its natal patch and hence of the body condition of its siblings, and bases its decision to disperse on this knowledge. (Alternatively, we can imagine that a mother has full information about patch quality and body condition distribution of its offspring and appoints which offspring shall disperse.)

In this article, we discuss the cases that the probability of surviving dispersal is either constant or depends positively or negatively on body condition. When dispersers survive with the same probability regardless of body condition, then we find neutrality such that infinitely many dispersal strategies fall into equivalence classes that are characterized by the weighted sum of non-dispersers in a patch. The equivalence class of strategies that yield the evolutionarily stable non-dispersing weight is the ESS. When body condition influences survival during dispersal, this neutrality is broken and a unique ESS exists.
2 The Model

We consider a spatial structure with infinitely many habitat patches that differ in the quality of the environment \( y \in (-\infty, +\infty) \) with \( \phi(y) \) being the probability density of patch qualities \( y \) such that \( \int_{-\infty}^{+\infty} \phi(y) \, dy = 1 \). Each patch can support only one (female) individual. (We do not include male individuals in our model but assume that there are sufficiently many males available to fertilize the females.) The course of one season is as follows (see also Figure 1 for a schematic illustration of the life cycle, and Table 2 for a list of parameters and variables used in this article):

1. **Survival.** At the beginning of the year, each patch is occupied by one juvenile individual. An individual survives until maturity with probability \( s \).

2. **Reproduction.** We assume that the species is semelparous, i.e., individuals die immediately after reproduction. The average number of offspring of one individual is \( B \). Since we eventually want to arrive at a deterministic model, we shall later take the limit \( B \to \infty \). The body condition \( z \in (-\infty, +\infty) \) of offspring individuals depends on the quality of the patch where they are born and offspring body condition in one patch follows a distribution \( \beta(z, y) \) with the patch quality \( y \) as its mean. This assumption is justified by the fact that good environmental conditions (e.g. high resource abundance or suitable nesting conditions) naturally yield strong offspring on average, but offspring of the same family exhibit some variation of body condition.

3. **Dispersal.** A mother’s interest is that its offspring occupy as many patches as possible in the next generation. To maximize the fitness of a family, part of the offspring disperse and the rest stay and defend their natal patch. For an offspring individual with body condition \( z \), let \( p(z, y) \) be the probability that it disperses from its natal patch that has quality \( y \), and let \( \Pi(z) \) denote the probability of surviving dispersal. \( \Pi \) will be either constant (Section 3.1) or a monotonically increasing or decreasing function of body condition (Section 3.2). There is no cost to staying in the home patch. Dispersal is global such that dispersers are distributed uniformly over patches and every patch receives an identical sample of immigrants from the dispersal pool.

4. **Competition.** Because a patch can maintain only one individual, after dispersal, immigrants and (if present) local non-dispersers compete in each patch and one individual establishes itself in the patch whereas all others die. Competitive ability depends on body condition such that stronger individuals establish themselves with higher probability. We assume a weighted lottery competition, which is the most common way to model asymmetric competition (see e.g. [5], [12] or [14]), with the weight function \( g(z) \) that is increasing with \( z \).

5. **Random Reassignment of Patch Qualities.** At the end of the season we randomly reassign patch qualities. This represents the underlying assumption that the environmental quality of the habitat fluctuates temporally and locally such that patch qualities are independent of the past and independent of one another. Note that the environmental quality of a patch in the new season and the body condition of the inhabiting individual are unrelated, and, because we do not assume any maternal effects, the body condition of the offspring is influenced solely by the new patch quality.

The model presented in this article is a generalization of our preceding model for body condition dependent dispersal under kin competition [16]. The key difference between the two models is that in [16], offspring born in one patch have all the same body condition as the environmental quality of their natal patch (i.e., the probability density of offspring with body condition \( z \) in a patch of quality \( y \) is \( \beta(z, y) = \delta(z - y) \) where \( \delta \) is the Dirac delta function), whereas in the model presented in this article, the body condition of offspring born in one patch is distributed around
Figure 1: Life cycle of the model species.

<table>
<thead>
<tr>
<th>Notation</th>
<th>Explanation</th>
</tr>
</thead>
<tbody>
<tr>
<td>( A(y) )</td>
<td>( B A(y) ) is the weighted sum of all offspring born in a patch of quality ( y ) (formula (20))</td>
</tr>
<tr>
<td>( B )</td>
<td>number of offspring per individual, ( B \to \infty )</td>
</tr>
<tr>
<td>( \beta(z, y) )</td>
<td>probability density of body condition ( z ) of offspring in a patch of quality ( y ) (in numerical examples: formula (31))</td>
</tr>
<tr>
<td>( g(z) )</td>
<td>weight given to an individual with body condition ( z ) for weighted lottery competition (in numerical examples: formula (32))</td>
</tr>
<tr>
<td>( I )</td>
<td>( B I ) is the weighted sum of immigrants in a patch (formula (6))</td>
</tr>
<tr>
<td>( n(p, y) )</td>
<td>( B n(p, y) ) is the weighted sum of non-dispersers in a patch of quality ( y ) which use strategy ( p ) (formula (4))</td>
</tr>
<tr>
<td>( p(z, y) )</td>
<td>dispersal probability of a resident individual with body condition ( z ) born in a patch of quality ( y )</td>
</tr>
<tr>
<td>( p_m(z, y) )</td>
<td>dispersal probability of a mutant individual with body condition ( z ) born in a patch of quality ( y )</td>
</tr>
<tr>
<td>( P_m(z, n(p_m, y)) )</td>
<td>probability that a non-dispersing mutant with body condition ( z ) retains the home patch of quality ( y ) (formula (3))</td>
</tr>
<tr>
<td>( P_1(z, n(p, y)) )</td>
<td>probability that an immigrant with body condition ( z ) wins competition in a patch of quality ( y ) with local non-dispersers (formula (7))</td>
</tr>
<tr>
<td>( P_2(z) )</td>
<td>probability that an immigrant with body condition ( z ) wins competition in a patch without local non-dispersers (formula (9))</td>
</tr>
<tr>
<td>( \Pi(z) )</td>
<td>survival probability during dispersal of an individual with body condition ( z ) (in numerical examples: formula (54), (42) resp. (43))</td>
</tr>
<tr>
<td>( \phi(y) )</td>
<td>probability density of patches of quality ( y ) (in numerical examples: formula (30))</td>
</tr>
<tr>
<td>( R(z) )</td>
<td>average probability that a successful disperser with body condition ( z ) wins competition in a resident patch (formula (9))</td>
</tr>
<tr>
<td>( s )</td>
<td>probability to survive until maturation (in numerical examples: formula (31))</td>
</tr>
<tr>
<td>( W(p_m) )</td>
<td>mutant fitness (formula (1))</td>
</tr>
<tr>
<td>( y )</td>
<td>patch quality</td>
</tr>
<tr>
<td>( z )</td>
<td>individual body condition</td>
</tr>
</tbody>
</table>

Table 1: Table of parameters and variables
the environmental quality of the patch in a Gaussian manner.

We shall investigate the fate of a rare mutant that occurs in the population when the resident population is in dynamical equilibrium by applying the theory of Adaptive Dynamics (see e.g. [13] for a general introduction to Adaptive Dynamics and [17] for adaptive dynamics in metapopulations). The resident population in our model is instantaneously in equilibrium because offspring body condition is determined only by patch qualities and therefore, at the time of reproduction, the fraction of offspring individuals with body condition \( z \) that are born in patches of quality \( y \) is the same every season.

A mutant offspring with body condition \( z \) disperses from a patch of quality \( y \) with probability \( p_m(z, y) \), whereas the resident uses the strategy \( p(z, y) \). There is no other difference between the mutant and the resident. The mutant fitness is the number of patches that are occupied by the offspring of one mutant individual in the next generation. Necessarily, the fitness of the resident equals one, since, at the population equilibrium, on average exactly one offspring individual from each patch will successfully establish itself in a patch. The mutant fitness is given by the functional

\[
W(p_m) = \int_{-\infty}^{+\infty} \int_{-\infty}^{+\infty} F(z, y, p_m, n) \, dz \, dy,
\]

where

\[
F(z, y, p_m, n) = \phi(y) \, s \, B(\beta(z, y) \left(1 - p_m(z, y)\right)P_m(z, n(p_m, y) + p_m(z, y)\Pi(z)R(z).\]

Here,

\[
P_m(z, n(p_m, y)) = g(z)\tilde{P}_m(n(p_m, y)),
\]

is the probability that a given non-dispersing mutant with body condition \( z \) retains its natal patch of quality \( y \), where \( Bn(p_m, y) \) is the weighted sum of all non-dispersing mutant offspring,

\[
n(p, y) = \int_{-\infty}^{+\infty} g(z) \beta(z, y)(1 - p(z, y)) \, dz,
\]

and

\[
\tilde{P}_m(n(p_m, y)) = \frac{1}{B \left(n(p_m, y) + I\right)}.
\]

\( BI \) is the weighted sum of immigrants, which is the same in every patch, where

\[
I = \int_{-\infty}^{+\infty} \int_{-\infty}^{+\infty} \phi(y) \, g(z) \, s \, \beta(z, y) \, p(z, y) \, \Pi(z) \, dz \, dy.
\]

That is, the probability of winning competition is the focal individual’s competitive weight divided by the weighted sum of all individuals present in that patch. \( P_m(z, n(p_m, y)) \) becomes infinitesimally small as \( B \to \infty \), but \( B \) in the denominator cancels against the factor \( B \) in \( F(z, y, p_m, n) \).

Further, \( R(z) \) denotes the probability that one mutant offspring with body condition \( z \) establishes itself in a resident patch given that it disperses \( p_m(z, y) \) and survives dispersal \( \Pi(z) \). A surviving disperser can immigrate into two kinds of patches: (i) patches where the individual that occupied the patch in the year before survived until reproduction; in these patches, local non-dispersers and immigrants compete; the fraction of such patches among all patches is \( s \); (ii)
patches where the individual in the previous year died before maturation and so did not produce offspring; in these patches, only immigrants compete; the fraction of these patches is \( 1 - s \). The probability that an immigrant with body condition \( z \) wins competition in a patch of type (i) with environmental quality \( y \) is given by

\[
P_1(z, n(p, y)) = \frac{g(z)}{B(n(p, y) + I)}.
\]

(7)

Let \( P_2(z) \) be the probability that an immigrant with body condition \( z \) wins competition in a patch of type (ii) (in such patches, patch quality does not play a role since no local non-dispersers exist),

\[
P_2(z) = \frac{g(z)}{BI}.
\]

(8)

Then,

\[
R(z) = \int_{-\infty}^{+\infty} \phi(y) \left( sP_1(z, n(p, y)) + (1 - s)P_2(z) \right) dy
\]

where

\[
\hat{R} = \int_{-\infty}^{+\infty} \phi(y) \left( \frac{s}{B(n(p, y) + I)} + \frac{1 - s}{BI} \right) dy
\]

depends only on the resident.

### 3 Evolutionarily Stable Strategies

We are interested in finding dispersal strategies \( \hat{p} \) that are evolutionarily stable strategies (ESSs), i.e., uninvadable if the entire population plays \( \hat{p} \). Because an ESS is a best reply strategy to itself, let us start with finding the best reply of a mutant, \( \hat{p}_m \), to a given resident strategy \( p \). Because the integrand in formula (1), \( F(z, y, p_m, n) \), depends explicitly as well as implicitly on \( p_m \), as the functional \( n(p_m, y) \) depends on \( p_m \), too (cf. formula (4)), we first assume that the value of the functional \( n(p_m, y) \) is given by \( N_m(y) \); later we maximize \( W \) with respect to \( N_m \). In a given patch \( y_0 \), the mutant’s best reply \( \hat{p}_m(\cdot, y_0) \) should maximize its fitness \( \int_{-\infty}^{+\infty} F(z, y_0, p_m, N_m(y_0)) \) and in addition satisfy

\[
n(\hat{p}_m, y_0) = N_m(y_0).
\]

(11)

With the method of Lagrange multipliers, \( \hat{p}_m(\cdot, y_0) \) necessarily has to maximize

\[
L(y_0, \lambda, N_m(y_0), p_m)
\]

\[
= \int_{-\infty}^{+\infty} F(z, y_0, p_m, N_m(y_0)) \ dz + \lambda \left( n(p_m, y_0) - N_m(y_0) \right)
\]

\[
= \int_{-\infty}^{+\infty} \left( F(z, y_0, p_m, N_m(y_0)) + \lambda g(z) \beta(z, y_0)(1 - p_m(z, y_0)) \right) \ dz - \lambda N_m(y_0)
\]

where \( \lambda \) is a Lagrange multiplier, or equivalently, \( \hat{p}_m(\cdot, y_0) \) must maximize

\[
L(y_0, \lambda, N_m(y_0), p_m) + \lambda N_m(y_0) = \int_{-\infty}^{+\infty} \mathcal{F}(z, y_0, p_m, N_m(y_0), \lambda) \ dz,
\]

(13)
3.1 Case I: All individuals survive dispersal with the same probability

\[ \mathcal{F}(z, y_0, p_m, N_m(y_0), \lambda) = F(z, y_0, p_m, N_m(y_0)) + \lambda g(z) \beta(z, y_0)(1 - p_m(z, y_0)). \] (14)

We apply the calculus of variations to the functional in (13). Euler’s equation is a necessary condition for the above functional to have an extremal at \( p_m = \hat{p}_m \) (see e.g. [8]). In our case, Euler’s equation,

\[ \frac{\partial \mathcal{F}}{\partial p_m} \bigg|_{p_m=\hat{p}_m} = 0, \] (15)

yields that

\[ \phi(y_0) s B \left( \Pi(z) \hat{R} - \hat{P}_m(N_m(y_0)) \right) = \lambda \] (16)

has to be satisfied for all \( z \) such that \( 0 < \hat{p}_m(z, y_0) < 1 \).

Whether (16) can be satisfied, and hence whether there is a best reply such that \( 0 < \hat{p}_m(z, y_0) < 1 \) for any open interval of \( z \), depends on the nature of the function \( \Pi \). If \( \Pi \) is a constant, then there exists a \( \lambda \) that satisfies (16). If \( \Pi(z) \) is a non-constant function of \( z \), then there is no \( \lambda \) such that (16) can be satisfied for all \( z \). We will deal with these two cases separately in the following sections.

3.1 Case I: All individuals survive dispersal with the same probability

If the probability of survival during dispersal, \( \Pi \), is constant, i.e., every individual, regardless of body condition, has the same probability to survive dispersal, the left hand side of condition (16) does not depend on \( z \) and, for given \( y_0 \), the Lagrange multiplier is

\[ \lambda = \phi(y_0) s B \left( \Pi(z) \hat{R} - \hat{P}_m(N_m(y_0)) \right). \] (17)

The only constraint on the best reply strategy \( \hat{p}_m \) is that it satisfies (11) for all \( y \) for the function \( N_m \) that maximizes the fitness (1). A strategy is now in fact determined by the weighted sum of offspring that stay in their natal patch, \( BN(y) \), rather than by \( p \). We can therefore define equivalence classes of dispersal strategies, such that one class includes all \( p \) such that the weight of non-dispersers in a patch \( y \) equals \( BN(y) \), as follows.

With constant \( \Pi \), we can now rewrite the mutant fitness (1) in terms of \( N \) and \( N_m \) only. Note that

\[ \int_{-\infty}^{+\infty} g(z) \beta(z, y) p_m(z, y) \, dz = -N_m(y) + A(y), \] (18)

where (cf. (11))

\[ N_m(y) = \int_{-\infty}^{+\infty} g(z) \beta(z, y)(1 - p_m(z, y)) \, dz \] (19)

and

\[ A(y) = \int_{-\infty}^{+\infty} g(z) \beta(z, y) \, dz \] (20)

such that \( BA(y) \) is the weighted sum of all offspring born in a patch of quality \( y \). We assume that \( A(y) \) is a monotonically increasing function, which will be true in virtually all realistic cases. The mutant’s fitness then reads

\[ W(N_m) = \int_{-\infty}^{+\infty} \tilde{F}(y, N_m) \, dy, \] (21)
where
\[ \tilde{F}(y, N_m) = \phi(y) s B \left( N_m(y) \tilde{P}_m(N_m(y)) + \Pi \tilde{R} (A(y) - N_m(y)) \right). \] (22)

We now once more apply the calculus of variations to find the function \( N_m \) that maximizes the fitness functional \( W \). Euler’s equation,
\[ \frac{\partial \tilde{F}}{\partial N_m} = \phi(y) s B \left( \tilde{P}_m(N_m) + N_m(y) \frac{\partial \tilde{P}_m}{\partial N_m} - \Pi \tilde{R} \right) = 0, \] (23)
is satisfied for
\[ N_m^{(1)}(y) = -I + \sqrt{\frac{I}{\Pi R B}} \] (24)
and
\[ N_m^{(2)}(y) = -I - \sqrt{\frac{I}{\Pi R B}}. \] (25)

Because the root \( N_m^{(2)} \) is always negative and moreover a minimum of \( W \) (which can easily be verified by evaluating \( \partial^2 \tilde{F} / \partial N_m^2 \)), we need to consider only \( N_m^{(1)} \) in the following calculations. \( N_m^{(1)} \) is positive when \( \Pi \Pi R B < 1 \) and always maximizes \( W \) (as \( \partial^2 \tilde{F} / \partial N_m^2 \))(\( N_m^{(1)} \)) < 0 always. We notice that \( N_m^{(1)} \) is independent of \( y \), i.e., a mutant’s best reply strategy to a given resident strategy is that a certain weight \( B N_m^{(1)} \) stays in the natal patch, independent of the environmental quality of the patch. Consequently, any ESS will imply that a fixed weighted sum of non-dispersers \( B \tilde{N} \) stays in every patch and \( \tilde{N} \) is the same in every patch, except, of course, in patches where the total weight of offspring, \( BA(y) \), is smaller than \( B \tilde{N} \); in these patches all offspring stay in the path (cf. [9] and [20]). The excess weight of offspring, \( B(A(y) - \tilde{N}) \), born in a patch where \( BA(y) > B \tilde{N} \), disperses. Let \( \hat{y} \) denote the patch quality for which
\[ A(\hat{y}) = \hat{N} \] (26)
will hold such that, at the ESS, the weighted sum of non-dispersers in a patch of quality \( y \) will be
\[ B \hat{n}(y) = \begin{cases} BA(y) & \text{for } y < \hat{y} \\ B \hat{N} & \text{for } y \geq \hat{y}. \end{cases} \] (27)

(Note that because \( A(y) \) is assumed to increase monotonically with patch quality, there exists exactly one such \( \hat{y} \).) We have to take this truncation into account in the formulas for \( I \) and \( \hat{R} \), yielding
\[ I(\hat{y}) = \int_{\hat{y}}^{+\infty} \phi(y) s \Pi (A(y) - A(\hat{y})) dy, \] (28)
\[ \hat{R}(\hat{y}) = \frac{s}{B} \left( \int_{-\infty}^{\hat{y}} \phi(y) dy + \frac{1}{A(\hat{y}) + I(\hat{y})} \int_{\hat{y}}^{+\infty} \phi(y) dy \right) + \frac{1 - s}{B I(\hat{y})}. \] (29)

(Note that we need to know how \( I \) and \( \hat{R} \) depend on \( \hat{y} \), which we temporarily indicate by the notations \( I(\hat{y}) \) and \( \hat{R}(\hat{y}) \), in order to solve for \( \hat{y} \); as soon as we have found \( \hat{y} \), we have also found \( I(\hat{y}) \) and \( \hat{R}(\hat{y}) \), and \( I \) and \( \hat{R} \) assume of course each a single value for the whole metapopulation.) With \( I(\hat{y}) \) and \( \hat{R}(\hat{y}) \) and with putting \( N_m = N \), we need to solve (24) for \( \hat{y} \) to obtain the values of
3.1 Case I: All individuals survive dispersal with the same probability

\( \hat{y} \) and hence \( \hat{N} \) that characterize the evolutionarily stable equivalence class of dispersal strategies. This class contains all strategies such that all individuals stay in patches with quality \( y < \hat{y} \), whereas a weighted sum \( \hat{N} \) of individuals stay and the rest disperse from patches of quality \( y > \hat{y} \). The (infinitely many) strategies of the evolutionarily stable class are selectively neutral to each other and uninvadable by any other strategy.

3.1.1 Numerical Example

In the following example, let patch qualities follow a truncated normal distribution such that

\[
\phi(y) = \begin{cases} 
\frac{(1/\sqrt{2\pi})e^{-\frac{y^2}{2}}}{\int_{-3}^{3}(1/\sqrt{2\pi})e^{-\frac{y^2}{2}}dy} & \text{for } y \in [-3, 3], \\
0 & \text{otherwise.}
\end{cases}
\]  

(30)

Similarly, let offspring body condition in a patch of quality \( y \) follow a truncated normal distribution with mean \( y \),

\[
\beta(z, y) = \begin{cases} 
\frac{(1/\sqrt{2\pi})e^{-\frac{(z-y)^2}{2}}}{\int_{y-3}^{y+3}(1/\sqrt{2\pi})e^{-\frac{(z'-y)^2}{2}}dz'} & \text{for } z \in [y-3, y+3], \\
0 & \text{otherwise,}
\end{cases}
\]

(31)

such that offspring exhibit body conditions \( z \in [y-3, y+3] \). As weight function for the weighted lottery competition we choose

\[ g(z) = e^z. \]

(32)

The probability to survive until maturation is

\[ s = 0.9, \]

(33)

and the probability to survive dispersal is

\[ \Pi = 0.5. \]

(34)

Then the above calculations yield \( \hat{y}_c \approx -0.84 \) (where the subscript \( c \) indicates that \( \Pi \) is constant) and \( \hat{N} \approx 0.7 \). The top panel in Figure 2 shows \( \hat{n}_c(y) \) for this case (the thin dashed line is the total competitive weight produced in a patch, \( A(y) \)). The average non-dispersing weight in a patch (including empty patches),

\[
B\hat{n} = sB\int_{-\infty}^{+\infty}\phi(y)\hat{n}(y)dy,
\]

(35)

assumes the value \( B\hat{n}_c \approx 0.58B \) and the average weight of dispersers,

\[
Bw = sB\int_{-\infty}^{+\infty}\int_{-\infty}^{+\infty}\phi(y)g(z)\beta(z, y)\hat{p}(z, y)dzdy,
\]

(36)

is \( Bw_c \approx 1.76B \). The values for \( I \) and \( B\hat{R} \) are \( I_c \approx 0.88 \) and \( B\hat{R}_c \approx 0.71 \). For the following discussion of the results, Table 2 might be found helpful as it compactly compares the values of the essential quantities for the different cases discussed in this article.
Figure 2: Two examples for \( \hat{p} \) that both imply that the same weighted sum of offspring \( B_N \) stays in every patch that yields dispersers. Top panel: The evolutionarily stable strategy is such that a family in a patch of quality \( y \) keeps the non-dispersing weight \( B\hat{n}_c(\hat{y}) \) at home and the rest disperse (if present). Panels (i): Example dispersal strategy such that in every patch the weakest individuals stay and the stronger individuals disperse. Panels (ii): Example dispersal strategy such that in every patch the strongest individuals stay and the weaker individuals disperse. Panels (a): Position of the jump of the step function \( \hat{p} \) as a function of patch quality. Panels (b): Expected probability that an individual with given body condition disperses. Panels (c): Body condition distribution of dispersers in the whole metapopulation. Panels (d): Body condition distribution of non-dispersers in the whole metapopulation.
### 3.1 Case I: All individuals survive dispersal with the same probability

<table>
<thead>
<tr>
<th></th>
<th>( \bar{I} )</th>
<th>I</th>
<th>( B\bar{R} )</th>
<th>( \hat{y} )</th>
<th>( \hat{n} )</th>
<th>w</th>
<th>D</th>
</tr>
</thead>
<tbody>
<tr>
<td>constant</td>
<td>0.5</td>
<td>0.88</td>
<td>0.71</td>
<td>-0.84</td>
<td>0.58</td>
<td>1.76</td>
<td>0.22 for case (i)</td>
</tr>
<tr>
<td>increasing</td>
<td>0.74</td>
<td>1.33</td>
<td>0.54</td>
<td>-1.14</td>
<td>0.54</td>
<td>1.8</td>
<td>0.24</td>
</tr>
<tr>
<td>decreasing</td>
<td>0.32</td>
<td>0.54</td>
<td>0.92</td>
<td>-1.61</td>
<td>0.66</td>
<td>1.69</td>
<td>0.69</td>
</tr>
</tbody>
</table>

Table 2: Table of different quantities at the ESS for different shapes of the function II: \( \bar{I} \) is the fraction of the weight of all dispersers that survives dispersal as defined in formula (48); I is defined in formula (6) such that \( BI \) is the immigrant weight in every patch; \( \bar{R} \) is defined in formula (10) such that \( g(z)\bar{R} \) is the probability that an immigrant with body condition \( z \) wins competition in an average patch; \( \hat{y} \) is the minimum patch quality of patches that yield dispersers (cf. equation (26)); \( B\hat{n} \) is the average non-dispersing weight in a patch (see formula (35)); \( Bw \) is the average weight of individuals that disperse from a patch (see formula (36)); \( D \) is the fraction of dispersers among the total number of individuals in the population (see formula (38)). In the main text, subscripts \( c \), \( i \) and \( d \) on these quantities indicate the cases with different shapes of II (constant, increasing and decreasing, respectively).

It is now important to remember that all \( \hat{p} \) that yield the given \( \hat{n}_c(y) \) are in one equivalence class, and therefore the equivalence class itself is the ESS and individual strategies \( \hat{p} \) in that equivalence class are neutral against each other. This means that a range of very different dispersal strategies are equally evolutionarily stable. These include, as the most disparate strategies, (i) that the weakest individuals in a patch stay such that \( \hat{N} \) is as given and the stronger individuals disperse and (ii) that the strongest individuals stay and the weaker individuals disperse. This implies that in case (i), \( \hat{p} \) is a step function with respect to \( z \) with a jump from zero to one in patches where \( A(y) \geq \hat{N} \) and \( \hat{p} \) is constant zero in patches where \( A(y) < \hat{N} \). The position of the jump \( \hat{z}_c(y) \) depends on \( y \). For given \( y_0 \), it is straightforward to find the value of \( \hat{z}_c(y_0) \) as the formula for \( \hat{n} \) simplifies to (cf. formula 4)

\[
\hat{n}(\hat{p}, y_0) = \int_{-\infty}^{\hat{z}_c(y_0)} g(z) \beta(z, y) \, dz
\]

and \( \hat{z}_c(y_0) \) must satisfy \( \hat{n}(\hat{p}, y_0) = \hat{N} \). (For a patch \( y_0 \), \( \hat{p}(z, y_0) \) is schematically sketched in Figure 2 above the panels (a) for the two cases (i) and (ii).) Figure 2(ii)(a) shows the position of the jump in every patch (thick curve). In case (ii), \( \hat{p} \) is a step function as a function of \( z \) with a jump from one to zero in patches where \( A(y) > \hat{N} \) and \( \hat{p} \equiv 0 \) where \( A(y) < \hat{N} \). The values of the jumps for this case are shown in Figure 2(ii)(a)(thick curve). (Note that in order to find \( \hat{z}_c(y_0) \), we have to change the integration interval of \( \hat{n} \) in formula (37) to \( (\hat{z}_c(y_0), +\infty) \).) The dashed lines in Figure 2, panels (a), outline the area of pairs \( (z, y) \) that occur in the population: in a patch of quality \( y \), offspring with body conditions \( z \in [y - 3, y + 3] \) are born. The grey areas indicate dispersal: individuals with body condition \( z \) born in a patch of quality \( y \) disperse if and only if \( (z, y) \) lies within the grey area. The fraction of dispersers among all individuals in the population,

\[
D = s \int_{-\infty}^{+\infty} \int_{-\infty}^{+\infty} \phi(y) \beta(z, y) \hat{p}(z, y) \, dz \, dy,
\]

assumes the value \( D_c^{(i)} \approx 0.22 \) in case (i) and \( D_c^{(ii)} \approx 0.61 \) in case (ii).
Patches of quality \( y < \hat{y}_c \) do not yield dispersers, because the total weight of offspring, \( A(y) \), is less than \( \hat{N} \): therefore, the function \( \hat{z}_c(y) \) exists for \( y > \hat{y}_c \) in both cases (i) and (ii). In case (i), in patches with \( y \) only slightly bigger than \( \hat{y}_c \), only few (strong) individuals disperse and \( \hat{z}_c(y) \) is large; \( \hat{z}_c(y) \) is decreasing with increasing \( y \) as patches become better and thus individuals born in these patches become stronger and fewer individuals need to stay in their natal patch such that the non-dispersing weight is \( \hat{N} \). However, there is a second mechanism at play: the fact that with increasing patch quality also the body condition of the weakest offspring in a patch increases (the weakest individuals in a patch have body condition \( z = y - 3 \)), shifts the position of the jump of the step function \( \hat{p} \) towards higher values of \( \hat{z}_c \). This mechanism counteracts and eventually outplays the first mechanism such that \( \hat{z}_c(y) \) increases for sufficiently large \( y \).

When, on the contrary, the strongest individuals stay in a patch (panel (ii)(a)) then these two mechanisms are cooperating and \( \hat{z}_c(y) \) is a monotonically increasing function of \( y \). As in case (i), from worse patches \( (y \) slightly bigger than \( \hat{y}_c) \), only few individuals can disperse, but now the weakest disperse and therefore \( \hat{z}_c(y) \) is very small. With increasing \( y \) and hence increasing body condition of offspring in these patches, the number of dispersers increases and \( \hat{z}_c(y) \) increases with \( y \).

Panels (b) – (d) in Figure 2 show some biologically relevant quantities and distributions that are observable in the field. Panels (b) show the average probability that an individual with given body condition \( z \) disperses,

\[
\mathbb{E}(\hat{p}(z, y)|z) = \frac{\int_{-\infty}^{+\infty} \phi(y) \beta(z, y) \hat{p}(z, y) \, dy}{\int_{-\infty}^{+\infty} \phi(y) \beta(z, y) \, dy}.
\]

From panel (i)(a) we see that individuals with body condition \( z < \hat{z}_{c,\min} \approx 0.8 \) (the minimum value of the function \( \hat{z}_c \)) never disperse. Consequently, \( \mathbb{E}(\hat{p}(z, y)|z) = 0 \) for \( z < \hat{z}_{c,\min} \). For \( z > \hat{z}_{c,\min} \), \( \mathbb{E}(\hat{p}(z, y)|z) \) is monotonically increasing because with increasing \( z \) the probability of being born in a better patch increases and hence the number of offspring dispersing from a patch increases; and also within a patch, the probability of having body condition \( z > \hat{z}_c(y) \) increases. In the example of Figure (2)(i), these two effects produce a sharp switch between non-dispersing weak and dispersing strong individuals.

In panel (i)(c), we show the observable body condition distribution among dispersers,

\[
d(z) = \frac{\int_{-\infty}^{+\infty} \phi(y) \beta(z, y) \hat{p}(z, y) \, dy}{\int_{-\infty}^{+\infty} \int_{-\infty}^{+\infty} \phi(y) \beta(z', y) \hat{p}(z', y) \, dz' \, dy},
\]

where the vertical line in the graph indicates the mean of the distribution. Because of the rapid increase of \( \mathbb{E}(\hat{p}(z, y)|z) \) after \( \hat{z}_{c,\min} \) up to 1, almost all individuals with \( z > \hat{z}_{c,\min} \) disperse in the population, and hence, above \( \hat{z}_{c,\min} \), \( d \) has a similar shape as the body condition distribution of all offspring born in the metapopulation, \( \int_{-\infty}^{+\infty} \phi(y) \beta(z, y) \, dy \).

Similarly, the body condition distribution of non-dispersers,

\[
f(z) = \frac{\int_{-\infty}^{+\infty} \phi(y) \beta(z, y) (1 - \hat{p}(z, y)) \, dy}{\int_{-\infty}^{+\infty} \int_{-\infty}^{+\infty} \phi(y) \beta(z', y) (1 - \hat{p}(z', y)) \, dz' \, dy},
\]

resembles the body condition distribution of all offspring born in the metapopulation for \( z < \hat{z}_{c,\min} \) as all offspring stay in their natal patches that are weaker than \( \hat{z}_{c,\min} \), and declines rapidly for \( z > \hat{z}_{c,\min} \). \( f \) is depicted in panel (i)(d) with the vertical line indicating its mean.
3.2 Case II: Body condition influences survival during dispersal

In case (ii), where the weakest individuals in a patch disperse and the strongest stay, the expected probability to disperse as a function of body condition is shown in Figure 2(ii)(b). From patches of quality \( y < y_c \approx -0.84 \), no one disperses and therefore \( \mathbb{E}(\hat{\rho}(z, y) | z) \) is positive only for values of \( z > y_c - 3 \approx -3.84 \).

Unlike the sharp switch of case (i), individuals with a wide range of body conditions disperse, and the probability of dispersal is highest for intermediate to strong (but not very strong) body conditions. Altogether more individuals disperse than in case (i) (compare \( D_c(i) \approx 0.22 \) and \( D_c(ii) \approx 0.61 \)) because when the strongest offspring stay in a patch, less are needed to retain \( \hat{N} \) as the non-dispersing weight and the rest all disperse. Moreover, with increasing patch quality also offspring body conditions increase so that we see many strong individuals dispersing. Very weak individuals, on the one hand, cannot disperse because they are born in very bad patches where all offspring have to stay at home. On the other hand, very strong individuals do not disperse either because they are always the strongest in a patch and therefore stay. (For example, an individual with the best possible body condition \( z = 6 \) can only be born in a patch with \( y = 3 \) and is always the strongest individual among its siblings and hence never disperses.)

However, the body condition distributions of dispersers, \( d \) (panel (ii)(c)), and of non-dispersers, \( f \) (panel (ii)(d)), show both that weak and strong individuals are almost equally represented in the two groups, although we see slightly more strong dispersers (\( d \) is skewed a bit to the right; \( f \) is slightly skewed to the left). The vertical lines in plots (ii)(c) and (d) indicate the means of \( d \) and \( f \), respectively. Unlike the clear contrast between dispersers and non-dispersers in case (i), the difference between the mean body conditions of dispersers and non-dispersers in panels (ii)(c) – (d) would probably be impossible to measure in the field. This outcome has important biological implications: Although the weakest individuals disperse from individual patches, the phenomenon of weak individuals dispersing cannot be detected by simply measuring the distribution of body conditions among dispersers.

Recall that cases (i) and (ii) both describe dispersal strategies within the same evolutionarily stable equivalence class (both strategies keep the same competitive weight in a patch); yet they are very different from each other (in case (i), the weakest individuals of a family stay, in case (ii), the strongest stay). This implies that, with constant \( \Pi \), it is impossible to predict what dispersal behaviour would be observed in the field: The contrasts between panels (b) – (d)(i) and (ii) are striking and nevertheless both strategies (and any other strategy in between that keeps the same weight \( \hat{N} \) in most patches) are equally evolutionarily stable.

3.2 Case II: Body condition influences survival during dispersal

It may be more realistic to assume that survival during dispersal is influenced by body condition. For example, fat individuals can sustain periods of low resource availability better than weak individuals because they live on the fat reserves accumulated earlier in their bodies. Being strong is also advantageous in fights or encounters with conspecifics or predators that might happen during dispersal. In this case, we let the survival probability during dispersal be the increasing sigmoid function

\[
\Pi(z) = 0.2 + \frac{0.6}{1 + e^{-z}},
\]

which is bounded away from both zero and one such that weak individuals have a small but still positive probability to survive dispersal and even strong individuals disperse at a certain risk.
When we, on the contrary, assume a decreasing function for the probability of survival during dispersal,
\[
\Pi(z) = 0.8 - \frac{0.6}{1 + e^{-z}},
\tag{43}
\]
we implement the assumption that weaker individuals are more successful during dispersal than strong individuals. This can be justified, e.g. if we interpret body condition \( z \) as body size or body weight: larger individuals are competitively superior, but smaller or lighter individuals can easier hide or escape from predators during the risky phase of dispersing through unfamiliar territory.

When \( \Pi \) is a non-constant function, then, according to condition (16), there is no Lagrange multiplier \( \lambda \) that is the same for all \( z \), and thus Euler’s equation (15) is not satisfied. \( W \) thus does not attain its maximum for \( 0 < \hat{\rho}_m(z, y) < 1 \) in any set of \( y \) and \( z \) of non-zero measure, and the maximum must be at the boundary, i.e., \( \hat{\rho}_m \) must assume values zero or one almost everywhere.

In the Appendix, we prove that, within the set of piecewise continuous functions \( p \) in the domain where \( \beta(z, y)g(z) > 0 \), the best reply strategy is a step function of \( z \) with at most one jump from zero to one (respectively from one to zero) if \( \Pi(z) \) is monotonically increasing (decreasing) with \( z \). This result is easy to understand heuristically from formulas (1) and (2): The probability of retaining the natal patch, \( P_n \), depends only on the competitive weight kept at home, \( Bn(p_m, y) \), but not on which offspring make up that weight. The best strategy is thus to disperse offspring that have high probability to survive dispersal and compose \( Bn(p_m, y) \) of offspring who have low \( \Pi(z) \) and would thus likely be lost during dispersal.

Let \( \hat{z}_i(y) \) (subscript \( i \) indicating the case with increasing \( \Pi \)) be the position of the jump such that with increasing \( \Pi \), the best reply strategy is
\[
\hat{\rho}_m(z, y) = \begin{cases} 
0 & \text{if } z < \hat{z}_i(y) \\
1 & \text{if } z > \hat{z}_i(y) 
\end{cases}
\tag{44}
\]
when \( \hat{z}_i(y) \) exists, and constant zero when \( \hat{z}_i(y) \) does not exist, i.e., in patches that do not produce dispersers (see Appendix; note that we do not specify the value \( \hat{\rho}_m(\hat{z}_i(y), y) \) since the value of \( \hat{\rho}_m \) at a single point \( z \) does not affect the value of the integral in formula (1)). To find the best reply, we maximize \( W(p_m) \) with respect to the function \( y \mapsto \hat{z}_i(y) \) or, equivalently, maximize
\[
\int_{-\infty}^{\hat{z}_i(y)} \frac{g(z) \beta(z, y)}{\int_{-\infty}^{\hat{z}_i(y)} g(z) \beta(z', y) \, dz'} \, dz + B \hat{R} \int_{\hat{z}_i(y)}^{+\infty} g(z) \beta(z, y) \Pi(z) \, dz
\tag{45}
\]
with respect to the (scalar) value of \( \hat{z}_i(y) \) for each \( y \) where a maximum of expression (45) exists. This yields the equation
\[
\Pi(\hat{z}_i(y)) B \hat{R} = \frac{I}{n(\hat{\rho}_m, y) + I}
\tag{46}
\]
where \( n(\hat{\rho}_m, y) \) has the form (37) (with \( \hat{z}_i(y) \) instead of \( \hat{z}_c(y_0) \)). To find the ESS, we equal resident and mutant strategy, and \( I \) assumes the form
\[
I = \int_{-\infty}^{+\infty} \int_{\hat{z}_i(y)}^{+\infty} \phi(y) g(z) s \beta(z, y) \Pi(z) \, dz \, dy.
\tag{47}
\]
Because \( \hat{z}_i(y) \) depends on \( y \) it is impossible to solve (46) for the ESS directly. We therefore use the following algorithm to find the ESS. For a given resident strategy \( p \) we know its corresponding
values \( I \) and \( B \bar{R} \). For a fixed patch \( y_0 \), we then find the value of \( \tilde{z}_i(y_0) \in [y_0 - 3, y_0 + 3] \) such that (46) is satisfied. (If there is no such \( \tilde{z}_i(y_0) \), then \( \tilde{p}_n(z, y_0) = 0 \) for all \( z \in [y_0 - 3, y_0 + 3] \).) This we repeat for a range of values of \( y \). Now we can calculate the new values of \( I \) and \( B \bar{R} \) (according to formulas (47), (10) and (37)) and compare them to the original values of \( I \) and \( B \bar{R} \). An ESS would imply that \( I \) and \( B \bar{R} \) do not change. This is a fixed point problem of a two dimensional discrete time dynamical system, which we solve numerically. Once the ESS values of \( I \) and \( B \bar{R} \) and thus the function \( \tilde{z}_i \) are known, the actual evolutionarily stable dispersal strategy \( \tilde{p} \) follows directly from formula (44). For decreasing II we find the ESS analogously.

### 3.2.1 Numerical Examples

Let the patch quality distribution \( \phi \), the distribution \( \beta \) of offspring body conditions, the weight function \( g \) and the probability to survive until maturity \( s \) be as defined in formulas (30)–(33). When II is increasing with \( z \) as in formula (42), according to the above described algorithm, we find an ESS with \( I_i \approx 1.33 \) and \( B \bar{R}_i \approx 0.54 \). Figure 3(a) shows the isoclines for \( I \) and \( B \bar{R} \), where the arrows indicate the changes in \( I \) and \( B \bar{R} \). The ESS is unique within the domain shown in Figure 3(a). In the case that weak individuals are good dispersers and strong individuals are bad dispersers, such that \( \Pi(z) \) is a decreasing function of body condition as in formula (43), the ESS values of \( I \) and \( B \bar{R} \) are \( I_d \approx 0.54 \) and \( B \bar{R}_d \approx 0.92 \) (Figure 4(a); the subscript \( d \) indicates the case with decreasing II).

Recall that if all individuals survive dispersal with the same probability (Section 3.1), then the
Figure 4: The evolutionarily stable strategy when weak individuals are better dispersers than strong individuals (decreasing $\Pi$). (a) Isoclines of $I$ and $B\tilde{R}$, ESS values at $I_d \approx 0.54$ and $B\tilde{R}_d \approx 0.92$ (initial grid resolution: steps of 0.1 for both $I$ and $B\tilde{R}$). (b) Position of the jump of the step function $\hat{p}$ as a function of patch quality. (c) Plot of $\hat{n}_d$ such that $B\hat{n}_d(y)$ is the weighted sum of non-dispersers in a patch of quality $y$. (d) Expected probability that an individual with given body condition will disperse. (e) Body condition distribution among the dispersers in the whole metapopulation. (f) Body condition distribution among the non-dispersers in the whole metapopulation.

ESS is an equivalence class of dispersal strategies that are neutral against each other. In contrast, if $\Pi$ is not a constant function, then this neutrality is broken and a unique ESS exists.

In both Figure 3 and Figure 4, panels (b) show the position of the jump $\hat{\pi}(y)$ as a function of patch quality, panels (c) show the graphs of $\hat{n}_d$ where $B\hat{n}_d(y)$ is the weighted sum of non-dispersers in a patch of quality $y$, panels (d) show the average probability of dispersal, $E(\hat{p}(z, y)|z)$, as a function of body condition $z$ (cf. formula (39)), panels (e) give the body condition distribution among dispersers (cf. formula (40)) and panels (f) among non-dispersers (cf. formula (41)).

The graphs for increasing $\Pi$ (Figure 3) qualitatively resemble the graphs when $\Pi$ is constant and we assume that only the strongest individuals in a patch disperse (Figure 2(i)), whereas when $\Pi(z)$ is a decreasing function of $z$ (Figure 4) the graphs look alike the graphs for constant $\Pi$ when the weakest individuals disperse from a patch (Figure 2(ii)). The average probability of surviving dispersal with either increasing or decreasing $\Pi$ is $\Pi = 0.5$, which is the same value as in the numerical example in Figure 2. Therefore we can directly compare these cases. At large, the same arguments that explain the qualitative properties seen in Figure 2 with constant $\Pi$ (see Section 3.1) also apply for non-constant $\Pi$: Dispersers emerge only from patches with better quality than a certain threshold $\hat{y}$, because in patches with $y < \hat{y}$ it is best to keep all offspring at home to increase the chance to retain the home patch. From patches with increasing patch quality and thus increasing body condition of local offspring, more and more individuals can disperse while still a proper weight stays to try to retain the patch. From patches with increasing $\Pi$, very strong individuals always disperse as they are the strongest individuals wherever they are born and have the highest probability to survive dispersal. With decreasing $\Pi$, the weakest individuals...
of a given patch disperse, but in the population as a whole, individuals with intermediate body conditions disperse most frequently while the very weak and the very strong individuals stay in the patch.

With non-constant \( \Pi \), the non-dispersing weight in a patch, \( B\hat{n}(y) \), depends on patch quality (panels (c) in Figures 3 and 4), unlike with constant \( \Pi \) where every family (in patches \( y > \hat{y}_i \)) keeps the same weight at home (Figure 2, top panel). With increasing \( \Pi \), \( \hat{n}_i(y) \) decreases for large \( y \) (Figure 3(c)), which is due to the fact that offspring born in good patches are not only good competitors but also good dispersers. With decreasing \( \Pi \), \( \hat{n}_d(y) \) monotonically increases with patch quality (Figure 4(c)) as offspring in good patches are bad dispersers.

Panels (d) – (f) show empirically observable quantities and distributions. The probability of dispersal given the body condition of the offspring, \( \mathbb{E}(\hat{p}(z, y)|z) \) defined in formula (39), is a sharply increasing function of \( z \) when \( \Pi \) is increasing (Figure 3(d)): in this case, the body condition dependence of dispersal is obvious in the population-wide data. In contrast, when \( \Pi \) is decreasing (Figure 4(d)), offspring with a broad range of intermediate body conditions disperse whereas those with extreme body condition do not; this pattern is more difficult to detect in empirical data. Panels (e) and (f) show the distribution of body condition among dispersers (cf. formula (40)) and among non-dispersers (cf. formula (41)), respectively. When \( \Pi \) is increasing (Figure 3(e) – (f)), these distributions have sharp cut-offs (due to the sharp switch in Figure 3(d)) and substantially different means (shown by vertical lines). However, when \( \Pi \) is decreasing, dispersers and non-dispersers have virtually the same distribution (Figure 4(e) – (f)). In empirical data, these distributions would not reveal the fact that dispersal depends on body condition.

Let us compare the case when \( \Pi \) is increasing with case (i) when \( \Pi \) is constant. Here, the reader is reminded of Table 2, which provides an overview of quantities at the ESS for different shapes of \( \Pi \), as we go on to compare these different cases. In both cases the dispersal strategy is a step function with at most one jump from zero to one in the \( z \)-direction for every \( y \), so that the strongest individuals in a patch disperse. When \( \Pi(z) \) is an increasing function of \( z \), altogether slightly more individuals disperse than when \( \Pi \) is constant: The grey area of dispersal in Figure 2(i)(a) is a subset of the grey area in Figure 3(b) as \( \hat{y}_i \approx -1.14 \) is smaller than \( \hat{y}_c \approx -0.84 \), i.e., more patches produce dispersers when \( \Pi \) is increasing, and, in every patch that produces dispersers, \( \hat{z}_i(y) < \hat{z}_c(y) \), i.e., more individuals disperse from a patch when \( \Pi \) is increasing.

The fraction of dispersers is \( D_i \approx 0.24 \) (compared to \( D_c^{(i)} \approx 0.22 \)) and the average weight of individuals dispersing from a patch is \( Bw_i \approx 1.8B \) (compared to \( Bw_c \approx 1.76B \)). Consequently, the average non-dispersing weight in a patch, \( \bar{B}\hat{n}_i \approx 0.54B \), is slightly less than the average non-dispersing weight when \( \Pi \) is constant \( (B\bar{n}_c \approx 0.58B) \). In both cases, all dispersers have positive body condition; since with increasing \( \Pi \), \( \Pi(z) > 0.5 \) for all positive \( z \), the immigrant weight is much higher with increasing \( \Pi \): \( BI_i \approx 1.33B \) compared to \( BI_c \approx 0.88B \). In fact, the fraction of the weighted sum of dispersers that survives dispersal,

\[
\bar{\Pi} = \frac{I}{w},
\]

is \( \bar{\Pi}_i \approx 0.74 \) and thus much higher than when \( \Pi \) is constant where \( \bar{\Pi}_c = 0.5 \). Higher \( I \) affects the probability of establishment after dispersal negatively while smaller \( \hat{n} \) affects this value positively; in our example with increasing \( \Pi \), the value of \( B\bar{R} \) decreases to \( B\bar{R}_i \approx 0.54 \). Note that the high immigrant weight and the small probability of establishment after dispersal select against dispersal, but the increasing function \( \Pi \) for the probability to survive dispersal selects for dispersal.
of strong individuals. At the ESS, we find these selective forces balanced, and, at least in our example, altogether more (strong) offspring disperse.

Next, we compare the case with decreasing $\Pi$ (Figure 4) with the case of constant $\Pi$ with a decreasing step function for the dispersal strategy (Figure 2(ii)). Again, we see altogether more dispersers when $\Pi$ is decreasing than when $\Pi$ is constant ($D_d \approx 0.69$ compared to $D_c^{(ii)} \approx 0.61$). This is due to increased dispersal from low quality patches ($\hat{z}_d(y) > \hat{z}_c(y)$ for $\hat{y}_d \approx -1.61 < y \lesssim -0.14$), which produce weaker dispersers that have a higher probability to survive dispersal ($\Pi(z) > 0.5$ for $z < 0$). In contrast, less individuals disperse from good quality patches ($y > -0.14$). As a result, the average weight dispersed from a patch, $B_{w_d} \approx 1.69B$, is smaller with decreasing $\Pi$ than with constant $\Pi$ ($B_{w_c} \approx 1.76B$), even though the lower dispersed weight corresponds to more dispersing individuals. Moreover, only a fraction $\Pi_d \approx 0.32$ per cent of the dispersed weight survives dispersal, so that the immigrant weight is much smaller ($B_{I_d} \approx 0.54B$) than with constant $\Pi$ ($B_{I_c} \approx 0.88B$). Even though the probability of winning a patch per dispersed weight increases ($B_{R_d} \approx 0.92$ compared to $B_{R_c} \approx 0.71$) and the reduced immigrant weight also selects for dispersing more competitive weight, these two effects are offset by the lower probability of survival during dispersal ($\Pi_d < \Pi_c = 0.5$) so that eventually less weight is dispersed.

4 Discussion

We presented a model for body condition dependent dispersal under kin competition and investigated the effect of within-family variation of body condition on dispersal behaviour. Applying the theory of adaptive dynamics, which determines the fate of an initially rare mutant or invader within an otherwise monomorphic resident population at dynamical equilibrium, we determined evolutionarily stable dispersal strategies (ESSs) under different model assumptions. Competitive ability depends on individual body condition such that stronger individuals win competition with higher probability than weak individuals. Competition is modelled by a weighted lottery, the most common way to model asymmetric competition.

When the probability of survival during dispersal, $\Pi$, is independent of the body condition of the disperser (Section 3.1), the evolutionarily stable strategy is characterized by the weighted sum of non-dispersing individuals in a patch: a fixed weight of offspring $\hat{N}$ stays in the patch and the excess weight disperses, where $\hat{N}$ is independent of patch quality. (If the weighted sum of all offspring born in a patch does not achieve $\hat{N}$, then all offspring stay in the patch.) This is analogous to the result of Ezoe and Iwasa [9] and of Kisdi [20] who showed that of identical offspring, a fixed number should stay in the natal patch. We assume variable body condition, and hence can ask which offspring should disperse. Remarkably, it does not matter how $\hat{N}$ or how the dispersed competitive weight is compounded, i.e., which individuals with which body condition contribute to it: all strategies with equal $\hat{N}$ form an equivalence class and are neutral to each other. This implies that e.g. a strategy where only weak individuals disperse is as evolutionarily stable as another strategy with only strong dispersers.

This finding is highly significant for interpreting observations in nature and should help explaining the seemingly contradictory or random dispersal patterns found in empirical studies. Field biologists need not necessarily be puzzled when monitoring one species and observing strong dispersers in one population but many more weak dispersers in another population living in a similar environment (see Figure 2(i) – (ii)). These patterns may represent alternative strategies
from the same ESS equivalence class. For instance, the authors of [33] and [34] studied different aspects of dispersal of roe deer populations. In [34], dispersers were found to be heavier than non-dispersers, but in [33], dispersers did not significantly differ. Even different families (patches) of the same population can decide arbitrarily which individuals shall disperse as long as the fixed weight $\hat{N}$ stays in the patch; i.e., strategies like in Figure 2(i) and (ii) (and many others) may occur in the same population.

This feature that a certain weighted sum of individuals stays in their natal patch and the rest disperse arises already in our previous model [16] with lottery competition: Example 3.1 in [16] is a special case of the present model with $\beta(z, y) = \delta(z - y)$ (i.e., all offspring born in one patch have the same body condition as the environmental quality of the patch). With constant $\Pi$ and $\beta(z, y) = \delta(z - y)$, the necessary condition (11) that a mutant’s best reply strategy $\hat{p}_m$ to a given resident strategy has to satisfy yields a unique solution for $\hat{p}_m$ rather than an equivalence class of strategies like when $\beta$ is a distribution.

With the assumption that individual body condition affects survival during dispersal (Section 3.2), the mathematical structure of the fitness functional does not allow anything else than a step function for the ESS. The evolutionarily stable probability of dispersal, $\hat{p}$, has at most one jump in the $z$-direction for every $y$. When strong individuals survive dispersal better than weak individuals ($\Pi(z)$ is an increasing function of body condition) then $\hat{p}$ is an increasing step function with respect to $z$ such that the strongest individuals of a patch, who have the highest probability of surviving dispersal, will disperse and the weak stay. For the whole metapopulation, there is practically a threshold body condition such that individuals with a body condition above this threshold disperse whereas individuals below do not (see Figure 3(d)). When weak individuals are better dispersers ($\Pi(z)$ is decreasing with body condition), then the weakest individuals disperse from each patch; but patches of higher quality disperse more individuals, so that eventually individuals with a wide range of body conditions disperse (Figure 4(d)). This means that an observer who only studies the distribution of body conditions among dispersers and non-dispersers does not discover the actual dispersal rules that apply in the individual patches. For example in Figure 4(c) – (f), there is hardly any difference between the distribution of dispersers and non-dispersers; one might conclude that body condition has no effect on dispersal, even though in reality only the weaker members of each family disperse. The dispersal behaviour of individuals and families can be extremely hard to measure and detect in the field. Nevertheless, our model advises that dispersal behaviour of families could be studied concerning the questions which individuals within a family disperse and whether families of strong offspring disperse more.

To return to the question we asked in the Introduction of this article, which individuals within a family disperse, we can now draw the main conclusion that individuals disperse who are more apt for dispersal, no matter how well they will perform during competition. When strong individuals are better dispersers then the strong disperse; when weak individuals are better dispersers then the weak disperse (although more individuals disperse when the weak in each family are dispersing and therefore also some relatively strong individuals disperse). This is true although the strong are always better competitors. Since we find that every evolutionarily stable strategy includes that a certain weight stays in a patch, individuals with higher probability to survive dispersal are sent away. Plants with heteromorphic seeds are examples for this finding (see e.g. [6] where dispersing seeds are heavier than non-dispersing seeds). Further, in [24], faster-running male neonates disperse further in a lizard species, and in [4], larger larvae disperse more frequently as they may be better adapted for dispersal. When survival during dispersal is independent of body condition we cannot predict which individuals disperse, as the evolutionarily stable strategy only
DISCUSSION

requires that a certain weight stays and defends the patch.

Throughout this article we assumed that a weighted lottery determines competition. With this, there exists a simple exchange such that few strong individuals are equivalent to many weak individuals. Thus, the key quantities of this model are really weighted sums of individuals. This is represented strikingly in the case when all individuals survive dispersal with the same probability (Section 3.1). At the ESS, infinitely many different dispersal strategies exist that all have in common that a certain weight stays in a patch and the excess weight disperses while it does not matter whether many weak individuals or few strong individuals stay in a patch. With other competition mechanisms, results may lack this feature.

Acknowledgements

This research was financially supported by the Graduate School in Computational Biology, Bioinformatics and Biometry (ComBi) of the Finnish Ministry of Education and by the Finnish Centre of Excellence in Analysis and Dynamics Research of the Academy of Finland. We thank two anonymous reviewers for valuable comments on the manuscript.

Appendix

In this Appendix, we consider a non-constant function \( \Pi \) and prove that the best reply mutant strategy in a patch of quality \( y \) is either \( \hat{p}_m(\cdot, y) \equiv 0 \) (no offspring disperse from a patch of quality \( y \)) or a step function of \( z \) with a single jump from 0 to 1 (respectively from 1 to 0) at some position \( \hat{z}(y) \) if \( \Pi(z) \) is monotonically increasing (decreasing) with \( z \). We make two technical assumptions: Firstly, we consider only piecewise continuous functions for \( \hat{p}_m \). Because the mutant’s fitness is a functional (formula (1)), changing \( \hat{p}_m(z, y) \) at a set of points of zero measure does not change fitness and therefore infinitely many selectively equivalent strategies exist; but biologically these are not relevant. Secondly, we assume for simplicity that \( \beta(z, y)g(z) > 0 \) for all \( z \) and \( y \). If this is not the case, i.e., if offspring with some body conditions are not produced \( (\beta(z, y) = 0) \) or always die during competition \( (g(z) = 0) \), then one can narrow the set of admissible \( z \) to exclude these; obviously, strategies that differ only for individuals not produced or not viable are selectively neutral. Further, we shall consider only the case of a monotonically increasing function \( \Pi \); for decreasing \( \Pi \), the proof is analogous.

Because with a non-constant function \( \Pi \), Euler’s equation (15) is not satisfied, the maximum must be on the boundary so that the best reply mutant strategy must be of the form

\[
\hat{p}_m(z, y) = \begin{cases} 
1 & \text{if } z \in U(y) \\
0 & \text{otherwise}
\end{cases}
\]

for some \( U(y) \subset \mathbb{R} \) (because \( p_m(\cdot, y) \equiv 1 \) is never a best reply for any \( y \), \( U(y) \) is always a proper subset of \( \mathbb{R} \)). If \( \hat{p}_m(\cdot, y) \equiv 0 \) is the best reply, then \( U(y) \) is the empty set; our claim is that otherwise, the best reply is a step function characterized by \( U(y) = (\hat{z}(y), \infty) \) for some finite \( \hat{z}(y) \) (the position of the jump). Suppose, to reach a contradiction, that there is a best reply strategy \( \hat{p}_m \) such that in a set of patch qualities \( y \) of non-zero measure, \( U(y) \) is neither empty nor
of the form \((\hat{z}(y), \infty)\). From formulas (1) and (2), the invasion fitness of strategy \(\hat{p}_m\) is given by
\[
W(\hat{p}_m) = \int_{-\infty}^{+\infty} \phi(y) s \left( B \hat{R} \int_{\bar{U}(y)} g(z) \beta(z, y) \Pi(z) \, dz + B \hat{P}_m(n(\hat{p}_m, y)) \int_{\bar{R}\backslash\bar{U}(y)} g(z) \beta(z, y) \, dz \right) \, dy
\]
(50)
where the second term in the brackets can be rewritten to obtain
\[
W(\hat{p}_m) = \int_{-\infty}^{+\infty} \phi(y) s \left( B \hat{R} \int_{\bar{U}(y)} g(z) \beta(z, y) \Pi(z) \, dz + B \hat{P}_m(n(\hat{p}_m, y)) n(\hat{p}_m, y) \right) \, dy. \quad (51)

Consider now the mutant strategy \(\hat{p}_m\) such that \(\hat{U}(y) = (\hat{z}(y), \infty)\) and \(n(\hat{p}_m, y) = n(\hat{p}_m, y)\) for all \(y\); strategy \(\hat{p}_m\) keeps the same competitive weight in every natal patch as \(\hat{p}_m\), but composes it differently such that from each patch, the strongest individuals \((z > \hat{z}(y))\) disperse. Define \(Y(p_m) = \{y : U(y) \neq \emptyset\}\) to be the set of patch qualities \(y\) of patches from where some individuals disperse; obviously, \(Y(\hat{p}_m) = Y(\hat{p}_m)\). For every \(y \in Y(\hat{p}_m), \hat{z}(y)\) exists and is unique. Expressing the invasion fitness of \(\hat{p}_m\) analogously to formula (51), we obtain
\[
W(\hat{p}_m) - W(\hat{p}_m) = \int_{\hat{Y}(\hat{p}_m)} \phi(y) s \left( B \hat{R} \int_{\bar{U}(y)} g(z) \beta(z, y) \Pi(z) \, dz - \int_{\bar{U}(y)} g(z) \beta(z, y) \Pi(z) \, dz \right) \, dy.
\]
(52)

Since \(\int_{\bar{U}(y)} \beta(z, y) g(z) \, dz = A(y) - n(p_m, y)\) (where \(A(y)\) is defined in formula (20a) such that \(B A(y)\) is the total competitive weight of offspring born in a patch of quality \(y\), the assumption \(n(\hat{p}_m, y) = n(\hat{p}_m, y)\) implies that \(\int_{\hat{U}(y)} \beta(z, y) g(z) \, dz = \int_{\bar{U}(y)} \beta(z, y) g(z) \, dz\) (the two strategies disperse the same total competitive weight). Because \(\beta\) and \(g\) are positive and \(\Pi\) is increasing by assumption, the value of the bracket in formula (52) is positive for every \(y \in Y(\hat{p}_m)\). Since \(Y(\hat{p}_m)\) has positive measure by the definition of \(\hat{p}_m\), this means that \(\hat{p}_m\) has higher fitness than \(\hat{p}_m\), i.e., strategy \(\hat{p}_m\) cannot be a best reply. This proves that for almost all \(y\), the best reply strategy in a patch of quality \(y\) must either be \(\hat{p}_m(\cdot, y) \equiv 0\) or the step function given in formula (44) in the main text.

References

REFERENCES


REFERENCES


Article III

With minor modifications adapted from

Submitted manuscript,
Body condition dependent dispersal in a heterogeneous environment,
Mats Gyllenberg, Éva Kisdi, Margarete Utz.
Abstract

We find the evolutionarily stable dispersal behaviour of a population that inhabits a heterogeneous environment where patches differ in safety (the probability that a juvenile individual survives until reproduction) and productivity (the total competitive weight of offspring produced by the local individual), assuming that these characteristics do not change over time. Offspring body condition varies within and between families. Offspring compete for patches in a weighted lottery such that dispersal is driven by kin competition. Survival during dispersal may depend on body condition, and competitive ability increases with increasing body condition.

The evolutionarily stable strategy predicts that patches are abandoned if they are too unsafe and / or have too low productivity. From families that invest in retaining their natal patches, individuals stay in the patch that are less suitable for dispersal whereas the better dispersers disperse. However, this clear within-family pattern is often not reflected in the population-wide body condition distribution of dispersers or non-dispersers. This may be an explanation why empirical data do not show any general relationship between body condition and dispersal.

When all individuals are equally good dispersers, then there exist equivalence classes defined by the competitive weight that remains in a patch. An equivalence class consists of infinitely many dispersal strategies that are selectively neutral. This provides an explanation why very diverse patterns found in body condition dependent dispersal data can all be equally evolutionarily stable.

Keywords: adaptive dynamics · body condition dependent dispersal · evolution · ESS · function-valued trait · kin competition · spatially structured population

Mathematics Subject Classification (2000): 92D15 · 92D40

1 Body Condition Dependent Dispersal

Body condition dependent dispersal is a widespread phenomenon among many kinds of animal and plant species. Dispersers often differ in morphology or behaviour from non-dispersers: In [2, 16, 19, 20], the authors found that dispersers were heavier or larger in an insect, reptile, bird, respectively mammal species; the authors of [6, 7, 18] observed that dispersers were less aggressive, smaller and less fecund, or lighter in a mammal, insect, respectively bird species, to name but a few examples from the vast amount of empirical studies on body condition dependent dispersal.

The mechanisms underlying body condition dependent dispersal are manifold and difficult to survey. For instance, if increased aggression or competitive ability comes along with larger body mass or size, then heavier or larger individuals might be more suitable to survive the risky
phase of dispersal [19]. But they might as well play out their strength in the natal environment and suppress weaker conspecifics and provoke the emigration of weaklings (social dominance hypothesis [5]; see examples in [4, 6, 12, 18]). On the other hand, lighter or smaller individuals might just as well be the better dispersers if, e.g., their body condition implies higher agility that helps to escape predators [15, 21], or if increased flight capacity is negatively correlated with body weight [7].

In [8, 9] we developed a model where the probability that offspring disperse from their natal patch is a function of individual body conditions. In a patchy heterogeneous environment, offspring body conditions are distributed around the natal patch quality such that individual body condition varies between families living in different patches as well as within families (the latter only in [9]). Offspring compete for sites such that kin competition selects for dispersal (as in the model by Hamilton and May [11]). Competitive ability increases with body condition. Whether an individual disperses or stays in its natal habitat patch depends on its own and its siblings’ body conditions as well as the distribution of body conditions among immigrants. We investigated which individuals within a family (only in [9]) and in the population as a whole disperse.

Because the environmental quality of natural habitats fluctuates temporally and spatially, in [8, 9] we made the assumption that patch qualities (productivity) are randomly reassigned every year. Under this assumption a family does not know what quality the natal patch will have in the next year and therefore what body conditions the offspring in the next year will have in case one current offspring can retain the patch. Therefore, every patch is equally worth keeping. Under the assumption of weighted lottery competition, and with constant survival probability during dispersal, every family retains the same competitive weight in the patch ([8](Example 3.1), [9]). If the total competitive weight of a family is sufficiently large that dispersing some competitive weight is more beneficial than keeping more weight in the patch, then this excess weight disperses. In other words, in patches of low quality, where mainly weak individuals are born, all offspring stay and defend the patch, whereas families in patches with higher productivity invest also in dispersal. The dispersed competitive weight increases with natal patch quality. When survival during dispersal depends on body condition, then the balance between the benefit of keeping more weight in the patch and the benefit of dispersing more weight depends on survival during dispersal. Then, within a family, individuals with the highest probability to survive dispersal disperse while individuals less suitable for dispersal stay and defend their natal patch (even if this strategy implies that the competitively weaker individuals disperse).

In this article, we assume that patch qualities are fixed in time. In reality, environmental fluctuations are often temporally correlated so that the assumption of a fixed environment is closer to reality than the temporally varying environment in [8, 9]. With permanent differences, patches are no longer equally worth keeping. Furthermore, we assume that patches may differ also in the probability that an individual established in a patch reaches maturity. We find the evolutionarily stable dispersal strategy in a fixed heterogeneous environment and compare the results with the case of a randomly fluctuating environment. We also discuss how our results may interpret the empirical data.

2 The Resident Population

We assume a heterogeneous environment where patches may differ in several respects such as resource abundance or predator presence. Every patch is labelled by a vector $y = (y_1, \ldots, y_n)^T \in \mathbb{R}^n$. The vector $y$ represents the quality of the habitat patch and can be used to describe various factors such as resource abundance, predator presence, or other environmental conditions.
that contains all patch characteristics and is constant in time. We denote the set of all feasible character vectors $y$ by $Y$, and denote the probability density of patches of character $y$ by $\phi(y)$ such that $\int_Y \phi(y) \, dy = 1$. There are infinitely many patches. In this paper, we assume that patches differ in safety (which affects the survival probability of a juvenile individual until maturation) and in productivity (the body conditions of offspring produced by the local inhabitant). Every patch can sustain at most one individual. Generations of the model species are non-overlapping, and at the beginning of every year, every patch is occupied by exactly one juvenile individual. Let $s(y)$ be the probability that an individual living in a patch $y$ survives until maturation and produces offspring. Patches with a high value of $s(y)$ are therefore ‘safe’ patches and patches with a low value of $s(y)$ are ‘unsafe’ patches. When an individual survives until reproduction, it produces $B$ offspring. We will take the limit $B \to \infty$, so that, in conjunction with the infinite number of patches, the model will be deterministic. The offspring have different body conditions $z \in \mathbb{R}$ such that there is variability between patches and within each family. $\beta(z, y)$ is the probability density of offspring with body condition $z$ born in a patch $y$. Individual body condition will influence competitive ability such that offspring with a high value of $z$ are ‘strong’ and have a high probability to win competition, whereas individuals with a low value of $z$ are ‘weak’ and win competition with a low probability. After reproduction, the parent immediately dies.

Patches are connected through global dispersal. An individual with body condition $z$ born in a patch $y$ disperses with probability $p(z, y)$. This is the evolving trait and we aim to find dispersal strategies $\hat{p}$ that are evolutionarily stable. A disperser survives dispersal with probability $\Pi(z)$. Dispersal survival $\Pi$ will be constant in Section 4 such that all individuals are equally good dispersers. In Section 5, $\Pi$ will be a monotonically increasing respectively decreasing function of body condition such that strong respectively weak individuals are better dispersers. Strong, e.g. fat and heavy, individuals can have advantages during starving periods and fights that occur during dispersal [19]. On the other hand, weak, e.g. small and light, individuals can often escape predators more agilely [15, 21]. A surviving disperser immigrates into a random patch.

Within each patch, immigrants compete with local non-dispersers (if present) and exactly one individual establishes itself in the patch. We will use a weighted lottery competition model where every individual is assigned a weight $g(z)$ according to its body condition. The function $g$ increases monotonically with $z$ such that competitive ability increases with body condition. (The life cycle is similar to the model in [9], except that here we assume that the ecological properties of a patch ($y$) remain fixed in time, and the probability of survival till maturity ($s$) may depend on the properties of the patch.)

In this deterministic model where individual body conditions are solely determined by the environment, the resident population is always in dynamical equilibrium. Every year at the time of reproduction, the number of offspring with certain body condition born in certain patches is the same no matter what dispersal strategy the resident plays (as long as the probability of dispersal is positive, otherwise the metapopulation will eventually die out).

3 The Mutant Fitness

Assume that a mutation occurs within the resident population in an infinitesimal fraction of patches such that, at the beginning of a year, these patches are occupied by mutant individuals. The mutant differs from the resident only in its dispersal behaviour: a mutant offspring with body condition $z$ that is born in a patch $y$ disperses with probability $p_m(z, y)$. In all other respects, the
resident and mutant population dynamics are identical, and the resident dynamics are not affected by the presence of a rare mutant.

Because mutants are rare we neglect the possibility that more than one mutant disperser immigrates into the same patch and competes against other mutant immigrants. A focal mutant immigrates with probability $s(y)$ into a patch where the individual established at the beginning of the year survived and produced offspring. There it competes against local non-dispersers and other immigrants. Let $Bn(p, y)$ be the weighted sum of non-dispersing individuals in a patch $y$, where

$$n(p, y) = \int_{-\infty}^{+\infty} \beta(z, y) g(z) (1 - p(z, y)) \, dz,$$

and let $BI$ be the weighted sum of immigrants coming into a patch, where

$$I = \int_{-\infty}^{+\infty} \int_{-\infty}^{+\infty} \phi(y) s(y) \beta(z, y) g(z) p(z, y) \Pi(z) \, dz \, dy,$$

which is the same in every patch. The probability that a mutant with body condition $z$ wins competition in such a patch is thus its own weight $g(z)$ divided by the total weight of all competitors,

$$\frac{g(z)}{B(n(p, y) + I)}.$$

With probability $1 - s(y)$ a mutant immigrates into a patch where the established individual at the beginning of the year did not survive so that there are no local non-dispersers but only immigrants to compete with. There the mutant wins competition with probability

$$\frac{g(z)}{BI}.$$

The probability that one of the non-dispersing mutant offspring wins competition and thereby the mutant family retains the natal patch $y$ is

$$s(y) \frac{Bn(pm, y)}{B(n(pm, y) + I)}.$$

The number of years a patch $y$ is retained by a descendant of the local family follows thus a geometric distribution with expectation

$$T(y, n(pm, y)) = \frac{1}{1 - s(y) \frac{Bn(pm, y)}{B(n(pm, y) + I)}}$$

$$= \frac{n(pm, y) + I}{(1 - s(y)) n(pm, y) + I}.$$

$T(y, n(pm, y))$ is thus the expected lifetime of a mutant family in a patch $y$.

Following the theory of structured metapopulations as outlined in Gyllenberg and Metz [10], we consider a generation expansion based on count in the dispersal pool. Let $M(z)dz$ be the number of mutant offspring with body condition $z$ who have dispersed, survived dispersal and are now leaving the dispersal pool and immigrating into patches. Let $W : \mathbb{R}^+_0 \rightarrow \mathbb{R}^+_0$ be the next generation operator that maps the present number of immigrating mutants $M(z)dz$ onto
the number \( M'(z')dz' \) of their descendants with body condition \( z' \) that disperse and survive dispersal during the lifetime \( T(y, n(p_m, y)) \) of their families. The resulting structured population model is thus given by

\[
M'(z')dz' = (WM)(z')dz' = \int_{-\infty}^{+\infty} M(z) \int_{Y} \phi(y) \left( \frac{s(y)g(z)}{B(n(p, y) + I)} + \frac{1 - s(y)}{BI} \right) \cdot s(y) B \beta(z', y) p_m(z', y) \Pi(z') T(y, n(p_m, y)) dz' dy dz.
\]

We can rewrite (7) in the form

\[
\int_{-\infty}^{+\infty} M'(z) g(z) dz = R_0(p_m) \int_{-\infty}^{+\infty} M(z) g(z) dz
\]

where \( \int_{-\infty}^{+\infty} M(z) g(z) dz \) is the total competitive weight of mutants and

\[
R_0(p_m) = \int_{Y} \phi(y) \left( \frac{s(y)}{B(n(p, y) + I)} + \frac{1 - s(y)}{BI} \right) \cdot s(y) B \left( \int_{-\infty}^{+\infty} \beta(z', y) g(z') p_m(z', y) \Pi(z') dz' \right) T(y, n(p_m, y)) dy
\]

is the spectral radius of the next generation operator \( \mathcal{W} \), i.e., the basic reproduction number of the gross mutant competitive weight. It is a functional that depends on the mutant strategy \( p_m \). Here, we exploit a special property of competition by weighted lottery in an essential way. In a weighted lottery, competitive ability (body condition) is exchangeable with the number of competing individuals so that many weak individuals compete as successfully as few strong ones. This exchangeability makes it possible to reduce the mutant dynamics to a single dimension, the generation expansion of total mutant competitive weight (‘weighted sum’) leaving the dispersal pool.

\( R_0(p) = 1 \) because the resident is in equilibrium, and if the resident is at the ESS then \( R_0(p_m) < 1 \) for all \( p_m \neq p \). A mutant’s best reply strategy \( \hat{p}_m \) to a given resident strategy \( p \) is one that maximizes \( R_0(p_m) \). We aim to find the evolutionarily stable strategy (ESS), that is, the strategy \( \hat{p} \) such that \( \hat{p}_m = \hat{p} \), i.e., the strategy that is the best reply to itself. Note that the best reply in our case maximizes \( R_0 \) and not the growth rate \( r \) (the Malthusian parameter, which is hard to calculate), but because \( R_0 > 1 \) (\( R_0 < 1 \)) is equivalent to \( r > 0 \) (\( r < 0 \)), this leads to the same result.

All quantities in formula (10) are positive; further, \( \phi, s, B, \beta, \Pi \) and \( g \) are given model ingredients, and \( I \) depends only on the resident dynamics. Therefore, in order to maximize \( R_0 \), we need to maximize the quantity

\[
T(y, n(p_m, y)) \int_{-\infty}^{+\infty} \beta(z, y) g(z) p_m(z, y) \Pi(z) dz
\]

for almost every \( y \). Note that the best reply can deviate from maximizing (11) on a set of points \( y \) of zero measure. The method how we will solve this optimization problem depends on the nature of the function \( \Pi \). Section 4 deals with the model for constant \( \Pi \), Section 5 for monotone \( \Pi \).
4 Case I. Constant Π All individuals are equally good dispersers

When Π is constant such that every disperser survives with the same probability regardless of its body condition, then we can simplify the quantity in (11) by rewriting it in terms of the weighted sum of non-dispersers only and hence eliminate the actual dispersal strategies \( p \) and \( p_m \) from the formula (cf. (1)). With a slight abuse of notation, let \( n(y) = n(p, y) \) denote the non-dispersing weight in a resident patch \( y \), and similarly let \( n_m(y) \) be the shorthand notation for \( n(p_m, y) \). Expression (11) becomes then

\[
T(y, n_m(y)) \Pi (A(y) - n_m(y)) = \Pi (A(y) - n_m(y)) \frac{n_m(y) + I}{(1 - s(y)) n_m(y) + I},
\]

where

\[
A(y) = \int_{-\infty}^{+\infty} \beta(z, y) g(z) \, dz
\]

such that \( BA(y) \) is the weighted sum of all offspring born in a patch \( y \), and \( I \) simplifies to (cf. (2))

\[
I = \Pi \int_Y \phi(y) s(y) \left( A(y) - n(y) \right) dy.
\]

Via the total competitive weight \( A(y) \) produced in a patch we can define ‘poor’ (patches with a low value of \( A(y) \)) and ‘rich’ patches (where \( A(y) \) is high).

Any dispersal strategy and hence also the evolutionarily stable strategy is now characterized solely by the weight of non-dispersers \( n \) in a patch rather than by the actual probability of dispersal \( p \). There exist equivalence classes of strategies defined by the non-dispersing weight. The ESS is the equivalence class of strategies that yield the ESS non-dispersing weight \( \hat{n} \). There are infinitely many such strategies and they are selectively neutral against each other. This implies that it can be practically very difficult to detect the actual dispersal behaviour in the field. Two families within a metapopulation that live in similar environmental conditions could show seemingly contradictory dispersal behaviour. For instance, one family would keep the weakest offspring in the patch and send away the strongest while another family would retain the strongest and disperse the weakest. Yet both families follow the same underlying rule to keep the same weight in the patch and disperse the rest. Similarly, it may be impossible to extrapolate dispersal behaviour from one metapopulation to another metapopulation of the same species in a similar environment and to make any predictions about body condition dependent dispersal.

Expression (12) has two extrema at

\[
n_{m}^{(1,2)}(y) = \frac{-I \pm \sqrt{s(y)I (1 - s(y)) A(y) + I}}{1 - s(y)}.
\]

\( n_{m}^{(2)} \) with the minus sign in the numerator is always negative and always minimizes (12), while \( n_{m}^{(1)} \) with the plus sign always yields a maximum of (12). \( n_{m}^{(1)}(y) \leq A(y) \) for all \( y \) and assumes positive values if and only if

\[
s(y) A(y) > I.
\]

In patches \( y \) for which \( s(y) A(y) < I \), (12) attains its maximum for a negative value of \( n_{m}(y) \). However, only values of \( n_{m}(y) \in [0, A(y)] \) are biologically sound, and therefore the best reply
strategy of the mutant in these patches is to disperse all offspring such that \( n_m(y) = 0 \). Patches that are too unsafe (low value of \( s(y) \)) or have too low productivity (low value of \( A(y) \), i.e., low total competitive weight produced) will therefore be abandoned by all best reply strategies, and therefore also by the ESS (which is the best reply to itself). Appendix A shows how the shape of \( n_m^{(1)} \) depends on \( s \) and \( A \). When \( y \) changes such that both \( s(y) \) and \( A(y) \) increase (decrease), then \( n_m^{(1)}(y) \) increases (decreases) as well. Only when either \( s(y) \) or \( A(y) \) increases and the other decreases, \( n_m^{(1)} \) possibly has extrema. (See the numerical examples below for illustration.)

To find the ESS, we need to solve the following fixed point problem for \( I \). Given a value for \( I \) which is set by the resident only, we calculate the mutant’s best reply strategy \( \hat{n}_m(y) \) for every \( y \) according to the formula for \( n_m^{(1)}(y) \) in (15) together with (16),

\[
\hat{n}_m(y) = \begin{cases} 
-\frac{I + \sqrt{s(y)I((1-s(y))A(y)+I)}}{1-s(y)} & \text{if } s(y)A(y) > I, \\
0 & \text{otherwise.}
\end{cases} \tag{17}
\]

Then we assume this strategy as the new resident strategy and calculate the new value for \( I \) (combine (14) and (17)),

\[
I_{next}(I) = I_{max} - I \int_{\hat{Y}(I)} \frac{\phi(y)s(y)}{1-s(y)} \left( \sqrt{s(y)I((1-s(y))A(y)+I-I)} \right) dy, \tag{18}
\]

where

\[
I_{max} = I \int_{Y} \phi(y) s(y) A(y) dy \tag{19}
\]

is the maximal weighted sum of immigrants per patch, which occurs if all offspring in the whole population disperse (when \( n(y) = 0 \) for all \( y \)). The subset \( \hat{Y}(I) \subseteq Y \) is the set within which condition (16) is satisfied; it depends on \( I \).

Because the ESS is the best reply to itself, at the ESS, the value of \( I \) does not change and the ESS value \( \hat{I} \) is the fixed point of (18). There always exists a unique ESS; the proof is outlined in Appendix B.

### 4.1 Numerical Examples for constant \( \Pi \)

With the above algorithm we have numerically calculated the ESS for the following assumptions. We assume a one-dimensional continuum of patch qualities, which follow a truncated standard normal distribution, such that \( y \in [-3, 3] \) and

\[
\phi(y) = \begin{cases} 
\frac{1}{\sqrt{2\pi}}e^{-y^2/2} & \text{for } y \in [-3, 3], \\
\int_{-\infty}^{y} \frac{1}{\sqrt{2\pi}}e^{-x^2/2} dx & \text{otherwise.}
\end{cases} \tag{20}
\]

Similarly, offspring body condition in a patch follows a truncated normal distribution around the patch quality such that offspring in a patch of quality \( y \) feature body conditions \( z \in [y-3, y+3] \),

\[
\beta(z, y) = \begin{cases} 
\frac{1}{\sqrt{2\pi}}e^{-\frac{(z-y)^2}{2}} & \text{for } z \in [y-3, y+3], \\
\int_{y-3}^{y+3} \frac{1}{\sqrt{2\pi}}e^{-\frac{(z'-y)^2}{2}} dz' & \text{otherwise.}
\end{cases} \tag{21}
\]
In the examples, patch quality $y$ is thus defined as the mean of $\beta$, and the variance of $\beta$ is constant. With these assumptions, the total weight produced in a patch, $A(y)$, increases with increasing $y$ and high values of $y$ correspond to rich patches and low $y$ to poor patches. Rich patches yield mainly strong offspring and in poor patches mainly weak offspring are produced. For the survival probability until maturation we choose the function

$$s(y) = s_1 + \frac{s_2 - s_1}{1 + e^{-2y}}.$$  \hspace{1cm} (22)

The survival probability during dispersal is $\Pi = 0.5$ and the weight function for the weighted lottery competition is

$$g(z) = e^z.$$  \hspace{1cm} (23)

**Example with constant $s$: All patches are equally safe** When $s = 0.5$ is constant, i.e., all patches are equally safe, then, at the ESS, the immigrant weight is $B\hat{I} \approx 0.58B$. The inequality in (16) is satisfied for $y > \hat{y} \approx -0.33$ (where $sA(\hat{y}) = \hat{I}$), such that only families in these patches keep a positive weight $\hat{n}(y)$ of their offspring at home. From patches with $y < \hat{y}$, all offspring disperse. The ESS $\hat{n}$ is shown in the left panel of Figure 1. In this case, environmental patch quality has an effect only on offspring body condition, and individual body condition affects only competitive ability (in a positive manner via the weight function $g$). Individuals born in poor patches ($y < \hat{y}$) all disperse because their patches are neither particularly safe nor have a high productivity in the sense that offspring born there will always be competitively weak. Conversely, individuals in a rich patch are interested in retaining the patch because then their descendants in the next year will be strong as well. They are able to keep a large weight in the patch as well as disperse a large weight due to the high total weight of offspring produced. With increasing patch quality, $\hat{n}(y)$ increases as patches become more valuable (i.e., increase in productivity).

**Examples with increasing $s$: Rich patches are also safe patches** When $s$ is a monotonically increasing function of patch quality, then rich patches are also safe and poor patches are unsafe, i.e., patch quality has a stronger influence on the success of the local family than in the previous example. The middle panel of Figure 1 shows the ESS $\hat{n}$ for three functions $s$ with increasing steepness (from the long-dashed to the short-dashed to the dotted line, the corresponding function $s$ has a wider range and higher slope at every $y$). The stronger $s$ increases with patch quality, the more competitive weight families in rich patches invest in retaining the natal patch because the natal patch is not only rich but also increasingly safe.

**Examples with decreasing $s$: Poor patches are safe and rich patches are unsafe** When $s$ decreases with patch quality such that poor patches are safe and rich patches are unsafe, then, compared to the previous two examples, the extreme advantages of individuals born in rich patches are dampened. Recall that only with decreasing $s$ it is possible that $\hat{n}$ is non-monotonic (see Appendix A). The right panel of Figure 1 shows the ESS $\hat{n}$ for three functions $s$ whose graphs are mirror images (mirrored at the ordinate) of the graphs of the functions used in the example with increasing $s$.

When $s$ decreases only mildly (long-dashed line), then the decreased safety in rich patches does not affect the qualitative dispersal behaviour in any patch. $\hat{n}$ is a monotonically increasing function of patch quality as in all previous examples, and the richest patches are still the most
valuable. But note that families in patches that have intermediate quality put now more effort into retaining their natal patch due to the increased safety in these patches compared to the example with increasing $s$. The weight kept in a patch of intermediate quality ($y < 0.21$) is higher than with increasing $s$. However, in richer and hence less safe patches ($y > 0.21$), the non-dispersing weight is less than with increasing $s$. (Note the different scale in the panel with decreasing $s$.)

When safety decreases strongly with $y$ (dotted line), then only families in patches of intermediate quality, which are both reasonably rich and safe, strive for retention of the home patch. Very safe patches are too poor ($y < \hat{y}_1 \approx -1.79$), and very rich patches are too unsafe to keep ($y > \hat{y}_2 \approx 1.79$). In fact, the richest patches are virtually lethal, e.g. $s(3) = 0.0025$ (for $s_1 = 1$, $s_2 = 0$; cf. (22)).

The short-dashed line represents an intermediate case where the drawbacks of decreasing safety in increasingly rich patches are clearly felt and $\hat{n}(y)$ decreases for $0.14 < y < 1$. However, this effect is overpowered in extremely rich patches such that $\hat{n}(y)$ increases again for $y > 1$.

5 Case II. Monotone $\Pi$. Individuals differ in dispersal ability

Let the probability to survive dispersal now depend on body condition. We assume that $\Pi(z)$ either monotonically increases or decreases with body condition $z$. In order to maximize the expression in (11) with respect to $p_m$, note that both the integral in (11) and $T$ (via $n$) depend on $p_m$. We therefore first search for $\hat{p}_m(\cdot, y)$ that maximizes (11) for a given $y$ and additionally satisfies the condition

$$n(\hat{p}_m, y) = N_m(y).$$

(24)

Later we would maximize (11) with respect to $N_m(y)$ (but we will see soon that this step is unnecessary). With the method of Lagrange multipliers, $\hat{p}_m(\cdot, y)$ must therefore maximize the
functional

\[ L(y, p_m, N_m, \lambda) = \int_{-\infty}^{+\infty} T(y, N_m(y)) \beta(z, y) p_m(z, y) \Pi(z) g(z) \, dz + \lambda \left( n(p_m, y) - N_m(y) \right), \]

where \( \lambda \) is a Lagrange multiplier, or equivalently, \( \hat{p}_m \) must maximize

\[ L(y, p_m, N_m, \lambda) + \lambda N_m(y) = \int_{-\infty}^{+\infty} F(z, y, p_m, N_m, \lambda) \, dz, \quad (25) \]

where

\[ F(z, y, p_m, N_m, \lambda) = T(y, N_m(y)) \beta(z, y) p_m(z, y) \Pi(z) g(z) + \lambda \beta(z, y) (1 - p_m(z, y)) g(z). \quad (26) \]

According to the calculus of variations, Euler’s equation is a necessary condition that has to be satisfied at an extremal of a functional. Euler’s equation for the functional in (25) is

\[ \frac{\partial F}{\partial p_m} \bigg|_{p_m=\hat{p}_m} = 0, \quad (27) \]

or explicitly written,

\[ \lambda = \Pi(z) \frac{N_m(y) + I}{(1 - s(y)) N_m(y) + I}. \quad (28) \]

For a given \( y \) and a non-constant function \( \Pi \), there cannot be a Lagrange multiplier \( \lambda \) that is the same for any open interval of \( z \). This means that expression (11) and thus the basic reproduction number of the gross mutant competitive weight, \( R_0 \) (formula (10)), does not have an extremal. But because \( p_m \) is a probability and thus must assume values in [0, 1], the functional (11) is maximized for \( \hat{p}_m (\cdot, y) = 0 \) if it is decreasing for given \( y \), or it is maximized for \( \hat{p}_m (\cdot, y) = 1 \) if it is increasing. Hence, \( \hat{p}_m \) as a function of \( z \) must be a step function that assumes values zero and one only. In Appendix C, we show that, for given \( y \), \( \hat{p}_m \) as a function of \( z \) has at most one jump from zero to one if \( \Pi \) is a monotonically increasing function of body condition and from one to zero if \( \Pi \) is a monotonically decreasing function. (But note that any function that differs from \( \hat{p}_m \) on a set of points \( (z, y) \) of zero measure still maximizes (10) because the value of the integral does not change.) This means that every family who invests in retaining the natal patch sends away individuals who have the highest probability to survive dispersal. In patches with \( y < \hat{y} \), which are too unsafe or have too low productivity, every offspring disperses and \( \hat{p}_m (z, y) = 1 \) for all \( z \).

With increasing \( \Pi \), in patches where not all offspring disperse, the best reply strategy \( \hat{p}_m \) has the form

\[ \hat{p}_m (z, y) = \begin{cases} 0 & \text{if } z < \hat{z}_m(y), \\ 1 & \text{if } z > \hat{z}_m(y), \end{cases} \quad (29) \]

where \( \hat{z}_m(y) \) is the (still unknown) position of the jump. With (29), we simplify the basic reproduction number of the gross mutant competitive weight and obtain

\[ R_0(\hat{z}_m) = \int_Y \phi(y) \left( \frac{s(y)}{Bn(p, y) + I} + \frac{1 - s(y)}{BI} \right) s(y) B \left( \int_{\hat{z}_m(y)}^{+\infty} g(z) \beta(z, y) \Pi(z) \, dz \right) T(y, \hat{n}_m(y)) \, dy, \quad (30) \]
where
\[ \hat{n}_m(y) = \int_{-\infty}^{\hat{z}_m(y)} \beta(z, y) g(z) \, dz. \]  \hfill (31)

Note that the functional \( R_0 \) now depends on the function \( y \mapsto \hat{z}_m(y) \). To maximize \( R_0 \), we apply the calculus of variations once more and differentiate the integrand in (30) with respect to the scalar value of \( \hat{z}_m(y) \) and obtain Euler’s equation
\[
0 = \phi(y) \left( \frac{s(y)}{B(n(p, y) + I)} + \frac{1 - s(y)}{BI} \right) s(y) B \cdot \frac{g(\hat{z}_m(y)) \beta(\hat{z}_m(y), y)}{(1 - s(y))\hat{n}_m(y) + I}^2 \cdot \left( s(y) I \int_{\hat{z}_m(y)}^{+\infty} \beta(z, y) \Pi(z) g(z) \, dz - \Pi(\hat{z}_m(y)) \left( \hat{n}_m(y) + I \right) \left( (1 - s(y))\hat{n}_m(y) + I \right) \right). \]  \hfill (32)

For given \( y \), the value of \( \hat{z}_m(y) \) for which the equation in (32) is satisfied needs to be found numerically. We calculated numerically the evolutionarily stable strategies \( \hat{p} \) by applying a similar algorithm as for the previous examples to solve the fixed point problem for \( I \) (compare Section 4); we first choose a value for \( I \); next, for every \( y \) for which \( \hat{z}_m(y) \) exists, we find numerically the value of \( \hat{z}_m(y) \) for which eq. (32) is satisfied; then we calculate the new value of \( I \). At the ESS, \( I \) does not change. From patches for which no \( \hat{z}_m(y) \) exists, all offspring disperse and \( \hat{p}_m(z, y) \equiv 1 \) for all \( z \). We also need to investigate numerically whether the ESS is unique, because we cannot write down explicitly the equation for the fixed point of \( I \) as in the case of constant \( I \). In all examples discussed in this article, we have found that the ESS is unique. The top left panels in Figures 2 and 3 show the best reply \( I_{\text{next}} \) as a function of the current \( I \) for the example with constant \( s \) and increasing \( \Pi \) (Figure 2) respectively decreasing \( \Pi \) (Figure 3). (We have calculated the values for \( I_{\text{next}} \) indicated by the dots, which are simply interpolated. On the straight line, \( I_{\text{next}} = I \). The ESS is found at the intersection of the two graphs.)

The case of decreasing \( \Pi \) follows analogously. The best reply strategy is then a decreasing step function,
\[ \hat{p}_m(z, y) = \begin{cases} 1 & \text{if } z < \hat{z}_m(y), \\ 0 & \text{if } z > \hat{z}_m(y), \end{cases} \]  \hfill (33)

where \( \hat{z}_m(y) \) is the position of the jump. The integration interval of the integrals with respect to \( z \) in \( R_0 \) (formula (30)) and in eq. (32) changes to \( (-\infty, \hat{z}_m(y)) \), and for the integral \( \hat{n}_m \) in (31) to \( (\hat{z}_m(y), +\infty) \).

### 5.1 Numerical Examples for monotone \( \Pi \)

In the following numerical examples we assume the same functions for \( \phi, \beta \) and \( g \) as with constant \( \Pi \), defined in (20), (21) and (23). In the main examples, all patches will be equally safe with \( s(y) = 0.5 \) (\( s_1 = 0.5 \), \( s_2 = 0 \)) for all \( y \), but in Appendix D we show examples with increasing and decreasing \( s \). For the probability to survive dispersal we choose either the increasing function
\[ \Pi(z) = 0.2 + \frac{0.6}{1 + e^{-z}}, \]  \hfill (34)

such that stronger individuals are better dispersers (Figure 2) or the decreasing function
\[ \Pi(z) = 0.8 - \frac{0.6}{1 + e^{-z}}, \]  \hfill (35)
such that weak individuals are better dispersers (Figure 3). Both choices are biologically justified (see Section 2).

In each figure, the top middle panel shows the position of the jump \( \hat{z}(y) \) as a function of patch quality. The two dashed parallel lines indicate the set of pairs \((z, y)\) that occur in the population due to the truncation of \( \phi \) and \( \beta \). An individual with body condition \( z \) born in a patch of quality \( y \) disperses if and only if \((z, y)\) lies within the grey shaded area in the plot. The evolutionarily stable dispersal probability is identically 1 for all \( z \) for \( y < \hat{y} \), i.e., from the poorest patches all offspring disperse. In every patch where the local family invests in retaining the patch \( y > \hat{y} \), the weakest (strongest) offspring stay and the strongest (weakest) disperse when \( \Pi \) is increasing (decreasing).

The top right panels show the ESS non-dispersing weight \( \hat{n} \) as a function of patch quality. From the examples in this article, it seems that \( \hat{n} \) is increasing when \( s \) is a constant or increasing function of patch quality, but can be non-monotone when \( s \) decreases with \( y \) (as was the case with constant \( \Pi \)). However, since we cannot write down an explicit expression for \( \hat{n} \), we cannot prove this analytically.

The expected probability that an individual with a given body condition disperses,

\[
E(\hat{p}(z, y)|z) = \frac{\int_{-\infty}^{\infty} \phi(y) s(y) \beta(z, y) \hat{p}(z, y) \, dy}{\int_{-\infty}^{\infty} \phi(y) s(y) \beta(z, y) \, dy},
\]

is plotted in the bottom left panels. Because the poorest patches are always abandoned, \( E(\hat{p}(z, y)|z) = 1 \) for very small values of \( z \) in all examples. With increasing \( \Pi \) (Figure 2), intriguingly, \( E(\hat{p}(z, y)|z) \) is a non-monotone function of body condition such that both the weakest and the strongest individuals in the population as a whole always disperse. The strongest individuals always disperse...
5.1 Numerical Examples for monotone II

![Graphs](image)

Figure 3: The ESS for an example with decreasing II and constant s. Top left panel: The best reply value of \( I \) as a function of the current value of \( I \). Top middle panel: The position of the jump \( \hat{\zeta} \) of the increasing step function \( \hat{\rho} \) as a function of patch quality. Top right panel: The weighted sum of non-dispersers \( \hat{n} \) (thick line) as a function of patch quality. (Thin dashed line: total competitive weight \( A(y) \) produced in a patch.) Bottom left panel: The expected probability to disperse given a body condition, \( \mathbb{E}(\hat{\rho}(z, y)|z) \). Bottom middle panel: The body condition distribution \( d \) of dispersers. Bottom right panel: The body condition distribution \( f \) of non-dispersers.

because the strongest offspring of each family disperse such that \( \mathbb{E}(\hat{\rho}(z, y)|z) = 1 \) for a wide range of positive values of \( z \).

With decreasing II (Figure 3), the weakest from every patch disperse and \( \mathbb{E}(\hat{\rho}(z, y)|z) = 1 \) for all negative \( z \). \( \mathbb{E}(\hat{\rho}(z, y)|z) \) then decreases and equals zero for very large \( z \) because the strongest individuals in the population do not disperse but defend their natal patches. This resembles the dispersal behaviour as predicted by the social dominance hypothesis (dispersal of weak individuals [5]) but stems from a different underlying mechanism. Here, dispersal of weak is provoked by kin competition (in contrast to the social dominance hypothesis that does not assume any kinship between individuals) and by the fact that the weak are more apt to disperse than the strong.

The bottom middle and right panels show the body condition distribution among dispersers,

\[
d(z) = \frac{\int_{-\infty}^{+\infty} \phi(y) s(y) \beta(z, y) \hat{\rho}(z, y) dy}{\int_{-\infty}^{+\infty} \int_{-\infty}^{+\infty} \phi(y) s(y) \beta(z', y) \hat{\rho}(z', y) dz' dy},
\]

and among non-dispersers,

\[
f(z) = \frac{\int_{-\infty}^{+\infty} \phi(y) s(y) \beta(z, y) (1 - \hat{\rho}(z, y)) dy}{\int_{-\infty}^{+\infty} \int_{-\infty}^{+\infty} \phi(y) s(y) \beta(z', y) (1 - \hat{\rho}(z', y)) dz' dy},
\]

respectively (the vertical lines in the plots indicate the means). These are the two graphs that are most relevant from an empirical point of view, as these show the quantities that can be measured in the field most easily and directly. Remarkably, the mean of \( d \) is very close to zero in both examples and \( d \) looks rather symmetric. Hence, \( d \) does not at all reveal the underlying dispersal strategy (the strongest respectively the weakest offspring of a family disperse in Figure 2 respectively 3). Neither does the graph of \( f \) in Figure 2 hint that the strong are dispersing. The graph
of \( f \) in Figure 3 exhibits that the weak disperse. However, because with decreasing \( \Pi \), dispersing offspring are a minority in the whole population (see the graph for \( \hat{z} \)), the graph of \( d \) looks like the body condition distribution of all offspring in the whole population. Measuring \( f \) precisely in the field is certainly difficult in such a case.

In all examples in this article, the weakest individuals in the whole population always disperse because they abandon their poor natal patches (cf. the graphs of \( E(\hat{p}(z, y)|z) \)). But these dispersers are rare and do not influence the shape of \( d \) and \( f \) enough so that their behaviour could be detected from studying \( d \) or \( f \). Only when weak are better dispersers (decreasing \( \Pi \)), then we can see from the graphs of \( E(\hat{p}(z, y)|z) \) and \( f \) that all weak in fact disperse but strong generally stay in their patches.

The examples in Appendix D, where both \( \Pi \) and \( s \) are monotone functions, give for the most part qualitatively similar results as with constant \( s \) in Figures 2 and 3, with variable contrast between the distribution of dispersers and non-dispersers. The bottom example in Figure D.4 (decreasing \( \Pi \) and strongly decreasing \( s \)) is an exception; here, strong individuals abandon their extremely unsafe natal patches and disperse with high probability such that \( E(\hat{p}(z, y)|z) \) is a non-monotone function of body condition.

In Appendix E, we compare the graphs of the non-dispersing weight \( \hat{n} \) for cases with the same function \( \Pi \) but changing \( s \), and for cases with the same \( s \) but different functions for \( \Pi \).

Note that these few examples can by no means give an exhaustive understanding of different phenomena that can be observed when studying body condition dependent dispersal. However, they teach us the important lesson that it can be extremely difficult in practice to detect the underlying dispersal strategy when it is only possible to study population-wide (and not within-family) behaviour. Although studying dispersal is generally challenging \([17]\), it may be relatively manageable to measure individual body conditions (e.g. body mass, weight, length of certain limbs or mandibles). But determining the environmental quality of a habitat (and hence, according to our model, the competitive weight of an inhabiting family) is often no simple undertaking \([13]\). Nevertheless, our findings suggest that habitat quality and within-family behaviour should be included when studying body condition dependent dispersal.

## 6 Discussion

The model in this paper progresses from two previous models for the evolution of body condition \([8, 9]\) and generalizes and complements these two previous studies in several crucial aspects.

The most significant difference to both previous models is that environmental patch qualities stay now constant rather than change randomly every year. The two extremes frame reality where patch qualities may change over time but not independently of the past. The two extremes also reveal opposite general dispersal attitudes: With random patch qualities as in \([8, 9]\), a family firstly invests in retaining the natal patch and secondly disperses some offspring if the total competitive weight is sufficiently large that dispersing some competitive weight is more beneficial than keeping more weight in the patch. This is because the future expectation of patch quality is the same for the home patch as well as all other patches, i.e., all patches are equally valuable. Yet, if dispersal survival is a monotone function of body condition, the competitive weight retained in a patch depends on patch quality because the success of dispersers depends on it. When survival dispersal is constant, then also the non-dispersing weight is the same in every patch. Conversely, in a fixed environment as in the present model, patches of low quality are always abandoned and only
sufficiently safe and productive patches are defended. Empirical studies confirmed that dispersal can be a strategy to avoid poor conditions in the natal habitat [1], and that dispersal probability decreases with increasing territory quality [3, 18].

When all individuals are equally fit to disperse (the probability Π to survive dispersal is constant), then the ESS is an equivalence class of selectively neutral dispersal strategies that all lead to the same non-dispersing weight. With monotonically increasing or decreasing Π, the offspring with the highest survival chances during dispersal do disperse from each family (the strongest when Π is an increasing function of body condition and the weakest when Π is decreasing). The same rules determine also the dispersal behaviour in the model in [9] for each family that invests in retention of the home patch.

Both models do in fact agree about when strong individuals should disperse. When the strong are better dispersers, then the strongest of families as well as of the whole population disperse (compare Figure 3 in [9] with Figure 2 in the present article, where the expected probability of dispersal \( E(\hat{p}(z, y) | z) = 1 \) for (very) high values of \( z \)). When strong individuals are bad dispersers, they never disperse, as \( E(\hat{p}(z, y) | z) = 0 \) for very large body conditions (compare Figure 4 in [9] with Figure 3 in the present article). But note the exception when safety \( s \) is a strongly decreasing function of patch quality (bottom example in Figure D.4): In this case the strongest individuals in the population abandon their extremely unsafe patches. This is a consequence of the fixed environment and cannot occur in a temporally fluctuating environment where it is not known how safe the patch will be in the next year.

Weak offspring disperse in a different way in fixed and randomly changing environments. In a fluctuating environment, families in poor patches invest everything in retention of the patch and hence \( E(\hat{p}(z, y) | z) = 0 \) for weakest body conditions (Figures 2 and 3 in [9]). In a fixed environment, however, the poorest patches are always abandoned and hence \( E(\hat{p}(z, y) | z) = 1 \) for small \( z \) in all examples in the present article. In other words, in a fixed environment, the expected probability of dispersal for the weakest is lifted from never to always, whereas it often does not change the dispersal behaviour of the strongest. We can therefore expect that, when the environment fluctuates and fluctuations are temporally correlated, then strong individuals disperse if they have better chances to survive dispersal (except when their rich natal habitat patch is too unsafe), whereas weak individuals disperse with low probability if environmental fluctuations are uncorrelated and with high probability when the environment is rather fixed.

Together with the models in [8, 9], the present model provides thus example scenarios both for dispersal of strong as well as for dispersal of weak. The emphasis in [8] lies on the effects of different competition mechanisms on body condition dependent dispersal in a fluctuating environment where all offspring born in one patch have the same body condition that corresponds to the environmental quality of the patch (the body condition distribution \( \beta \) is a delta peak at the patch quality). We found the ESS dispersal strategies \( \hat{p} \) for the four mechanisms: (i) a weighted lottery; (ii) a mixture of pure and weighted lottery (in a small fraction of patches, the winner is determined by pure chance, and in all other patches by a weighted lottery); (iii) the infinite number of competitors in a patch is randomly reduced to a fixed number and then the strongest individual establishes itself in a patch; and (iv) the strongest among a random number of survivors establishes itself. In all these examples, the strong disperse always or with a very high probability, while the weak only disperse when competition is independent of body condition in some patches in the metapopulation (as in case (ii) and in case (iv) when the fraction of patches where the number of competitors is reduced to 1 is not too small; see Figures 3 and 4 in [8]). However, the mechanism that leads to dispersal of weak is different in the two models. In the present model,
weak abandon their poor natal patches. In [8], a family of weak avoid dying altogether in their natal patch by dispersing, but only when they have a chance to establish themselves in the patches where body condition does not play a role during competition. Moreover, these two mechanisms provide explanations for dispersal of weak that are essentially different from the proposition of the social dominance hypothesis. The social dominance hypothesis assumes no kinship between competitors. In our models, however, dispersal is driven by kin competition, and weak individuals disperse as soon as their chances to gain other patches increase due to partly random competition or if their patches are not valuable enough to retain.

Another significant difference between the models in [8, 9] and the present model is that different fitness measures are maximized. In the previous models with randomly changing patch qualities, the number of mutant offspring that occupy patches in the next year is maximized (denoted by $W$, defined e.g. in eq. (1) in [9]). In the present model, however, the basic reproduction number $R_0$ of the mutant weight is maximized (defined in formula (10)). $W$ is an annual quantity because every year the random reassignment of patch qualities wipes out any relation between the body conditions of an individual’s offspring and the environmental quality of the individual’s natal patch. Contrarily, $R_0$ describes the life-time reproductive success of an individual that includes all offspring as long as the family retains the natal patch (which always has the same quality). This is similar to a model for individual optimization by Kisdi et al [14]. They modelled the optimal reaction norm of fecundity as a function of individual body condition for an iteroparous organism. Two special cases were considered: (i) individual quality varies randomly every year (the quality distribution in the whole population is fixed), and (ii) individual quality is fixed during lifetime. The optimal reaction norm maximizes the population rate of increase such that in case (i), the annual growth rate of individuals with given body condition is maximized, whereas in case (ii), the life-time reproductive success of an individual is maximized.

Both the model in [9] and the present one offer a possible explanation why it is so difficult to interpret data on body condition dependent dispersal. It can be impossible to detect clear within-family patterns from population-wide data. In almost all examples, the body condition distribution $d$ of dispersers does not indicate that body condition influences dispersal behaviour. In some examples the body condition distribution $f$ of non-dispersers reflects the underlying dispersal strategy that either the strongest or the weakest from each family disperse, but in other examples it does not.

The fact that for constant survival probability during dispersal there exist infinitely many dispersal strategies that are all selectively neutral and equally evolutionarily stable, is consistent with empirical data where there is no consistent pattern found in body condition dependent dispersal. Each family within a metapopulation can ‘randomly’ decide which of its offspring to disperse as long as the competitive weight that stays at home has the ESS value.

Acknowledgements

This research was financially supported by the Finnish Graduate School in Computational Sciences (FICS) and by the Finnish Centre of Excellence in Analysis and Dynamics Research of the Academy of Finland.
Appendix A

In this Appendix, we show for Case I when $\Pi$ is constant, how the mutant’s best reply non-dispersing weight $\hat{n}_m(1)$ (formula (17) in the main text) and hence the ESS non-dispersing weight depends on patch safety $s$ and productivity $A$.

The partial derivatives of $\hat{n}_m(1)$ with respect to $s$ respectively $A$ are

$$\frac{\partial \hat{n}_m(1)}{\partial s} = \frac{I \left( A(y)(1 - s(y)) + I(1 + s(y)) - 2\sqrt{s(y)}I((1-s(y))A(y) + I) \right)}{2(1 - s(y))^2 \sqrt{s(y)}I((1-s(y))A(y) + I)} \quad (39)$$

$$\frac{\partial \hat{n}_m(1)}{\partial A} = \frac{s(y)I}{2\sqrt{s(y)}I((1-s(y))A(y) + I)} \quad (40)$$

Both derivatives are always positive, i.e., $\hat{n}_m(1)$ increases with increasing $s$ and with increasing $A$. Therefore, when $y$ changes such that both $s(y)$ and $A(y)$ increase (decrease), then $\hat{n}_m(1)$ increases (decreases) as well. $\hat{n}_m(1)$ can have extrema only if $s(y)$ increases but $A(y)$ decreases or vice versa.

In the examples in the main text, we assume a one-dimensional continuum of patch qualities $y$. Productivity $A(y)$ always increases with $y$. The ESS non-dispersing weight $\hat{n}$ therefore monotonically increases with patch quality when $s$ is constant or increasing, and can have extrema only when $s$ is decreasing.

Appendix B

In this Appendix, we prove that with constant $\Pi$, there always exists a unique ESS. The best reply value for $I$ as a function of the current $I$ is

$$I_{next}(I) = I_{max} - \Pi \int_{\tilde{Y}(I)} \frac{\phi(y)s(y)}{1 - s(y)} \left( \sqrt{s(y)}I(1-s(y))A(y) + I - I \right) dy, \quad (41)$$

which is formula (18) in the main text. $I_{max} = \Pi \int_{Y} \phi(y) s(y) A(y) dy$ is the maximal weighted sum of immigrants per patch, and $\tilde{Y}(I) \subseteq Y$ is the set where (16) holds.

The best reply to $I = 0$ is $I_{next}(0) = I_{max}$, and $I_{next}(I_{max}) < I_{max}$; therefore, there exists at least one $I$ that is the best reply to itself.

The derivative of $I_{next}$ with respect to $I$ is

$$\frac{\partial I_{next}}{\partial I} = \Pi \int_{\tilde{Y}(I)} \phi(y) \left[ \frac{s(y)}{1 - s(y)} \left( 1 - \frac{s(y)(1-s(y))A(y) + 2s(y)I}{2\sqrt{s(y)}I((1-s(y))A(y) + I)} \right) \right] dy \quad (42)$$

(note that the integrand in (41) is zero at the boundary of $\tilde{Y}(I)$). Straightforward algebra shows that the term in the square brackets in (42) is less than 1 for all $y$. Because $\int_{-\infty}^{+\infty} \phi(y) dy = 1$ and $\Pi \leq 1$, the derivative of $I_{next}$ is always less than one. This implies that the fixed point of $I$ is unique, i.e., there is always a unique ESS.
Appendix C

In this Appendix, we give the proof that the best reply mutant strategy in a patch of quality $y$ is either $\hat{p}_m(z, y) \equiv 1$ for all $z$ (all offspring disperse from a patch with quality $y$) or a step function of $z$ with a single jump from 0 to 1 (respectively from 1 to 0) at some position $\hat{z}_m(y)$ if $\Pi(z)$ is monotonically increasing (decreasing) with $z$. The proof follows essentially the proof in the Appendix of [9], where we prove a similar statement for the model in [9].

We consider piecewise continuous functions for $\hat{p}_m$, and assume that $\beta(z, y)g(z) > 0$ for all $z$ and $y$. For increasing $\Pi$, the best reply mutant strategy has the form

$$\hat{p}_m(z, y) = \begin{cases} 0 & \text{if } z \in U(y) \\ 1 & \text{otherwise} \end{cases}$$

for some $U(y) \subset \mathbb{R}$ (because $p_m(\cdot, y) \equiv 0$ is never a best reply for any $y$, $U(y)$ is always a proper subset of $\mathbb{R}$). If $\hat{p}_m(\cdot, y) \equiv 1$ is the best reply, then $U(y)$ is the empty set.

The claim is that $U(y) = (-\infty, \hat{z}_m(y))$ for some finite $\hat{z}_m(y)$ (the position of the jump).

Consider the strategy $\bar{p}_m$ for which, in a set of patch qualities $y$ of non-zero measure, $\bar{U}(y)$ is neither empty nor of the form $(-\infty, \hat{z}_m(y))$. Then

$$R_0(\bar{p}_m) = \int_Y \phi(y) \left( \frac{s(y)}{B(n(p, y) + 1)} + \frac{1 - s(y)}{BI} \right) \cdot s(y) B \left( \int_{\bar{U}(y)} g(z') \beta(z', y) \bar{p}_m(z', y) \Pi(z') dz' \right) T(y, n(\bar{p}_m, y)) dy.$$ 

Consider also the strategy $\hat{p}_m$ for which $\hat{U}(y) = (-\infty, \hat{z}_m(y))$ and $n(\hat{p}_m, y) = n(\bar{p}_m, y)$ for all $y$; strategy $\hat{p}_m$ keeps the same competitive weight in every patch as $\bar{p}_m$, but composes it differently such that from each patch, the strongest individuals ($z > \hat{z}_m(y)$) disperse. Define $\mathcal{Y}(p_m) = \{y : U(y) \neq \emptyset\}$ to be the set of patch qualities $y$ of patches where some individuals stay; obviously, $\mathcal{Y}(\bar{p}_m) = \mathcal{Y}(\hat{p}_m)$. Then,

$$R_0(\bar{p}_m) - R_0(\hat{p}_m) = \int_Y \phi(y) \left( \frac{s(y)}{B(n(p, y) + 1)} + \frac{1 - s(y)}{BI} \right) s(y) B T(y, n(\bar{p}_m, y)) \cdot \left[ \int_{\mathcal{Y}(\bar{p}_m)} g(z) \beta(z, y) \Pi(z) dz \right. - \left. \int_{\mathcal{Y}(\hat{p}_m)} g(z) \beta(z, y) \Pi(z) dz \right] dy$$

Because $n(\hat{p}_m, y) = n(\bar{p}_m, y)$, it follows that $\int_{\mathcal{Y}(\bar{p}_m)} g(z) \beta(z, y) \Pi(z) dz = \int_{\mathcal{Y}(\hat{p}_m)} g(z) \beta(z, y) \Pi(z) dz$. Because $\beta$ and $g$ are positive and $\Pi$ is increasing, the value of the square bracket in (46) is positive for every $y \in \mathcal{Y}(\bar{p}_m)$. $\hat{p}_m$ is thus no best reply, and the best reply must have the form (29).

Appendix D

This Appendix shows the ESS for four examples with different shapes of the functions $\Pi$ and $s$. The function for increasing $\Pi$ is given in formula (14) in the main text and for decreasing $\Pi$ in formula (35). $s$ is given in formula (22). $I$ is the ESS value of the immigrant weight (see (2)). $\bar{y}$ is the threshold value of patch qualities below which patches are too unsafe or have too low
productivity such that all offspring disperse and \( \hat{p}_m(z, y) = 1 \) for all \( z \). If two values are given, then patches with \( y < \hat{y}_1 \) and with \( y > \hat{y}_2 \) are abandoned.

In each figure, the panels give the following quantities:

*top middle panel:* the position of the jump \( \hat{z}(y) \) of the evolutionarily stable dispersal probability \( \hat{p} \) (that is a step function with respect to \( z \)) as a function of patch quality \( y \).

*top right panel:* the non-dispersing weight \( \hat{n}(y) \) as a function of patch quality \( y \).

*bottom left panel:* the expected probability \( \mathbb{E}(\hat{p}(z, y) | z) \) to disperse given a body condition \( z \).

*bottom middle panel:* the body condition distribution \( d \) among dispersers.

*bottom right panel:* the body condition distribution \( f \) among non-dispersers.

**Increasing \( \Pi \) and \( s \) (Figure D.1)**

mildly increasing \( s \): \( s_1 = 0.25, s_2 = 0.75 : \hat{I} \approx 0.72, \hat{y} \approx -0.5 \),
middiately increasing \( s \): \( s_1 = 0.08, s_2 = 0.92 : \hat{I} \approx 0.78, \hat{y} \approx -0.37 \),
strongly increasing \( s \): \( s_1 = 0, s_2 = 1 : \hat{I} \approx 1, \hat{y} \approx -0.3 \).

**Increasing \( \Pi \) and decreasing \( s \) (Figure D.2)**

mildly decreasing \( s \): \( s_1 = 0.75, s_2 = 0.25 : \hat{I} \approx 0.58, \hat{y} \approx -1.44 \),
intermediately decreasing \( s \): \( s_1 = 0.92, s_2 = 0.08 : \hat{I} \approx 0.4, \hat{y} \approx -1.89 \),
strongly decreasing \( s \): \( s_1 = 1, s_2 = 0 : \hat{I} \approx 0.33, \hat{y}_1 \approx -2.15, \hat{y}_2 \approx 2.26 \).

**Decreasing \( \Pi \) and increasing \( s \) (Figure D.3)**

mildly increasing \( s \): \( s_1 = 0.25, s_2 = 0.75 : \hat{I} \approx 0.41, \hat{y} \approx -0.85 \),
intermediately increasing \( s \): \( s_1 = 0.08, s_2 = 0.92 : \hat{I} \approx 0.43, \hat{y} \approx -0.61 \),
strongly increasing \( s \): \( s_1 = 0, s_2 = 1 : \hat{I} \approx 0.41, \hat{y} \approx -0.55 \).

**Decreasing \( \Pi \) and \( s \) (Figure D.4)**

mildly decreasing \( s \): \( s_1 = 0.75, s_2 = 0.25 : \hat{I} \approx 0.29, \hat{y} \approx -1.96 \),
intermediately decreasing \( s \): \( s_1 = 0.92, s_2 = 0.08 : \hat{I} \approx 0.23, \hat{y} \approx -2.34 \),
strongly decreasing \( s \): \( s_1 = 1, s_2 = 0 : \hat{I} \approx 0.2, \hat{y}_1 \approx -2.35, \hat{y}_2 \approx 2.23 \).
Figure D.1: The ESS for increasing $\Pi$ and different increasing $s$. For each example: Top middle panel: The position of the jump $\hat{z}$ of the increasing step function $\hat{p}$ as a function of patch quality. Top right panel: The weighted sum of non-dispersers $\hat{n}$ (thick line) as a function of patch quality. (Thin dashed line: total competitive weight $A(y)$ produced in a patch.) Bottom left panel: The expected probability to disperse given a body condition, $E(\hat{p}(z, y)|z)$. Bottom middle panel: The body condition distribution $d$ of dispersers. Bottom right panel: The body condition distribution $f$ non-dispersers.
Figure D.2: The ESS for increasing $\Pi$ and different decreasing $s$. For each example: *Top middle panel:* The position of the jump $\hat{z}$ of the increasing step function $\hat{p}$ as a function of patch quality. *Top right panel:* The weighted sum of non-dispersers $\hat{n}$ (thick line) as a function of patch quality. (Thin dashed line: total competitive weight $A(y)$ produced in a patch.) *Bottom left panel:* The expected probability to disperse given a body condition, $E(\hat{p}(z, y) | z)$. *Bottom middle panel:* The body condition distribution $d$ of dispersers. *Bottom right panel:* The body condition distribution $f$ non-dispersers.
Figure D.3: The ESS for decreasing $\Pi$ and different increasing $s$. For each example: 

*Top middle panel:* The position of the jump $\hat{z}$ of the increasing step function $\hat{p}$ as a function of patch quality. 

*Top right panel:* The weighted sum of non-dispersers $\hat{n}$ (thick line) as a function of patch quality. (Thin dashed line: total competitive weight $A(y)$ produced in a patch.) 

*Bottom left panel:* The expected probability to disperse given a body condition, $E(\hat{p}(z, y)|z)$. 

*Bottom middle panel:* The body condition distribution $d$ of dispersers. 

*Bottom right panel:* The body condition distribution $f$ non-dispersers.
Figure D.4: The ESS for decreasing $\Pi$ and different decreasing $s$. For each example: Top middle panel: The position of the jump $\hat{z}$ of the increasing step function $\hat{p}$ as a function of patch quality. Top right panel: The weighted sum of non-dispersers $\hat{n}$ (thick line) as a function of patch quality. (Thin dashed line: total competitive weight $A(y)$ produced in a patch.) Bottom left panel: The expected probability to disperse given a body condition, $E(\hat{p}(z, y)|z)$. Bottom middle panel: The body condition distribution $d$ of dispersers. Bottom right panel: The body condition distribution $f$ non-dispersers.
Appendix E

In this Appendix, we compare different examples with the same function for $\Pi$ but different $s$ (Figure E.1), respectively examples with the same $s$ but different $\Pi$ (Figure E.2). $\Pi$ is either the increasing function in (34) or the decreasing function (35). $s$ is given in formula (22). We focus our comparisons on the graphs for the ESS non-dispersing weight $\hat{n}$ (thick lines) as the quantity that can be computed in all examples in this article. The parameter values for $s_1$ and $s_2$ and the corresponding styles of the graph of $\hat{n}$ for the different shapes of $s$ are as follows:

- **constant $s$ (solid):** $s_1 = 0.5, s_2 = 0$
- **mildly increasing $s$ (long-dashed):** $s_1 = 0.25, s_2 = 0.75$
- **intermediately increasing $s$ (short-dashed):** $s_1 = 0.08, s_2 = 0.92$
- **strongly increasing $s$ (dotted):** $s_1 = 0, s_2 = 1$

(51)

- **mildly decreasing $s$ (long-dashed):** $s_1 = 0.75, s_2 = 0.25$
- **intermediately decreasing $s$ (short-dashed):** $s_1 = 0.92, s_2 = 0.08$
- **strongly decreasing $s$ (dotted):** $s_1 = 1, s_2 = 0$

The value of $s$ for patches with $y = 0$ remains constant as $s(0) = 0.5$ for all shapes of $s$. The thin dashed line is the graph of the total competitive weight $A(y)$ produced in a patch $y$.

**Comparison of examples with the same $\Pi$** In every panel in Figure E.1, $\Pi$ is either increasing or decreasing while $s$ changes shape (steepness). When comparing examples with the same $\Pi$ but different shapes of $s$, mainly three mechanisms explain the changes in the ESS $\hat{n}$. The first relates to how the function $s$ (patch safety) changes. When $s$ increases in steepness, safety in a rich patch ($y > 0$) increases and safety in a poor patch ($y < 0$) decreases when $s$ is an increasing function of patch quality. If $s$ is a decreasing function of patch quality, then increasing steepness of $s$ implies that poor patches become safer and rich patches become unsafer. Changing $s$ from constant to increasing (left column) selects for an increase in $\hat{n}$, changing $s$ from constant to decreasing (right column) selects for a decrease in $\hat{n}$. Simultaneously, the threshold patch quality $\hat{y}$ below which patches are abandoned increases when $s$ changes from constant to increasing and decreases when $s$ changes from constant to decreasing.

The second mechanism has to do with changes of the value of the incoming immigrant weight $B\hat{I}$. For instance, when $s$ changes from constant to increasing (left column), then $\hat{I}$ increases. This selects for an increase of $\hat{n}$, because families counteract the increased immigrant weight by an increased non-dispersing weight. We see increased $\hat{n}$ in rich patches. When $s$ changes from constant to decreasing (right column), then $\hat{I}$ decreases, which selects for a decrease of $\hat{n}$.

A closer look at least at the panels for increasing $\Pi$ reveals that the intersection point of the graphs of $\hat{n}$ are not at $y = 0$ but a bit shifted to the right. Patches with $y$ between zero and the intersection point become safer (less safe) with increasing (decreasing) $s$, but families invest less (more) in retention ($\hat{n}$ decreases (increases)). Both mechanisms mentioned above do not explain this phenomenon. However, these mechanisms only look at the situation within a local patch (i.e., safety and immigrant weight), but do not consider how the probability that dispersers gain other patches might change. For instance, with increasing $s$, more poor patches (and hence more patches altogether) are abandoned when $s$ becomes steeper, and this may select for dispersal. This might be a reason why families in patches slightly richer than average behave contrarily to what we might at first expect.
**Comparison of examples with the same $s$** Let us now compare two examples with increasing $s$ but different $\Pi$. With mildly increasing $s$, when $\Pi$ changes from constant (solid line in the left top panel in Figure E.2) to increasing (long-dashed line), then strong individuals become better dispersers than weak. The value of $\hat{I}$ therefore increases from $\hat{I} \approx 0.68$ to $\hat{I} \approx 0.96$. There are two selective forces at work: increased immigrant weight selects against dispersal and thus for an increase of $\hat{n}(y)$ and a decrease of $\hat{y}$. Because the probability to survive dispersal now increases with body condition, adding more dispersers would mean to add individuals with a worse chance to survive dispersal (than their siblings). This also selects against dispersal. Indeed, $\hat{n}(y)$ increases (the long-dashed line lies slightly above the solid line) and $\hat{y}$ decreases from $\hat{y} \approx -0.12$ (with increasing $s$ and constant $\Pi$) to $\hat{y} \approx -0.5$ (with increasing $\Pi$). The same holds for all other examples with increasing $s$ and a switch from constant to increasing $\Pi$ (see other panels in the left column of Figure E.2). Even for the examples with decreasing $s$, changes in the ESS $\hat{n}$ can be explained by this reasoning. When $s$ is e.g. mildly decreasing and $\Pi$ changes from constant to increasing (solid and long-dashed lines in the top right panel in Figure E.2), then the value of $\hat{I}$ increases from $\hat{I} \approx 0.44$ (with constant $\Pi$) to $\hat{I} \approx 0.58$ (with increasing $\Pi$). Consequently, $\hat{n}(y)$ increases for every $y$ and $\hat{y}$ decreases from $\hat{y} \approx -0.92$ to $\hat{y} \approx -1.44$ such that the range of patches where families invest in retaining their natal patch increases. (With strongly decreasing $s$, the value of $\hat{y}_1$ decreases and $\hat{y}_2$ increases.)

Switching from constant to decreasing $\Pi$ implicates a less straightforward change in dispersal behaviour. See e.g. the top left panel in Figure E.2 for a comparison of the example with mildly increasing $s$ and constant $\Pi$ (solid line) with the example with decreasing $\Pi$ (short-dashed line). The two selective forces described above are now working against each other. In all examples with decreasing $\Pi$, the value of $\hat{I}$ is smaller than in the corresponding examples with constant $\Pi$. This selects for dispersal and for a decrease of $\hat{n}$. But if a family wanted to add more dispersers, the additional dispersers would be stronger individuals who have a smaller survival probability during dispersal. Hence, dispersal is also selected against. Decreased $\hat{I}$ seems to have a stronger impact in rich patches such that in these patches $\hat{n}$ decreases. In patches of intermediate quality, the second mechanism prevails and $\hat{n}$ increases and simultaneously $\hat{y}$ decreases. The case with strongly decreasing $s$ is an exception again (bottom right panel); then all families invest more in retention of their natal patches and the value of $\hat{y}_1$ decreases and $\hat{y}_2$ increases.
Figure E.1: The ESS weighted sum of non-dispersers $\hat{n}(y)$ as a function of patch quality for different shapes of $s$ and $\Pi$. In each panel, $\Pi$ is either increasing or decreasing, and $s$ differs in steepness. Thin dashed line: $A(y)$. Thick lines: $\hat{n}(y)$ (solid: constant $s$; long-dashed: $s$ mildly increasing resp. decreasing; short-dashed: $s$ intermediately increasing resp. decreasing; dotted: $s$ strongly increasing resp. decreasing).

Figure E.2: The ESS weighted sum of non-dispersers $\hat{n}(y)$ as a function of patch quality for different shapes of $s$ and $\Pi$. In each panel, $s$ has a fixed shape, but $\Pi$ changes. Thin dashed line: $A(y)$. Thick lines: $\hat{n}(y)$ (solid: constant $\Pi$; long-dashed: increasing $\Pi$; short-dashed: decreasing $\Pi$).
References


Article IV

Bulletin of Mathematical Biology 69, 2007, pp 1649–1672,
Quasi-local competition in stage-structured metapopulations:
A new mechanism of pattern formation,
Margarete Utz, Éva Kisdi, Michael Doebeli.

Printed with kind permission from Springer Science + Business Media.
Quasi-Local Competition in Stage-Structured Metapopulations: A New Mechanism of Pattern Formation

Margarete Utz, Éva Kisdi, Michael Doebeli

Abstract

A central question of ecology is what determines the presence and abundance of species at different locations. In cases of ecological pattern formation, population sizes are largely determined by spatially distributed interactions and may have very little to do with the habitat template. We find pattern formation in a single-species metapopulation model with quasi-local competition, but only if the populations have (at least) two age or stage classes. Quasi-local competition is modelled using an explicit resource competition model with fast resource dynamics, and assuming that adults, but not juveniles, spend a fraction of their foraging time in habitat patches adjacent to their home patch. Pattern formation occurs if one stage class depletes the common resource but the shortage of resource affects mostly the other stage. When the two stages are spatially separated due to quasi-local competition, this results in competitive exclusion between the populations. We find deep similarity between spatial pattern formation and population cycles due to competitive exclusion between cohorts of biennial species, and discuss the differences between the present mechanism and established ways of pattern formation such as diffusive instability and distributed competition with local Allee-effects.

Keywords: metapopulation · pattern formation · quasi-local competition · spatially distributed competition · stage structure

1 Introduction

Biomathematicians have been intrigued by pattern formation ever since the ground-breaking work of Turing (1952). Naive intuition suggests that a homogeneous environment should be uniformly populated. There are, however, striking examples of ecological pattern formation, such as striped vegetation in semiarid areas (Klausmeier, 1999), which show that the environmental symmetry can be broken.

The classic mechanism of pattern formation involves an activator-inhibitor system where the inhibitor diffuses better than the activator (Turing, 1952; see e.g. Edelstein-Keshet (1988), Murray (2003) for detailed introductions and Holmes et al. (1994) for a succinct review of various ways of pattern formation). In an ecological context, this can be realized by a predator-prey system where the predator is more mobile (Segel and Jackson, 1972; de Roos et al., 1998). Pattern formation in homogeneous environments is, however, also possible in single-species systems if two conditions are satisfied (Levin and Segel, 1985; Britton, 1989; Furter and Grinfeld, 1989): First, competition is spatially distributed such that individuals would experience large competitive impact in areas adjacent to existing populations (lateral inhibition); and second, Allee-effects enhance population
growth in existing populations, but this effect is more localized than competition (local activation).

In metapopulations consisting of discrete habitat patches, pattern formation amounts to the destabilization of the homogeneous equilibrium and the stabilization of inhomogeneous equilibria. At an inhomogeneous equilibrium, the habitat patches differ in their equilibrium population density despite the fact that environmental conditions are identical. The analogue of distributed competition in discrete metapopulations is termed quasi-local competition, where individuals of one population compete also with members of adjacent populations (Gyllenberg et al., 1999; Doebeli and Killingback, 2003; Kisdi and Utz, 2005). Quasi-local competition results if the resource disperses between neighboring populations such that consumption by one population depletes the resource also of the adjacent populations (Levin, 1974), or if, as in our model, individuals spend a certain fraction of their foraging time in the adjacent populations. 'Apparent competition' can also be quasi-local or non-local if a mobile predator or parasite regulates population growth (de Roos et al., 1998). In contrast to competition, Allee-effects are assumed to operate only within populations.

Most metapopulation models assume that local populations are connected only by dispersal. Under this assumption, Allee-effects can stabilize existing spatial patterns by preventing population growth in empty habitat patches and thus stabilizing inhomogeneous equilibria (Gruntfest et al., 1997; Amarasekare, 1998). In absence of lateral inhibition provided by quasi-local competition, however, the homogeneous equilibrium remains locally stable (Gyllenberg et al., 1999). Indeed, Rohani et al. (1996) have proved under rather general conditions that when identical patches are connected only by passive dispersal, the homogeneous equilibrium is stable if and only if the dynamics of a single isolated population has a stable fixed point. Allee-effects (local activation) without quasi-local competition (lateral inhibition) can conserve but cannot generate spatial patterns (Gyllenberg et al., 1999).

Is quasi-local competition sufficient by itself to destabilize the homogeneous equilibrium? Many classic examples show that in order to form patterns, lateral inhibition must be accompanied by some form of local activation, such as Allee-effects from enhanced reproduction or predator saturation, or locally favorable conditions in heterogeneous environments (Segel and Jackson, 1972; Levin, 1974; Gurney and Nisbet, 1976; Levin and Segel, 1985; Britton, 1989; Furter and Grinfeld, 1989; de Roos et al., 1998; Sasaki, 1997; Gyllenberg et al., 1999). In sharp contrast to previous results, however, Doebeli and Killingback (2003) found pattern formation in a metapopulation subject only to quasi-local competition, without any form of local Allee-effects and in a homogeneous environment.

The model of Doebeli and Killingback (2003) contained only one species and incorporated quasi-local competition via an extension of the Beverton-Holt equation. The Beverton-Holt model has recently been derived from an explicit resource-consumer model (Geritz and Kisdi, 2004). To see whether the results of Doebeli and Killingback (2003) hold also when quasi-local competition is mechanistically derived from underlying interactions, Kisdi and Utz (2005) extended the resource-consumer model of Geritz and Kisdi (2004) to metapopulations following the assumptions of Doebeli and Killingback (2003) and assuming a simple life history of the consumer without essential age- or stage-structure (mature adults emerge from completely inert eggs). The results of this mechanistic model contradicted those of Doebeli and Killingback (2003): No pattern formation was found in homogeneous environments.

In the present article, we show that pattern formation with quasi-local competition but without Allee-effects is in fact possible also in a mechanistic resource competition model, if we assume
that the consumer population has a simple stage-structure. Here we extend the model of Kisdi and Utz (2005) such that the consumer has a fixed, one-year long juvenile (larval) stage before reaching maturation. The juveniles feed on the same limiting resource as the adults. Adults, but not juveniles, are mobile and forage also in adjacent patches. We show that spatial patterns form under two sets of conditions: If the adults deplete the resource and this has a large effect on juvenile survival; and in the reverse case, if the juveniles deplete the resource and this has a large effect on the adults. To our knowledge, this represents a novel mechanism of pattern formation. We do not assume any Allee-effects (directly or indirectly as e.g. from predator saturation) for local activation. Instead, the stage-structure of the population plays a central role.

Most of the present analysis focuses on a two-patch metapopulation for simplicity. After describing the model in section 2, we first show that the inclusion of the juvenile stage (with the consequent time delay) does not alter the stability of the equilibria as long as the juveniles do not interact with the resource (section 3.1). Pattern formation is possible, i.e., the homogeneous equilibrium can become unstable and inhomogeneous equilibria can be stable, only when both the juveniles and the adults interact with the same resource (section 3.2). In section 4, we show an example of pattern formation in large metapopulations.

Section 5 contains an alternative model, which is biologically somewhat less realistic but simpler, analytically tractable, and captures the essential elements of our first model. Here we focus on four limiting cases and show that patterned equilibria are stable under the same qualitative conditions as in the first model. In section 6, we show that the equation used by Doebeli and Killingback (2003) can be derived as a particular case of our model in section 5. This derivation gives a mechanistic underpinning to the model of Doebeli and Killingback (2003) and highlights the role of stage structure in their results.

The Appendix briefly shows yet another related model with pattern formation, which is set in continuous time without seasonality. We use this model only to investigate the relationship between the present mechanism and Turing-type diffusive instability (see Discussion).

2 The model

2.1 Assumptions

We consider a metapopulation occupying \( n \) habitat patches that lie on a circle, such that each patch has two direct neighbors and the \( 1st \) and \( nth \) habitat patches adjoin. The environment is homogeneous, i.e., the conditions in each patch are assumed to be the same.

Each habitat patch \( i \) \((i = 1, \ldots, n)\) is inhabited by a consumer population feeding on a single resource with abundance \( R_i \). The consumer population consists of eggs (with density \( E_i \)), juveniles \( (y_i) \), and adults \( (x_i) \). Adults consume the resource in their home patch \( i \), but spend also a fraction of their foraging time in the two neighboring patches \( i - 1 \) and \( i + 1 \) (quasi-local competition). The adults convert the resource into eggs, which are laid in the home patch. The eggs are completely inert during the season and hatch only at the beginning of the next year. Juveniles eat the same resource as the adults, but they do not forage in patches other than their home patch. Juveniles reach maturity after one year. Eggs and juveniles may die during the reproductive season as well as in winter; for simplicity, however, we assume that adult consumers die only in winter but not during the season (see Geritz and Kisdi, 2004, on the consequences of relaxing this assumption).
The model has two parts, continuous-time differential equations for the within-season dynamics (1) - (4) and discrete-time equations for the between-season dynamics (5) - (8) as in Geritz and Kisdi (2004). Let $m$ denote the year and $t$ the time within a season; $t$ runs from 0 to 1. For patches $i = 1, \ldots, n$, the within-season dynamics is given by

$$
\frac{dR_i^{(m)}(t)}{dt} = \alpha \cdot \left[ R_i^{(m)}(t) f \left( R_i^{(m)}(t) \right) 
- \beta R_i^{(m)}(t) \left( (1 - p)x_i^{(m)}(t) + \frac{p}{2} \left( x_{i-1}^{(m)}(t) + x_{i+1}^{(m)}(t) \right) \right)
- \delta R_i^{(m)}(t) y_i^{(m)}(t) \right]
$$

(1)

$$
\frac{dE_i^{(m)}(t)}{dt} = \gamma \beta x_i^{(m)}(t) \left( (1 - p)R_i^{(m)}(t) + \frac{p}{2} \left( R_i^{(m)}(t) + R_{i+1}^{(m)}(t) \right) \right)
- \mu E_i^{(m)}(t)
$$

(2)

$$
\frac{dy_i^{(m)}(t)}{dt} = -\eta \left( R_i^{(m)}(t) \right) y_i^{(m)}(t)
$$

(3)

$$
\frac{dx_i^{(m)}(t)}{dt} = 0
$$

(4)

Here $\alpha R_i^{(m)}(t) f \left( R_i^{(m)}(t) \right)$ describes resource dynamics in absence of the consumer, with the function $f$ specified below. To ease the calculations below, we have factored out $\alpha$ in the right hand side of (1); accordingly, $\alpha \beta$ and $\alpha \delta$ denote the consumption rates of adult and juvenile consumers, respectively. Since the per capita rate of consumption is $\alpha \beta R$ and the per capita rate of egg production is $\gamma \beta R$, $\gamma / \alpha$ is the conversion factor of food into eggs. The adults spend a fraction $p$ of their time by foraging outside their home patch. For $n > 2$, this time is split evenly between the two neighboring patches, such that $(1 - p)$ of foraging time is spent in the home patch and $p/2$ of time is spent in each of the neighboring patches. All eggs are laid in the adult’s home patch. During the season, eggs die at a constant rate $\mu$ whereas the death rate of juveniles, $\eta(R)$, depends on the amount of food they consume. No adult mortality is assumed during the season.

For the between-season dynamics, we have

$$
R_i^{(m+1)}(0) = \phi R_i^{(m)}(1)
$$

(5)

$$
E_i^{(m+1)}(0) = 0
$$

(6)

$$
\theta_i^{(m+1)}(0) = \sigma E_i^{(m)}(1)
$$

(7)

$$
x_i^{(m+1)}(0) = \xi y_i^{(m)}(1) + \theta x_i^{(m)}(1)
$$

(8)

where $\phi$, $\sigma$, $\xi$ and $\theta$ are the overwinter survival probabilities of the resource, eggs, juveniles and adults, respectively. Notice that unlike in Kisdi and Utz (2005), adults may survive and reproduce several times. For simplicity, we assume no dispersal between patches, i.e., neither juveniles nor adults change their home patch.

To complete the model, we need to specify the functions $f(R)$ and $\eta(R)$. For the resource dynamics, we assume $\alpha R f(R) = \alpha - (\alpha R / K)$, such that there is a constant inflow of the resource at rate $\alpha$ into the system and an efflux at a per capita rate $\alpha / K$; without consumers, the equilibrium
resource level is then $K$. The juveniles’ death rate is assumed to decrease with increasing resources according to the positive function $\eta(R) = \frac{a}{b+R} + c$. Note that the death rate remains finite when there are no resources at all ($\eta(0) = \frac{a}{b} + c$) and there is natural juvenile death even if plenty of resources are available ($\lim_{R \to \infty} \eta(R) = c$).

Without loss of generality, we can simplify the model by scaling resource density such that $K = 1$ and scaling consumer density such that $\beta = 1$. Since the density of adults remains constant within a season, we shall suppress $t$ in connection with $x$.

Following Geritz and Kisdi (2004), we assume that the within-season dynamics of the resource is much faster than the dynamics of the consumer, i.e., we assume that $\alpha$ is large. Then $R^i((m))i(t)$ can be approximated by its time-varying quasi-equilibrium,

$$\hat{R}_i^{(m)}(t) = \frac{1}{1 + (1 - p)x_i^{(m)} \frac{p}{2} (x_i^{(m)} + x_i^{(m+1)}) + \delta y_i^{(m)}(t)}$$

which tracks the changes in juvenile density during the season. Because of the time scale separation between the resource and the consumer dynamics, the value of $\gamma > 0$, the overwinter survival probability of the resource, is irrelevant.

In the virgin environment, $\hat{R} = 1$ and the lifetime reproductive success of an adult is

$$L = \frac{1}{1 - \theta} \cdot \sigma \frac{\gamma}{\mu} (1 - e^{-\mu}) \cdot \xi e^{-(\frac{a}{R+1} + c)}$$

where $\frac{1}{1 - \theta}$ is the expected lifetime of an adult, $\sigma \frac{\gamma}{\mu} (1 - e^{-\mu})$ is the number of surviving eggs per adult per season and $\xi e^{-(\frac{a}{R+1} + c)}$ is juvenile survival. The consumer is viable if $L > 1$, which we shall always assume in the subsequent analysis. Note that for $L < 1$, the nontrivial equilibria of the model assume negative values; when $L = 1$, the homogeneous equilibria undergo a transcritical bifurcation, i.e., they coincide with the trivial equilibrium.

### 2.2 Dynamics of an isolated population

Let us first briefly look at an isolated population ($n = 1$) before investigating metapopulations. Assume that juvenile consumption is negligible ($\delta = 0$) and juvenile mortality does not depend on the resource ($a = 0$) for example because juveniles utilize a different and non-limiting resource. The resource then attains the constant quasi-equilibrium $\hat{R}^{(m)} = \frac{1}{1 + x^{(m)}}$ and the within-season dynamics in (2) – (4) can easily be integrated. For the between-season dynamics of adults one obtains the second order difference equation

$$x^{(m+2)} = \frac{(1 - \theta)Lx^{(m)} + \theta x^{(m+1)}}{1 + x^{(m)}}$$

The model has two fixed points, $(x^0, y^0) = (0, 0)$ and $(x^*, y^*) = ((L - 1), \frac{1 - \theta}{\xi e^{-\theta}} x^*)$. The trivial equilibrium $(x^0, y^0)$ is unstable and the non-trivial equilibrium $(x^*, y^*)$ is asymptotically stable whenever the population is viable ($L > 1$). Notice that with semelparous adults ($\theta = 0$), the second order map $x^{(m)} \mapsto x^{(m+2)}$ in (11) has the form of the Beverton-Holt (1957) model. Although in effect two independent populations live together (one reproduces in odd years and the other in even years), both equilibrate at the globally stable fixed point of the Beverton-Holt model.
and Kisdi (2004) derived the Beverton-Holt dynamics for the first order map $x^{(m)} \mapsto x^{(m+1)}$ from a consumer-resource model similar to ours but without the juvenile stage. The presence of juveniles introduces a time delay of one year but does not alter the qualitative behavior of the model as long as the juveniles do not interact with the resource ($\delta = 0$ and $a = 0$). This is of importance because the first order Beverton-Holt map formed the basis of the metapopulation models of Doebeli and Killingback (2003) and of Kisdi and Utz (2005), with whom we shall contrast the present results.

If the juveniles interact with the resource such that $\delta > 0$ and/or $a > 0$, then the non-trivial fixed point cannot be found analytically and thus the model has to be investigated by numerical methods. Juveniles can destabilize the fixed point when they interact with the resource, and therefore the stage-structured population can exhibit population cycles. This happens if the juveniles consume the resource heavily but depend on it only weakly ($\delta$ is large but $a$ is small) and also in the converse case, i.e., if juveniles consume little but their survival strongly depends on the resource ($\delta$ is small but $a$ is large). In the first case, the resource is depleted and adults can hardly reproduce in years when juvenile density is large; as a consequence, there will be few juveniles in the next year but many adults, which will then produce many juveniles by the year after. In the second case, most juveniles die in years when adult density is high; there will be few adults in the next year but many juveniles, which enjoy high resource abundance and mature into many adults by the year after. These cycles are essentially the same as the single cohort dynamics found by Bulmer (1977) and analyzed in detail by Davydova et al. (2003; see Discussion). In this paper we concentrate on stationary spatial pattern formation and thus do not pursue temporal cyclic behavior further.

3 Two-patch metapopulations

We analyze the model in detail for two habitat patches ($n = 2; i, j \in \{1, 2\}, i \neq j$). In this case, pattern formation amounts to the destabilization of the homogeneous equilibrium $(x^*, y^*, x^*, y^*)$ and the stabilization of boundary equilibria where only one of the patches has a breeding population ($(\hat{x}, \hat{y}, 0, 0)$ and $(0, 0, \hat{x}, \hat{y})$). Numerical simulations show that pattern formation occurs under similar conditions also in larger metapopulations (see section 4). In this section, we first show that the homogeneous equilibrium remains stable if the juveniles do not interact with the resource, thus the mere presence of juveniles does not induce patterns. Next, we investigate pattern formation when both the juveniles and the adults interact with the same resource.

3.1 Juveniles do not interact with the resource

Here we investigate a two-patch metapopulation assuming that juvenile consumption is negligible ($\delta = 0$) and juvenile survival does not depend on the amount of available resource ($a = 0$). This is the case if juveniles feed on some other resource that is not limiting population growth.

This case can be solved fully analytically. The quasi-equilibrium of the resource is constant during the season ($\hat{R}^{(m)}_i = \frac{1}{1+(1-p)x^{(m)}_i + px^{(m)}_j}$) and $\eta(R) = c$, i.e., juveniles die at a constant rate. Integrating the within-season dynamics yields the following equations for the between-season
3.1 Juveniles do not interact with the resource

dynamics:
\[
\begin{align*}
x_i^{(m+1)} &= \xi y_i^{(m)}(0)e^{-c} + \theta x_i^{(m)}, \\
y_i^{(m+1)}(0) &= L\left(\frac{1-\theta}{\xi e^{-c}}x_i^{(m)}\right) \cdot \left(\frac{1-p}{1+(1-p)x_i^{(m)}+px_j^{(m)}} + \frac{p}{1+(1-p)x_j^{(m)}+px_i^{(m)}}\right).
\end{align*}
\]

Besides the trivial fixed point \((x^0, y^0, x^0, y^0) = (0, 0, 0, 0)\), this model has a homogeneous equilibrium \((x^*, y^*, x^*, y^*)\) where the two patches have equal population densities as well as boundary equilibria \((\hat{x}_1, \hat{y}_1, 0, 0), (\hat{x}_2, \hat{y}_2, 0, 0), (0, 0, \hat{x}_1, \hat{y}_1)\) and \((0, 0, \hat{x}_2, \hat{y}_2)\) where one of the two populations is extinct. The equilibrium densities are
\[
\begin{align*}
x^* &= (L - 1) \\
y^* &= \frac{1-\theta}{\xi e^{-c}}x^* \\
\hat{x}_{1,2} &= \frac{2p(1-p)L - 1 \pm \sqrt{1 - 4p(1-p)(1-p(1-p)L^2)}}{2p(1-p)} \\
\hat{y}_i &= \frac{1-\theta}{\xi e^{-c}}\hat{x}_i & i = 1, 2
\end{align*}
\]

Because \(\hat{x}_2\) and thus \(\hat{y}_2\) are negative, there is only one pair of biologically relevant boundary equilibria, \((\hat{x}_1, \hat{y}_1, 0, 0)\) and \((0, 0, \hat{x}_1, \hat{y}_1)\).

The trivial fixed point is unstable whenever the population is viable. At the homogeneous equilibrium \((x^*, y^*, x^*, y^*)\), the eigenvalues of the Jacobian matrix are
\[
\begin{align*}
\lambda_{1,2} &= \frac{1}{2}\left(\theta \pm \sqrt{4\left(\frac{1}{L}\right)(1-\theta) + \theta^2}\right) \\
\lambda_{3,4} &= \frac{1}{2}\left(\theta \pm \sqrt{\frac{1}{L}\left(4 + 16p(1-p)(L-1)(1-\theta) + \theta(L\theta - 4)\right)}\right)
\end{align*}
\]

\(|\lambda_{1,2}| < 1\) for \(L > 1\); \(|\lambda_3| < 1\) for \(p \neq \frac{1}{2}\) and \(|\lambda_4| < 1\) unless both \(p = \frac{1}{2}\) and \(\theta = 0\). The homogeneous equilibrium is thus generically stable. The eigenvalues at the boundary equilibria are too complicated to be shown here, but we have proved that the boundary equilibria are unstable for \(p \neq \frac{1}{2}\) (a Mathematica notebook is available on request).

In the degenerate case \(p = \frac{1}{2}\), there is a line of equilibria given by \(\frac{x_1 + x_2}{2} = (L - 1)\) and \(y_i = \frac{1-\theta}{\xi e^{-c}}x_i\ (i = 1, 2)\), which includes the homogeneous equilibrium and the boundary equilibria of the generic case. This line is attracting, but points on the line are neutral. When the adults split their time evenly between the two patches and the juveniles do not consume, the resource is depleted evenly and total population size equilibrates irrespectively of where the adults lay their eggs. If \(\theta = 0\) in addition to \(p = \frac{1}{2}\), the model shows neutral out-of-phase cycles. With semelparous adults, there are two independent metapopulations (reproducing respectively in odd and in even years), which equilibrate on the neutral line but usually at different points. Consequently, the total number of adults and the total number of juveniles are constant over time but the distribution of
individuals over the patches oscillates in a two-year cycle. Recall that with semelparous adults but \( p \neq \frac{1}{2} \), the metapopulations reproducing in odd and in even years both equilibrate to the stable homogeneous fixed point.

We conclude that if the juveniles do not affect the resource level and the resource does not affect juvenile survival, then generically the homogeneous equilibrium is the only stable equilibrium. The model of Kisdi and Utz (2005), which did not include the juvenile stage, gives the same result (except the neutral cycles of a highly degenerate case); even the equilibrium densities of adults are the same in the two models. As in the case of a single isolated population (section 2.2), the time delay caused by the juvenile stage does not alter the behavior of the model per se.

### 3.2 Juveniles interact with the resource

Let us now consider the full model where juveniles consume a non-negligible amount of the resource \((\delta > 0)\) and their survival depends on resource abundance \((a > 0)\). With \( a > 0 \), the fixed point of the between-season dynamics cannot be given explicitly; and with \( \delta > 0 \), we cannot solve the differential equations for the eggs and juveniles analytically. Therefore we investigated the model numerically using the software package *Mathematica* (Wolfram Research). As we concentrate on the role of juveniles in spatial pattern formation, we use \( \delta, a \) and \( p \) as bifurcation parameters and fix the values of \( b, c, \mu, \sigma, \xi, \theta \) and \( L \). The latter determines the value of \( \gamma \); notice that as we vary \( a \), we also vary \( \gamma \) such that \( L > 1 \) stays constant and the population remains viable.

In the numerical procedure, we choose a value for \( \delta \) and start with locating an approximate equilibrium for \( a = 0 \) and \( p = 0 \) by simulation. This approximate result is used to obtain the precise equilibrium densities by numerically integrating the within-season dynamics and finding the root of the between-season difference equations. To establish the stability of the equilibrium, we differentiate the between-season equations numerically and calculate the eigenvalues of the Jacobian matrix. Next, we change the values of \( p \) and \( a \) incrementally and use a continuation technique to find the equilibria and their stability.

The results of the numerical analysis are shown in Fig. 1. Stationary pattern formation occurs in the shaded areas of the parameter space. In these areas, the homogeneous equilibrium \((x^*, y^*, x^*, y^*)\) is unstable and two inhomogeneous (boundary) equilibria, \((\hat{x}, \hat{y}, 0, 0)\) and \((0, 0, \hat{x}, \hat{y})\), are locally asymptotically stable. As before, one of the two patches is empty at the inhomogeneous equilibrium in the sense that no adult lays eggs here and there are no juveniles, but both patches contain foraging adults. The dynamics of a metapopulation where only one patch has a breeding population is thus different from a single isolated population unless \( p = 0 \). Outside the shaded areas the boundary equilibria are unstable, but the homogeneous equilibrium is not stable everywhere.

Next to the homogeneous and inhomogeneous equilibria, the model has also cyclic attractors. When an isolated patch exhibits cyclic dynamics (see section 2.2), then the two-patch metapopulation must have both in-phase and out-of-phase cycles for \( p = 0 \) and, by continuity, also for sufficiently small values of \( p \). There is a part of the parameter space where neither the homogeneous nor the inhomogeneous equilibria are stable; all orbits must then converge to some non-equilibrium attractor (within the parameter range shown in Fig. 1, this occurs for large values of \( \delta \) below the dotted lines). Moreover, for some parameters a cyclic attractor exists simultaneously with the locally stable inhomogeneous equilibria such that it depends on the initial population densities whether the metapopulation converges to a stationary pattern or exhibits sustained os-
Figure 1: Stationary pattern formation in two-patch metapopulations. Stable patterns exist, i.e., the inhomogeneous equilibria are locally asymptotically stable inside the shaded areas. In the parameter range shown, the homogeneous equilibrium is stable outside the shaded areas but for large values of δ, only above the dotted line. The inhomogeneous equilibria are unstable outside the shaded areas. Other parameter values are b = c = 1, μ = σ = ξ = θ = 0.2, L = 5.5.
cillations. In the remainder of this article, however, we focus on stationary patterns only.

In Fig. 1, there exist two distinct areas where stationary patterns are stable: There is an upper region that shrinks with increasing $\delta$ and disappears at about $\delta = 0.015$ and there is a lower region that increases as $\delta$ increases. These two areas correspond to two different mechanisms of pattern formation. For a heuristic interpretation, assume that the first patch is occupied by a large population but the second patch contains only a small population. Consider first the upper region, i.e., assume that $\delta$ is small but $a$ is large: Juveniles consume only a small amount of the resource, but this consumption is nevertheless important for their survival. When $p$ is sufficiently large, adults from the first patch deplete the resource in the second patch. Juveniles in the second patch have high mortality, thus the small population of the second patch declines further and the metapopulation attains an inhomogeneous (boundary) equilibrium.

Consider now the situation when $\delta$ is large and $a$ is small, as in the lower regions of pattern formation in Fig. 1. In this case juveniles consume the resource more heavily than adults, but juveniles suffer less when the resource is in short supply (e.g. they can switch to an alternative resource when necessary). This situation is thus the reverse of the previous one. When there are many juveniles in the first patch, they deplete the local resource, but this does not harm them and, assuming that $p$ is large, does not harm the adults of the first patch since they mainly forage in the second patch. The adults of the second patch, however, find little food when they forage in the first patch. A small population in the second patch thus will decline and the inhomogeneous equilibrium is stable. For intermediate values of $\delta$ both mechanisms are at work; when $\delta$ increases, the first mechanism weakens and hence the upper region decreases, whereas the second mechanism becomes stronger and the lower region grows.

In a two-patch metapopulation, both mechanisms work only if $p$ exceeds $\frac{1}{2}$, i.e., if adults spend more time in the other patch than in their home patch. This may in fact be adaptive (to decrease competition with the immobile juveniles), but may nevertheless look less realistic for many biological systems. In larger metapopulations, however, $p$ exceeds $\frac{1}{2}$ if e.g. adults split their foraging time evenly among the three patches they can reach, i.e., the home patch and the two neighboring patches.

In the model of Doebeli and Killingback (2003), pattern formation occurs whenever $p$ exceeds $\frac{1}{2}$. In our model, the inhomogeneous equilibria are stable for all $p > \frac{1}{2}$ only if either $\delta = 0$ and $a$ is sufficiently large or $\delta$ is large and $a = 0$ (Fig. 1). These two cases thus qualitatively correspond to the model of Doebeli and Killingback (2003; see also sections 5-6 below). In both models, the homogeneous equilibrium can be destabilized only if the adults spend more than half of their foraging time outside their home patch.

4 Large metapopulations

We investigated pattern formation in a larger metapopulation ($n = 10$) relying on simulations only. In Fig. 2, the parameters are chosen such that a two-patch metapopulation would exhibit a stable pattern. The 10-patch model may converge to a number of inhomogeneous equilibria for the same parameter values, depending on the initial values of $x_i^{(1)}$ and $y_i^{(1)}(0)$ ($i = 1, \ldots, 10$). Some of these equilibria are simply shifted along the row of patches (see the panels (c) – (e) of Fig. 2). The groups of shifted equilibria are analogous to the pairs of boundary equilibria in the two-patch model. Although shifts are mathematically trivial, for the population in a certain patch it makes a big difference on which attractor the metapopulation settles. Other equilibria
Figure 2: Different stationary attractors of a 10-patch metapopulation. The bars show the density of adults that reproduce in a given patch, $x_i$. Parameter values are $\delta = 0.1$, $a = 1$, $p = 0.8$, $b = c = 1$, $\mu = \sigma = \xi = \theta = 0.2$, $L = 5.5$.

are qualitatively different (panels (a) and (b) of Fig. 2), with different arrangement and population size of patches occupied by breeding populations (recall that empty patches also contain foraging adults). The existence of multiple stationary attractors is very similar to the results of Doebeli and Killingback (2003).

5 An alternative model

The model we investigated above is a natural extension of Geritz and Kisdi (2004) and Kisdi and Utz (2005) to populations with separate juvenile and adult stages. This extension had to be analyzed numerically, because (for $\delta > 0$) the within-season dynamics could not be integrated explicitly.

In this section, we consider an alternative model where we can obtain limiting results analytically. Moreover, in this alternative model we do not need to assume particular functions for the resource dynamics ($f(R)$) and for the juvenile death rate ($\eta(R)$): The results of this section hold for arbitrary decreasing functions (see below).

To achieve such general results, we need to assume that within each season, the number of juveniles attains an equilibrium. Biologically, this assumption may be less realistic: it implies that not only the resource but also juvenile density changes fast on a time scale where adult density is assumed constant. Mathematically, however, this assumption eliminates the need for integrating the within-season dynamics and thereby greatly simplifies the analysis.
We further simplify the model by eliminating the inert egg stage. In this model, we shall thus assume that adults produce juveniles throughout the season depending on how much resources they consume; juveniles die at rate $\eta(R)$ whereas adult density stays constant. The dynamics within a season is given by

\[
\frac{dR_i(t)}{dt} = R_i(t) f(R_i(t)) \tag{20}
\]
\[
- \beta R_i(t) [(1 - p)x_i + (p/2)(x_{i-1} + x_{i+1})] - \delta R_i(t)y_i(t)
\]
\[
\frac{dy_i(t)}{dt} = x_i \phi ((1 - p) R_i(t) + (p/2)(R_{i-1}(t) + R_{i+1}(t)) \tag{21}
\]
\[
- \eta (R_i(t)) y_i(t)
\]
\[
\frac{dx_i(t)}{dt} = 0 \tag{22}
\]

Here $\phi(R)$ is the number of juveniles produced by an adult who consumed $\beta R \cdot dt$ resources in time $dt$. Birth rate may be proportional to the amount of resource consumed ($\phi(R) = \gamma \beta R$ as in our first model) but this is not necessary: We assume only that $\phi(R)$ is an increasing function. The resource dynamics need not be specified, but we assume that $f(R)$ is strictly decreasing (no Allee-effects) and that resource density is scaled such that its carrying capacity is 1 ($f(1) = 0$). The death rate of juveniles, $\eta(R)$, is either constant or an arbitrary decreasing function of resource density that assumes non-negative values.

The within-season dynamics in (20) - (22) is run to equilibrium. At the beginning of the next season, we have

\[
R_i^{(m+1)}(0) = \bar{R}^{(m)}_i \tag{23}
\]
\[
y_i^{(m+1)}(0) = 0 \tag{24}
\]
\[
x_i^{(m+1)} = \xi y_i^{(m)} + \theta x_i^{(m)} \tag{25}
\]

where bars denote the equilibrium densities obtained from (20) - (22); note that the value of $\varrho > 0$ is irrelevant due to the assumption of within-season equilibrium.

In this section, we consider only two patches and only the local stability of stationary patterns. Therefore, we investigate the local stability of the inhomogeneous equilibrium $(\hat{x}_1, 0)$ against the invasion of a population breeding in patch 2, assuming that the equilibrium is stable with respect to perturbations of $x_1$. Note, however, that the latter condition does not always hold: The model can exhibit cyclic behavior in the discrete between-year dynamics.

Below we consider four limiting cases of the model, which differ whether the juveniles or the adults deplete the resource and whether the juveniles or the adults depend on the resource. In comparison to our first model (Fig. 1), these four cases correspond respectively to (1) $\delta = 0$, large $a$; (2) large $\delta$, $a = 0$; (3) $\delta = 0$, $a = 0$; and (4) large $\delta$, large $a$.

**Case 1: Only the adults deplete the resource but only the juveniles depend on it.** Assume that juvenile consumption is negligible ($\delta = 0$) but juvenile mortality nevertheless depends on the little food juveniles consume ($\eta(R)$ is strictly decreasing). Adults consume the resource ($\beta > 0$) but their reproduction does not depend on it ($\phi(R) \equiv k$ is constant); this is possible if the adults can use also an alternative resource for reproduction when necessary. If patch 2 has no breeding...
When patch 2 has no breeding population, we have $f(\bar{R}_2) = \beta p x_1$ and $\bar{y}_1 = k x_1 / \eta(\bar{R}_1)$. The between-season dynamics of adults is thus given by

$$x_1^{(m+1)} = \left[ \frac{\xi k}{\eta (f^{-1}(\beta (1-p)x_1^{(m)}) + \theta) x_1^{(m)}} \right] x_1^{(m)}$$

(26)

and the fixed point $\hat{x}_1$ can be determined by setting the expression between the brackets equal to 1.

Analogously, the between-year dynamics of a small population introduced into patch 2 when the population in patch 1 has reached $\hat{x}_1$ is $x_2^{(m+1)} = \left[ \frac{\xi k}{\eta (f^{-1}(\beta (1-p)x_1^{(m)}) + \theta) x_2^{(m)}} + \theta \right] x_2^{(m)}$. This population dies out if and only if

$$\frac{\xi k}{\eta (f^{-1}(\beta px_1^{(2)}) + \theta)} + \theta < 1 = \frac{\xi k}{\eta (f^{-1}(\beta (1-p)x_1^{(2)}) + \theta)}$$

(27)

Because $f(R)$ and $\eta(R)$ are strictly decreasing, this condition is equivalent to $p > 0.5$. The pattern $(\hat{x}_1, 0)$ is thus stable against a perturbation of $x_2$ whenever $p$ is greater than a half. (Recall that in larger metapopulations, $p > 0.5$ is achieved e.g. if the adults split their foraging time equally between the home patch and its two neighboring patches.)

**Case 2: Only the juveniles deplete the resource but only the adults depend on it.** This is the opposite case: Here we assume that juveniles consume the resource ($\delta > 0$) although their survival does not depend on their consumption ($\eta(R) \equiv h$ is constant). The consumption of adults is negligible ($\beta = 0$), yet their reproduction depends on the very small amount they eat of this resource ($\phi(R)$ is strictly increasing).

When patch 2 has no breeding population, then it has no juveniles and therefore $\bar{R}_2 = 1$. In patch 1, the equilibrium density of juveniles is $\bar{y}_1 = x_1 \phi ((1-p)\bar{R}_1 + p) / h$, and the between-season dynamics of adults is given by

$$x_1^{(m+1)} = \left[ (\xi / h) \phi ((1-p)\bar{R}_1^{(m)} + p) + \theta \right] x_1^{(m)}$$

(28)

A small population in patch 2, introduced when the population in patch 1 has attained its fixed point $\hat{x}_1$, is exponentially declining if and only if

$$(\xi / h) \phi (p\bar{R}_1 + (1-p)) + \theta < 1 = (\xi / h) \phi ((1-p)\bar{R}_1 + p) + \theta$$

(29)

Since $\bar{R}_1 < 1$ at the inhomogeneous equilibrium $(\hat{x}_1, 0)$ and $\phi(R)$ is increasing, the above condition is equivalent to $p > 0.5$. Just as in the previous case, the pattern is stable whenever $p$ exceeds 0.5.

**Case 3: Juveniles do not interact with the resource.** Now we assume that juveniles neither consume the resource ($\delta = 0$) nor depend on it ($\eta(R) \equiv h$ is constant), whereas adults deplete the resource ($\beta > 0$) and their reproduction is affected ($\phi(R)$ is strictly increasing). When patch 2 has no breeding population, we have $f(\bar{R}_1) = \beta (1-p)x_1$, $f(\bar{R}_2) = \beta px_1$ and $\bar{y}_1 = x_1 \phi ((1-p)\bar{R}_1 + p\bar{R}_2) / h$ in the within-season equilibrium. The between-season dynamics of adults is then

$$x_1^{(m+1)} = \left[ (\xi / h) \phi ((1-p)f^{-1}(\beta (1-p)x_1^{(m)}) + p f^{-1}(\beta px_1^{(m)})) + \theta \right] x_1^{(m)}$$

(30)
A small population introduced in patch 2 cannot invade if and only if

\[
(\xi/h)\phi \left( (1 - p)f^{-1}(\beta px_1^{(m)}) + pf^{-1}(\beta(1 - p)x_1^{(m)}) \right) + \theta < 1 = (\xi/h)\phi \left( (1 - p)f^{-1}(\beta(1 - p)\hat{x}_1) + pf^{-1}(\beta p\hat{x}_1) \right) + \theta
\]  

Because \(\phi(R)\) is increasing, this condition is equivalent to

\[
(1 - 2p)f^{-1}(\beta p\hat{x}_1) < (1 - 2p)f^{-1}(\beta(1 - p)\hat{x}_1)
\]

where \(f^{-1}\) is decreasing. If \(p < 0.5\), this condition simplifies to \(p > 1 - p\), which is impossible. If \(p > 0.5\), then the condition reduces to \(p < 1 - p\), which is also contradictory. The pattern \((\hat{x}_1, 0)\) is therefore not stable against an invading population in patch 2 (\(p = 0.5\) is neutral). The case of inactive juveniles is similar to the model of Kisdi and Utz (2005) and section 3.1 above, except for the assumption of within-season equilibrium. In agreement with the previous models, the pattern is not asymptotically stable for any value of \(p\) and neutrally stable for \(p = 0.5\).

**Case 4: Adults do not interact with the resource.** If juveniles consume the resource (\(\delta > 0\)) and depend on it (\(\eta(R)\) is strictly decreasing) but adults consume only an alternative and non-limiting resource such that \(\beta = 0\) and \(\phi(R) \equiv k\) is constant, then the within-season dynamics simplifies to

\[
\frac{dR_i(t)}{dt} = R_i(t)f(R_i(t)) - \delta R_i(t)y_i(t)
\]

\[
\frac{dy_i(t)}{dt} = kx_i - \eta(R_i(t))y_i(t)
\]

Quasi-local competition \((p)\) plays no role in this case. Each patch equilibrates independently of the other, and an empty patch can always be invaded: No pattern is stable.

### 6 Mechanistic underpinning to the model of Doebeli and Killingback (2003)

Doebeli and Killingback (2003) investigated quasi-local competition using the model

\[
x_i^{(m+1)} = \frac{\lambda x_i^{(m)}}{1 + A \left[ (1 - p)x_i^{(m)} + \frac{p}{2}(x_{i-1}^{(m)} + x_{i+1}^{(m)}) \right]}
\]  

(cf. their equation (8)). This model can be derived as a special case of our model in section 5 as follows. Let \(f(R) = \alpha(1 - R/K)\), \(\eta(R) = a - bR\), and let \(\phi(R) = k\) constant, \(\delta = 0\) and \(\theta = 0\) in equations (20)-(22) and (23)-(25). These assumptions correspond to Case 1 of the previous section with a logistically growing resource and a linear relationship between resource abundance and juvenile death rate. Because \(\eta(R)\), the death rate of juveniles, should not become negative in the range of interest, assume further that \(a - bK \geq 0\). Under these assumptions, equations (20) - (22) yield

\[
y_i^{(m)} = \frac{kx_i^{(m)}}{a - bK + \frac{bK}{\alpha} \left[ (1 - p)x_i^{(m)} + \frac{p}{2}(x_{i-1}^{(m)} + x_{i+1}^{(m)}) \right]}
\]
for the the within-season equilibrium of juveniles. With semelparous adults ($\theta = 0$), the between-year dynamics of adults is simply $x_i^{(m+1)} = \xi y_i^{(m)}$ and therefore has the form of the Doebeli-Killingback equation in (35) with parameters $\lambda = \frac{\xi k}{a-b K}$ and $A = \frac{\delta b K}{a(a-b K)}$.

7 Discussion

We investigated pattern formation under quasi-local competition in a metapopulation where local populations have a simple stage-structure: We assumed that a fixed-length juvenile period precedes maturation and only adults exert competition outside their home patch. In our main model (sections 2-4), we found that the homogeneous equilibrium can be destabilized and inhomogeneous (boundary) equilibria can be locally stable in a metapopulation with quasi-local competition under two sets of conditions: either

(1) mainly the adults deplete the limiting resource and the shortage of resource harms mainly the juveniles

or

(2) mainly the juveniles deplete the limiting resource and the shortage of resource harms mainly the adults.

In the first case, the adult stage is the "critical" stage (Charlesworth, 1980) that has the largest impact on the limiting resource whereas the juvenile stage is the most "sensitive" stage. In the second case, the roles are reversed. At a first glance, it may seem counterintuitive that impact and sensitivity can differ, for example that individuals may consume a resource and yet not be much harmed when the resource is in short supply. This can however be the case when there is another, substitutable resource available. It is also possible that consumption is small, yet this little amount is essential. The potential difference between impact and sensitivity has profound implications on species coexistence (Leibold, 1995; Meszéna et al., 2006) and on the dynamics of single populations with stage-structure (Davydova et al., 2003; see below). We find that the same difference is essential also in pattern formation under quasi-local competition in metapopulations.

The results of our main model (sections 2-4) are corroborated by a simpler alternative model described in section 5. A considerable advantage of the analysis in section 5 is that we did not have to commit ourselves to specific functions to describe resource dynamics and the resource-dependent mortality rate of juveniles.

In both models, we assumed that for adults, the amount of available resource affects fecundity but not survival. Adult mortality was assumed to occur between, but not during, the reproductive seasons. Relaxing these simplifying assumptions could lead to much more complicated dynamics even in a single, isolated population (Wikan and Mjolhus, 1995; Geritz and Kisdi, 2004); we wanted to avoid this complication in order to focus on stationary patterns. As a further simplification, we assumed no dispersal between the patches, in the sense that offspring have always the same home patch as their parents. Passive dispersal would likely hinder pattern formation (as in the models of Doebeli and Killingback, 2003 and Kisdi and Utz, 2005; see however Hastings, 1992).

In Fig. 1, we performed a bifurcation analysis of equilibria in our main model with respect to three key parameters, $p$ (the fraction of time adults spend foraging outside their home patch), $a$ (how strongly juvenile survival depends on the resource) and $\delta$ (how much juveniles deplete
the resource). Unfortunately, the model has seven more parameters \((b, c, \mu, \sigma, \xi, \theta, \text{and } \gamma)\); the latter we varied simultaneously with \(a\) such that \(L\), the lifetime reproductive success in a virgin environment, stays constant and greater than 1 in order to ensure viability). A full bifurcation analysis of the model would therefore be prohibitively demanding. By continuity, however, we know that small changes in the fixed parameters of Fig. 1 will not lead to qualitative differences in the bifurcation diagrams. Similarly, introducing small dispersal between the patches will not destroy the patterns. In contrast to the model of Kisdi and Utz (2005), here we find robust patterns that do not depend on fine-tuning the model parameters.

Pattern formation includes the destabilization of the homogeneous equilibrium as well as the stabilization of patterns. In Fig. 1, the homogeneous equilibrium is always unstable when the inhomogeneous equilibria are stable (i.e., in the shaded areas). It does not follow, however, that every perturbation of the homogeneous equilibrium is attracted to a patterned equilibrium, because the system can also exhibit cyclic dynamics. Indeed, we found that an in-phase cycle can be stable also simultaneously with the inhomogeneous equilibria (data not shown).

Pattern formation found in the model of Doebeli and Killingback (2003) corresponds to the stable patterns we find in Case 1 of our alternative model in section 5, and is similar to pattern formation in our main model with \(\delta = 0\) and large \(a\). While our results are close to those of Doebeli and Killingback (2003), we could find a mechanistic underpinning to that model only by assuming stage-structured populations where different stage classes compete for the same resource with different impacts and sensitivities. In section 6, we derived the model of Doebeli and Killingback (2003) explicitly from our simplified model in section 5, assuming that adults deplete a resource that is important for juvenile survival and using a particular choice of functions for the resource dynamics and for the death rate of juveniles. As shown by this derivation as well as by the negative results of Kisdi and Utz (2005) and section 3.1, the stage-structure is essential for pattern formation. For a heuristic explanation of this fact, notice that the model of Doebeli and Killingback (2003; see equation (35) above) assumes that individuals of a focal patch exert competitive influence on the neighboring patches without being affected by competition experienced outside the home patch (the denominator of (35) above) assumes that individuals of a focal patch exert competitive influence on the neighboring patches without being affected by competition experienced outside the home patch (the denominator of (35) contains competition in the focal patch only; see Kisdi and Utz, 2005). Exerting competition while not being affected by competition is hard to realize in an unstructured population where all individuals are alike, but possible to obtain, as a limiting case, when the population is structured such that different individuals have different impacts and sensitivities. In the present case, mobile adults exert competition in the neighboring patches but they are not sensitive to competition.

An important common conclusion of Doebeli and Killingback (2003) and the present study is that in large metapopulations (section 4, Fig. 2), a large number of locally stable equilibria exist for the same set of parameter values. An important difference is, however, that our main model (sections 2-4) often exhibits cyclic dynamics, and cycles may occur also in the alternative model of section 5 with certain choices of functions. This is not the case in the model of Doebeli and Killingback (2003). By continuity, cycles will not occur in our alternative model if functions are chosen to be sufficiently similar to those assumed in section 6 to derive the equation used by Doebeli and Killingback (2003).

### 7.1 Alternative mechanisms of pattern formation

The best-known mechanism of pattern formation is diffusive instability (Turing, 1952). For stationary patterns to form in a reaction-diffusion system, there must be (at least) two interacting
7.1 Alternative mechanisms of pattern formation

chemicals, species, etc., with the following three properties (Segel and Jackson, 1972; see e.g. Edelstein-Keshet, 1988, for review): (i) diffusion rates must be different, (ii) all interactions between the participants (including e.g. competition and reproduction) are local, and (iii) without diffusion, when the dynamics are given by an ODE system, the Jacobian must have one of two possible sign structures, namely either one row or one column of the Jacobian must contain positive elements and the other row or column must contain negative elements.

In our models, there are two different active stages and they have contrasting mobility as only adults forage outside their home patch; this is similar to (i). There is however an important difference between quasi-local competition and diffusion regarding (ii): We assume that adults foraging outside the home patch deplete the resource of adjacent patches, but produce offspring in the home patch. These adults thus have a negative effect on the adjacent patches via competition and, at the same time, a positive effect on the home patch via reproduction. In contrast, reaction-diffusion models assume that individuals affect the population only at one place at a time.

Because quasi-local competition violates property (ii) above, the dynamics in a single isolated patch need not conform with the two possibilities in (iii). In the Appendix, we briefly describe a model closely related to the ones analyzed in the main text, but set in continuous time. This facilitates the comparison with the continuous-time models of diffusive instability. We indeed find that in our model, the sign structure of the Jacobian is different from the two types that are possible with diffusive instability.

Due to the assumption of fast resource dynamics, the present model is in essence a single-species model; the densities of juveniles and adults at time $t$ unequivocally determine the quasi-equilibrium resource abundance and hence juvenile mortality and adult fecundity at $t$. Pattern formation is known to occur in a single species if quasi-local competition operates together with local Allee-effects (Gyllenberg et al., 1999) or, in continuous space, if the characteristic range of competition is larger than that of an Allee-effect (Levin and Segel, 1985; see also Britton, 1989; Furter and Grinfeld, 1989). In both cases, competition provides lateral inhibition and the Allee-effects ensure local activation, which together lead to patterns. There is lateral inhibition also in our models (either the adults suppress juveniles in the adjacent patches or juveniles of the focal patch suppress reproduction of those adults which arrive to forage from the neighboring patches). However, we do not have any Allee-effect: In a single patch, population growth is in no way enhanced by increased density of either juveniles or adults. Local activation could be substituted by locally favorable conditions when the environment is slightly heterogeneous (Gurney and Nisbet, 1976; Sasaki, 1997; Kisdi and Utz, 2005), but we assume a perfectly homogeneous environment.

Hastings (1992) found that age-dependent dispersal can destabilize the homogeneous equilibrium in age-structured metapopulations. In his two-age class model, Hastings (1992) assumed that juveniles are nondispersing whereas adults are fully mixed. The homogeneous equilibrium can then be destabilized in two different ways: Either an eigenvalue becomes less than -1 and cyclic/chaotic dynamics appear (even if the corresponding single-patch system is stable), or the same eigenvalue exceeds 1, suggesting the formation of stationary patterns. It may not be immediately obvious from Hastings’ (1992) paper (which focuses on the first possibility), but the latter case can occur only in the presence of Allee-effects. (To see this, calculate the left-hand side of Hastings’ inequality (12) explicitly. Even if juveniles reproduce, their fecundity cannot exceed 1 in equilibrium; hence the inequality can be satisfied only if the per capita fecundity of juveniles or that of adults increases with the density of juveniles at the equilibrium of a single patch.) In contrast to Hastings’ (1992) model, we obtain pattern formation without assuming Allee-effects.

Quasi-local competition amounts to some release from competition in the home patch, and
this facilitates local population growth. Passive diffusion has a similar effect as it removes competitors from densely populated areas, yet diffusion does not lead to pattern formation in single-species models. As discussed above, the key difference is whether adults foraging elsewhere still reproduce in their home patch. Quasi-local competition releases local competition but retains reproduction.

In our models, quasi-local competition leads to pattern formation only if the two stage classes have opposite impacts and sensitivities towards the common resource (cf. the two sets of conditions described at the beginning of the Discussion). In competition between species, analogous conditions lead to mutual competitive exclusion. Indeed, pattern formation in our two-patch models can be explained as competitive exclusion of one population by the other as follows (cf. 3.2; see also Doebeli and Killingback, 2003). Assume that adults deplete the resource and juveniles are sensitive to the shortage of resource (case (1) above). When \( p \) is large enough, the adults of the first population deplete mainly the resource in the second patch, on which resource the growth of the second population mostly depends. At the same time, adults of the first population do not consume much resource in the first patch, i.e., they do not harm their own juveniles. Competition between the two populations is thus stronger than competition within a population, which implies competitive exclusion. The other possibility is that juveniles deplete the resource and adults are sensitive to it (case (2) above). Then the juveniles of the first population deplete the resource at the place where the adults of the second population come to forage; at the same time, the adults of the first population are not harmed by their own juveniles because they forage in the second patch. Again, between-population competition is the stronger.

Levin (1974) described a simple way of pattern formation based on competitive exclusion. Assume that two species can mutually exclude one another, i.e., a single population can have either species 1 or species 2 in a stable equilibrium. If two patches are uncoupled, they can contain different species, resulting in patterns in both species’ abundances. By continuity, the pattern remains stable also for moderate values of dispersal.

Although both Levin’s (1974) and our mechanisms are based on competitive exclusion, note the following differences. In our case, competitive exclusion occurs between the populations that reproduce in the two patches, not between two species within the same patch. Accordingly, our mechanism does not require that a single population has multiple stable equilibria. In Levin’s model, the two homogeneous equilibria (where both patches are occupied by the same species) are always stable, whereas in our model, the (unique) homogeneous equilibrium is not always stable. The mobility of adults is essential in our case to obtain quasi-local competition, whereas in Levin’s model mobility amounts to passive dispersal and the pattern is most robust if the individuals are immobile.

When an isolated patch exhibits complex population dynamics, dispersal can lead to pattern formation in the form of spatio-temporal chaos, in which spatial patterns exhibit complicated temporal dynamics (Kaneko, 1998). However, even with complex single-patch dynamics, dispersal between patches may also stabilize the dynamics and may lead to oscillating or even static patterns in space (e.g. the "crystal lattice" pattern obtained by Hassell et al., 1991; see also Hastings, 1993; Gyllenberg et al., 1993; Doebeli, 1995; Lloyd, 1995; Kaneko, 1998; Doebeli and Ruxton, 1998). The formation of static patterns through dispersal can also be interpreted as competitive exclusion (Doebeli and Ruxton, 1998). Assume that before dispersal, there is high population density in the first patch and low density in the second. If most individuals disperse, then densities after dispersal are reversed. Under strong density dependence, the few individuals left in the first patch can produce many offspring whereas the population of the second patch crashes, which re-
stores the original pattern of densities a full generation later. By sending many dispersers, the first population keeps the second patch at low density or excludes the second population altogether. This mechanism of pattern formation works only if large populations crash (overcompensation), which is not necessary in our models. For example, the model of Doebeli and Killingback (2003), which we derive as a special case in section 6, never exhibits cycles and its single-patch version is the well known undercompensating Beverton-Holt (1957) model.

7.2 Pattern formation and single-cohort cycles

Opposite impacts and sensitivities of the stage classes are strikingly similar to the conditions under which a structured population exhibits single-cohort cycles (Bulmer, 1977; Davydova, 2003). Indeed, we also find cyclic dynamics already in a single isolated patch, and under qualitatively the same conditions as pattern formation in metapopulations (see section 2.2).

In the case of semelparous adults ($\theta = 0$), the organism in our main model (sections 2-4) is a strictly biennial species and the population consists of two isolated cohorts reproducing in even and odd years, respectively. Cycles result from competitive exclusion of one cohort by the other (Bulmer, 1977), such that in even (odd) years there are only juveniles (adults) or vice versa. Analyzing strict biennials, Davydova et al. (2003) found that the single-cohort dynamics is stable if one age class has higher (expected) competitive impact but the other age class is more sensitive to it. In this case, either cohort can competitively suppress the other because their high-impact age class is present when the other cohort has the high-sensitivity class. (Davydova et al. (2003) assumed a Ricker-type density dependence instead of explicit resource competition; because this allows for complex dynamics, they have the above result only for moderate fecundities.)

We emphasize the common structure of mutual competitive exclusion between different patches and between different cohorts of biennials. In both cases, the high-impact stage has the opportunity to suppress the competitor’s high-sensitivity stage. Under quasi-local competition, adults of one population live (mainly) together with the juveniles of the other population; during the single-cohort cycles of biennials, adults of one cohort live together with juveniles of the other cohort. When different stage classes of the competitors live together, mutual exclusion results if one class has high impact and the other class has high sensitivity.

Single-cohort cycles can also occur in longer-lived semelparous populations, such as periodical cicadas. These cycles require that one cohort can exclude all other cohorts, i.e., if the competitive effect on itself, averaged over the lifetime, is smaller than on any other cohort (Bulmer, 1977). The single-cohort dynamics of longer-lived species is analogous to a larger metapopulation where only one patch contains a breeding population. By analogy to the single-cohort case, this pattern would result if the adults venturing out from one patch deplete the resource in every other patch more than in their home patch; to this end, they must be able to reach every other patch (not only neighboring patches) and $p$ must exceed $1 - \frac{1}{n}$. Patterns with several occupied patches form much more readily in large metapopulations (Fig. 2). This would, in turn, correspond to multiple-cohort cycles; we are not aware of a detailed analysis of such cycles, but they may require unlikely changes of impact and sensitivity with age.

We find cycles in isolated patches also with iteroparous adults ($\theta > 0$), i.e., when reproduction necessarily occurs every year and there are no isolated cohorts. These cycles are qualitatively similar to the biennial case ($\theta = 0$), with the only difference that neither juveniles nor adults are fully absent in any year, only their abundances oscillate. The analysis of cycles in metapopulations where local populations are coupled by quasi-local competition (and/or dispersal) is an exciting
task but beyond the scope of this paper.

Acknowledgements

This research was financially supported by the Academy of Finland, by the German Academic Exchange Service (DAAD), and by the Computational Biology graduate school of the Ministry of Education in Finland. M.D. was supported by NSERC (Canada).

Appendix

In order to facilitate the comparison between our models and diffusive instability, here we briefly describe a model closely related to the one in sections 2-4 but set in continuous time. Accordingly, we do not assume seasonality and a fixed-length juvenile period. Juveniles mature into adults at a constant rate $m$, and adults die at a constant rate $\mu$. For simplicity, we omit the inert egg stage, i.e., adults produce juveniles. The population dynamics in a single patch is given by

$$\frac{dR(t)}{dt} = \alpha [R(t)f(R(t)) - \beta x R(t) - \delta y R(t)]$$
$$\frac{dx(t)}{dt} = my(t) - \mu x(t)$$
$$\frac{dy(t)}{dt} = \gamma \beta R(t)x(t) - my(t) - \eta(R(t))y(t)$$

where notation is as in eqs. (1)-(4). As in the main text, we assume that $\alpha$ is large and therefore the resource is always in quasi-equilibrium. The two-patch version of this model with quasi-local competition modelled as in eqs. (1)-(4) can exhibit pattern formation such that perturbations of the homogeneous equilibrium converge to one of the boundary equilibria.

When the resource is in quasi-equilibrium, the above equations reduce to a two-dimensional ODE system for $x$ and $y$. For diffusive instability, the Jacobian of the ODE system should have positive and negative elements either like $\begin{bmatrix} - & - \\ + & + \end{bmatrix}$ or like $\begin{bmatrix} - & + \\ - & - \end{bmatrix}$. Substituting the resource quasi-equilibrium and differentiating the above equations, it is easy to see that the Jacobian of our model has the signs $\begin{bmatrix} - & + \\ ? & - \end{bmatrix}$, where "?" can be either positive or negative. In either case, this sign structure does not conform with diffusive instability.

References


