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Evolution of body condition-dependent dispersal in metapopulations

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Abstract

Body condition-dependent dispersal strategies are common in nature. Although it is obvious that environmental constraints may induce a positive relationship between body condition and dispersal, it is not clear whether positive body conditional dispersal strategies may evolve as a strategy in metapopulations. We have developed an individual-based simulation model to investigate how body condition–dispersal reaction norms evolve in metapopulations that are characterized by different levels of environmental stochasticity and dispersal mortality. In the model, body condition is related to fecundity and determined either by environmental conditions during juvenile development (adult dispersal) or by those experienced by the mother (natal dispersal). Evolutionarily stable reaction norms strongly depend on metapopulation conditions: positive body condition dependency of dispersal evolved in metapopulation conditions with low levels of dispersal mortality and high levels of environmental stochasticity. Negative body condition-dependent dispersal evolved in metapopulations with high dispersal mortality and low environmental stochasticity. The latter strategy is responsible for higher dispersal rates under kin competition when dispersal decisions are based on body condition reached at the adult life stage. The evolution of both positive and negative body condition-dependent dispersal strategies is consequently likely in metapopulations and depends on the prevalent environmental conditions.

Introduction

Dispersal of organisms has important consequences for gene flow, the genetic cohesions of species, the global persistence of species in the face of local extinction, speciation, inbreeding depression, the evolution of sociality and the evolution of life-history traits (Clobert *et al.*, 2001; Kokko & Lopez-Sepulcre, 2006; Ronce, 2007). Although dispersal has been simplified towards an unconditional genetic property of individuals within metapopulations in many theoretical studies (e.g. Comins *et al.*, 1980; McPeek & Holt, 1996; Travis & Dytham, 1998), empirical work has demonstrated the existence of conditional strategies, i.e. plastic responses (Clobert *et al.*, 2009). Dispersal strategies are, for instance, adjusted in function of local environmental conditions like population density (e.g. Donohue, 1999; Andreassen & Ims, 2001; Gaggiotti *et al.*, 2002; Mennechez *et al.*, 2003), the presence of predators (e.g. Kaufman *et al.*, 2000; Powell & Frasch, 2000; Roy *et al.*, 2001; Camphuysen, 2002), parasitism (e.g. Tschirren *et al.*, 2007; Salek & Marhoul, 2008) and experienced climatological conditions (Tuda & Shima, 2002; Bonte *et al.*, 2007, 2008b). This conditional dispersal may also be dependent on an individual's internal state. Sex and age, for instance, determine conditional strategies in populations with an age- (Ronce *et al.*, 1998, 2000) or sex-biased population structure (Bowler & Benton 2005; Beirinckx *et al.*, 2006; Bonte *et al.*, 2009) or in organisms that experience sex-specific dispersal costs (Gros *et al.*, 2008). This is as expected according to the hypothesis that, unless variation in habitat quality is highly unpredictable or information acquisition is costly, the most successful strategy over evolutionary

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time has been for individuals to make dispersal decisions based on information (Dall *et al.*, 2005) obtained during their lifetime (Ronce, 2007). Obviously, organisms that are not capable of using direct cues, because they have limited sensory capacities or because they are only mobile during a limited time window, should rely on information that has been transmitted into body condition. A lowered body conditions may, for instance, provide information on increasing levels of resource competition.

When environmental conditions in the natal habitat alter the quantity and quality of dispersing individuals, profound consequences for metapopulation and metacommunity dynamics can be expected. These changes not only alter connectivity between populations and subsequent gene flow (Bohonak, 1999), but also local population dynamics (e.g. Andreassen & Ims, 2001) and metapopulation persistence (e.g. Hanski & Gilpin, 1997; Hanski, 1999). Feedback mechanisms between the quality of individual dispersers, the number of dispersers and landscape properties will eventually affect the potential for local adaptation within metapopulations (Kinnison & Hairston, 2007).

It is not well understood how interacting effects of environmental conditions on body condition (so the quality of individuals within a metapopulation) may affect dispersal (Benard & McCauley, 2008). There is empirical evidence that changes in body condition due to altered natal habitat quality may proximately constrain dispersal due to correlations with dispersal mortality during transfer, mating success and overall competitive ability in new habitat or due to negative correlations with body mass in, e.g. flying insects (Benard & McCauley, 2008). Obviously, changes in body condition may induce constraints on the dispersal ability when individuals fail to transpose energetic reserves into dispersal movement. The existence of this constraint mechanism on dispersal traits has been observed in insects (Sundstrom, 1995) and vertebrates (e.g. Belthoff & Dufty, 1998; Lena *et al.*, 1998; Meylan *et al.*, 2002; Barbraud *et al.*, 2003; Hockey *et al.*, 2003), but it remains questionable to what extent this pattern is confounded with a decreased survival during transfer. Environmental and social conditions in the natal habitat can consequently interfere with emigration, transfer and immigration processes. Of course, these observations point at proximate effects of body condition on dispersal but do not implicate that evolutionary changes are not involved in the organisms' informed dispersal decision process (Clobert *et al.*, 2009).

Unpredictability of the habitat (Poethke *et al.*, 2003), increased population densities (e.g. Donohue, 1999; Andreassen & Ims, 2001; Gaggiotti *et al.*, 2002), kin competition (e.g. Le Galliard *et al.*, 2003) and the presence of predators (e.g. Höller *et al.*, 1994; Kaufman *et al.*, 2000; Powell & Frasch, 2000; Roy *et al.*, 2001) induce evolution towards higher dispersal rates. Evidently, this evolutionary response is due to increased fitness benefits (including inclusive fitness; Hamilton & May, 1977), and is potentially caused by increased competitive abilities in the new habitat (Stamps, 2006). However, even when competitively strong individuals pay lower costs of dispersal compared with subordinate ones, it is not well understood why they engage in costly dispersal while they could easily retain their natal habitat. By contrast, the ideal despotic distribution hypothesis (Fretwell, 1972) would predict weaker, subordinate individuals to be 'kicked out' of the populations under strong local competition. Gyllenberg *et al.* (2008) demonstrated that dispersal of competitively strong individuals may be a common outcome under kin competition.

In metapopulations with inherent stochastic temporal and spatial changes in habitat quality, competitive abilities are likely to be related to fecundity; individuals that grow up in high-quality habitat have higher fecundity in the adult life stage (Pulliam & Danielson, 1991; Ferrer *et al.*, 2006). The number of surviving offspring will, in case of density-regulated population growth, depend on the population density relative to the carrying capacity K of the patch. The balance between local density, expected density in other patches and dispersal mortality is therefore expected to determine an individual's fitness expectations in metapopulations (Pulliam & Danielson, 1991).

Dispersing individuals may have higher fecundity compared with philopatric individuals when only those individuals that are in the best general condition are capable of dispersing. Trade-offs between dispersal and fecundity can be shaped by phenotypic plasticity due to, e.g. the allocation of energy for reproduction towards flight structures (for a review, see Zera & Denno, 1997) or due to the fact that individuals in the worst body condition (with eventually the lowest fecundity) are forced to leave populations at or above K. As opposed to this constraints hypothesis, adaptive strategies for fecundity–dispersal reaction norms can be expected when body condition provides direct and reliable information on the prevalent local population density conditions. This may be especially true for species that are immobile and/or lack sufficiently developed sensory organs to sample the local density (e.g. plants, sessile animals). In the butterfly *Melitaea cinxia*, positive correlations between dispersal ability and realized fecundity alternatively arise because individuals in the best condition are the dispersing phenotype and experience longer time windows for egg deposition (Saastamoinen & Hanski, 2008). For other organisms like the spider *Erigone atra*, it is less clear whether a positive relationship between fecundity and dispersal is due to environmental constraints or due to adaptive phenotypic plasticity (Bonte *et al.*, 2008b).

Because of the increasing awareness of the importance of environmental conditions in the natal habitat (Benard & McCauley, 2008), there is a clear need for theory to understand how natal effects on body condition affect the evolution of conditional dispersal strategies in spatially structured populations. Earlier theoretical research emphasized predominantly the evolution of body condition-dependent dispersal within a tight kin competition framework. Ezoe & Iwasa (1997) and Kisdi (2004) were the first to extend Hamilton & May's (1977) model of dispersal under kin competition by considering the dispersal strategy to be dependent on the number of siblings within a patch under extremely low K. Recently, Gyllenberg *et al.* (2008) showed that the dispersal of strong individuals within a kin-structured population may be the outcome under different scenarios of competition. In their model, body condition was determined by local habitat quality and related to dispersal mortality and dominance in competitive interactions. This theoretical contribution consequently points to the importance and relevance of conditional dispersal strategies, but variation in body condition is predominantly determined between patches, and the question remains which individuals within a family or a population disperse.

Our aim was to investigate how the combined effect of environmental stochasticity and dispersal mortality affect the evolutionarily stable reaction norm for dispersal propensity as a function of body condition. We therefore developed an individual-based model to extend these findings towards an explicitly spatially structured metapopulation. In the model, individual body condition is determined by local density and local habitat quality of the habitat. We did not directly model competitive benefits of those individuals in the best condition (contest competition) but instead allowed for competitive abilities that are determined by fecundity. Individuals in the best condition consequently contribute more to the next generation. In case of natal dispersal, body condition is a maternally determined trait; in case of adult dispersal (Meylan *et al.*, 2007), body condition and potential fecundity are determined during the individual's development.

The model

The landscape

For our simulation experiments we use an extended version of an individual-based model of insect dispersal in patchy landscapes of 100 habitat patches (p) with carrying capacities K (Poethke *et al.*, 2003; Bonte *et al.*, 2008a). Patch capacity was set to K = 100 individuals.

Population dynamics

Internal patch dynamics are modified after Hassell & Comins (1976). Local population dynamics are governed by density-dependent reproduction of individuals. Each generation, a female gives birth to Λ offspring, where Λ is a Poisson-distributed number with a patch- and time-specific mean, $\Lambda_{mean}(t, \text{patch})$. For each generation, the mean value of $\Lambda_{mean}(t, \text{patch})$ is drawn from a log-normal distribution with mean Λ and a standard deviation σ. In our simulations, λ was set to 4, 6 and 10, typical of arthropod demography (Bellows, 1981). σ subsequently determines the degree of environmental fluctuations which are assumed to be uncorrelated in space and time. Offspring develop into mature individuals with a density-dependent survival probability s due to contest competition:

$$s = \frac{1}{(1 + aN_p)} \quad \text{with } a = \frac{\lambda - 1}{K_p}. \tag{1}$$

Here N_p represents the population size after birth in patch p. K_p is the carrying capacity of patch p.

Body condition

We assume body condition (ζ) to be affected by: (i) local habitat quality and (ii) local density with individuals that develop under conditions lower than K to be in a better condition because they are assumed to experience less competition for resources.

$$\zeta = \frac{\wedge s}{\lambda\sigma^2} \tag{2}$$

Because we modelled body condition to be directly related to reproduction Λ, ζ is similarly density dependent. Therefore, ζ equals the ratio of realized number of offspring/maximal number of offspring in the metapopulation, with values within the range [0, 1]. The maximal body condition ζ equals one if it equals the largest achievable fecundity in the metapopulation. This is defined by the 95% upper confidence interval of log-$N(\Lambda, \sigma)$, i.e. $\Lambda\sigma^2$ (Limpert *et al.*, 2001). We chose this confidence interval in order to create a more evenly distributed variation in body condition, especially because body conditions equalling or exceeding one were never achieved due to the introduced density regulation.

Dispersal

Individuals disperse according to their body condition-determined dispersal probability d, which is constrained between the interval [0, 1]. We implemented a simple linear function between dispersal and body condition (realized fecundity):

$$d = i + r\zeta. \tag{3}$$

This function allows for flexible relationships between dispersal d and body condition (ζ), ranging between trade-offs ($r < 0$), body condition-independent dispersal ($r \sim 0$) and positive condition-dependent dispersal ($r > 0$). We assume global dispersal; i.e. a successful

disperser reaches any patch in the landscape (except its home patch) with the same probability $(1 - \mu)/(n - 1)$. Dispersal alleles were allowed to change by mutation, thus allowing for the evolution of the conditional strategy. Individuals disperse simultaneously according to their individual probability d. Two different modelling scenarios were implemented:

1 Maternal condition-dependent dispersal (MCD): body condition is determined by 'inherited' body condition of the mother (Meylan *et al.*, 2007). The quality of the mother's habitat consequently influences Λ. Dispersal takes place in the early juvenile life phase (natal dispersal).
2 Adult condition-dependent dispersal (ACD): body condition is determined by the local conditions where an individual develops. The quality of the natal patch consequently determines Λ. Dispersal takes place after maturation but before reproduction (adult dispersal).

The individual

For simplicity, individuals are parthenogenetic. Each individual is characterized by its affiliation with a specific patch (p) and by two alleles that code for, respectively, the intercept i and slope r from eqn 3. Dispersing individuals die with probability μ (dispersal mortality), regardless of patch origin. In case of MCD, offspring inherit the body condition as reflected in the maternal environmental conditions. A schematic representation of the species life cycle and feedbacks with patch attributes for both ACD and MCD is given in Fig. 1.

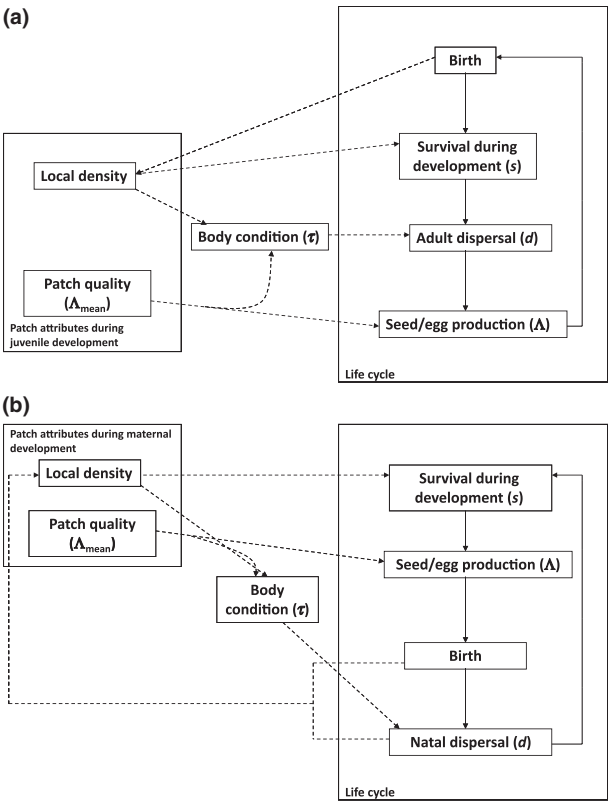

Fig. 1 Schematic representation of the implemented algorithm for the organism's life cycle and feedbacks with patch attributes. (a) ACD; (b) MCD. Dashed arrows indicate feedback mechanisms; full arrows indicate successive steps during the organism's life cycle.

Mutation rate and stochasticity

The genetic parameters have independent probabilities of mutation. To promote greater variability of genotypes in the first generations and to reduce the influence of mutations on the stability of the final result, we let mutation rates exponentially decrease from ~0.1 to < 0.001 over the course of the simulation experiments (t = 10 000 generations; for details, see Poethke & Hovestadt, 2002). Mutations change the value of i within the range [0, 1], and the value of r more gradually within the range [value − 10%, value + 10%]. No external catastrophes were simulated; instead we allowed demographic stochasticity and environmentally caused fluctuations ($1 \leq \sigma \leq 5$) in offspring number (Λ).

Parameter initialization

In the beginning , the metapopulation is initialized with 100 individuals within each of the 100 patches (N = 10 000). Initial genetic parameters are randomly drawn from a uniform [0, 1] distribution for i and from a uniform [−1, +1] distribution for s.

Simulation experiment

Simulations for both scenarios MCD and ACD were replicated (n = 100) for dispersal mortality μ = 0.1, 0.15, 0.20, 0.25, 0.30, 0.35, 0.40, 0.45 and 0.5 and environmental stochasticity σ = 1, 1.5, 2, 2.5, 3, 3.5, 4, 4.5 and 5. Because the choice of Λ (i.e. 4, 6 or 10) did not affect the qualitative results of the simulation model (D. Bonte and E. de la Peña, unpublished data), we present only outcomes for Λ = 4. For each simulation, we calculated the average allele values, the average values of body condition and the proportion of dispersing individuals. Coefficients of variation for body condition (CV_c) and slope r (CV_r) were additionally calculated in order to get an idea of their variation relative to their average value. Average local population extinction probability was calculated as the number of extinct local patches within the metapopulation at t = 10 000 divided by the total number of replicates (= 100) for the respective scenario. Evolutionarily stable strategies (visual inspection) were reached after 3000 generations in all simulation scenarios.

Results

Because body condition is inversely proportional to σ^2, it decreases with increasing environmental stochasticity for ACD and MCD. Individual variation in body condition (CV_c) within the metapopulation increased accordingly. Under the evolved conditional dispersal, we recorded only local patch extinctions with frequency < 0.01 under scenarios for $\mu = 5$ and $\sigma = 4.5$–5 (ACD) and with frequency < 0.01 under scenarios for $\sigma = 4.5$–5 (all μ, MCD).

Dispersal propensity increased as expected with environmental stochasticity and decreasing dispersal mortality. Dispersal rates decreased with increasing λ in both dispersal scenarios (overall average dispersal rates for ACD: $\lambda = 4$: 0.26, $\lambda = 6$: 0.18; $\lambda = 10$: 0.08; for MCD: $\lambda = 4$: 0.29, $\lambda = 6$: 0.17, $\lambda = 10$: 0.26). No prominent differences are obvious between scenarios for adult and natal dispersals (Fig. 2a and b), but dispersal rates increase again under high μ and low σ in the adult condition-dependent dispersal scenario (compare inlet panels Fig. 2a and b). The dispersal strategy appears to be predominantly determined by the intercept of the conditional dispersal function (Fig. 3a and b). However, under metapopulation conditions that result in dispersal rates approximating 10–20%, the dispersal reaction norm was determined for less than 50% by the genetic component (i.e. the intercept; Fig. 4a and b). Under metapopulation conditions that select for high dispersal rates, the genetic component of the reaction norm accounted for more than 90% of the observed dispersal rates (Fig. 4a and b).

Average slope allele values for the condition-dependent dispersal show similar patterns in the two scenarios: positive slopes under environmental condition of high environmental stochasticity and negative slopes under conditions of high dispersal mortality and low environmental stochasticity (Fig. 5a and b). CV for slope

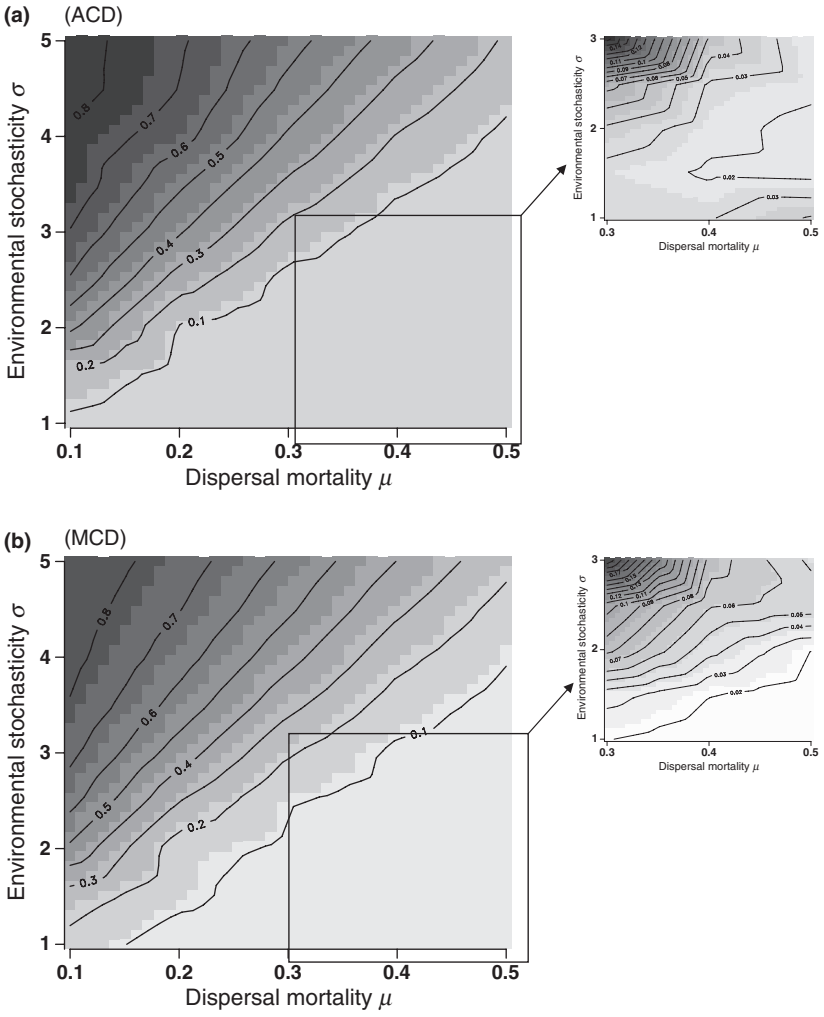

Fig. 2 Average dispersal probabilities *d* for body condition-dependent strategies in conditions when body condition is determined during juvenile development with subsequent adult dispersal [a, ACD] and when body condition is determined through maternal effects with subsequent natal dispersal (b, MCD). *d* is the result of the body condition–dispersal reaction norm (see Fig 3 and 5 for parameter values of this function). The *x*-axis gives dispersal mortality (μ), the *y*-axis environmental variability (σ).

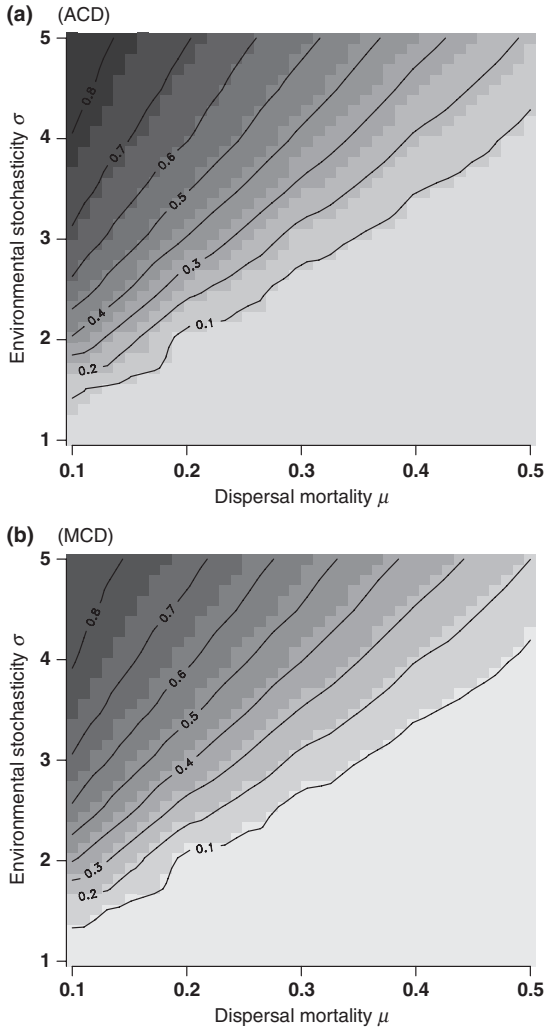

Fig. 3 Average intercept values of the body condition–dispersal reaction norms. (a) Body condition is determined during juvenile development with subsequent adult dispersal (ACD); (b) body condition is determined through maternal effects with subsequent natal dispersal (MCD). The *x*-axis gives dispersal mortality (μ), the *y*-axis environmental variability (σ).

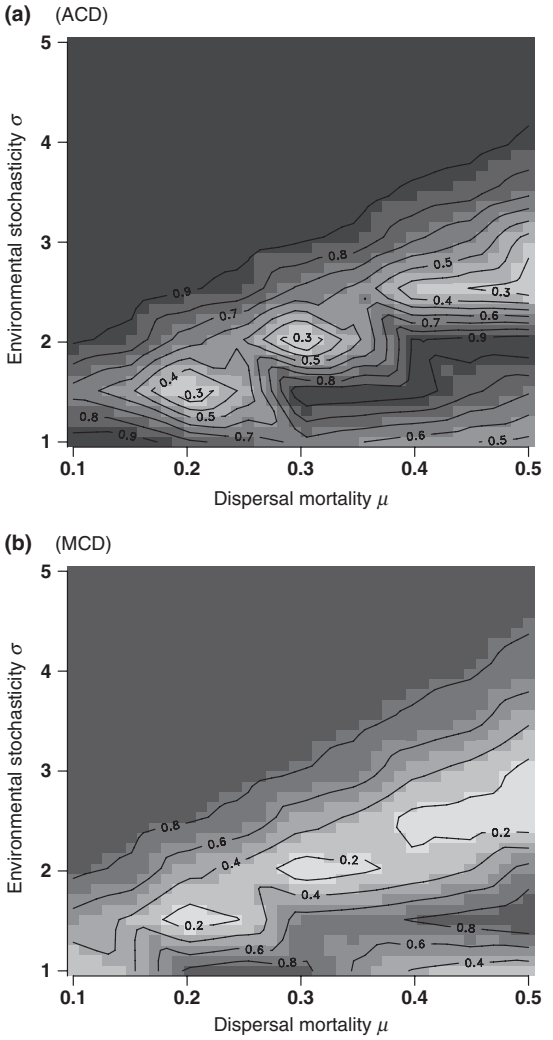

Fig. 4 Average proportion of the variation in dispersal rates determined by the genetically fixed component of the reaction norm (intercept). (a) Body condition is determined during juvenile development with subsequent adult dispersal (ACD); (b) body condition is determined through maternal effects with subsequent natal dispersal (MCD). The *x*-axis gives dispersal mortality (μ), the *y*-axis environmental variability (σ).

values (Fig. 5c and d) point, however, at the existence of a wide array of possible conditional dispersal strategies under conditions of low environmental stochasticity and high dispersal mortality.

Discussion

Because competitively strong individuals experience similar dispersal costs during transfer as conspecifics under lower body conditions, it is not well understood why strong individuals may engage in costly dispersal while they could easily retain their natal habitat. We performed simulations to investigate the evolution of body condition-dependent dispersal in a metapopulation. Body condition is solely related to fecundity and no other competitive interactions after settlement induce fitness benefits. Dispersal rates were mainly determined by the fixed 'genetic' property (intercept allele), but plasticity evolved under a wide range of metapopulation conditions. In general, the dispersal–body condition reaction norm shifts from a negative towards a strong positive relationship when metapopulations increase in environmental stochasticity and decrease in dispersal mortality. Plastic strategies of dispersal in relation to body condition can consequently evolve as a strategy

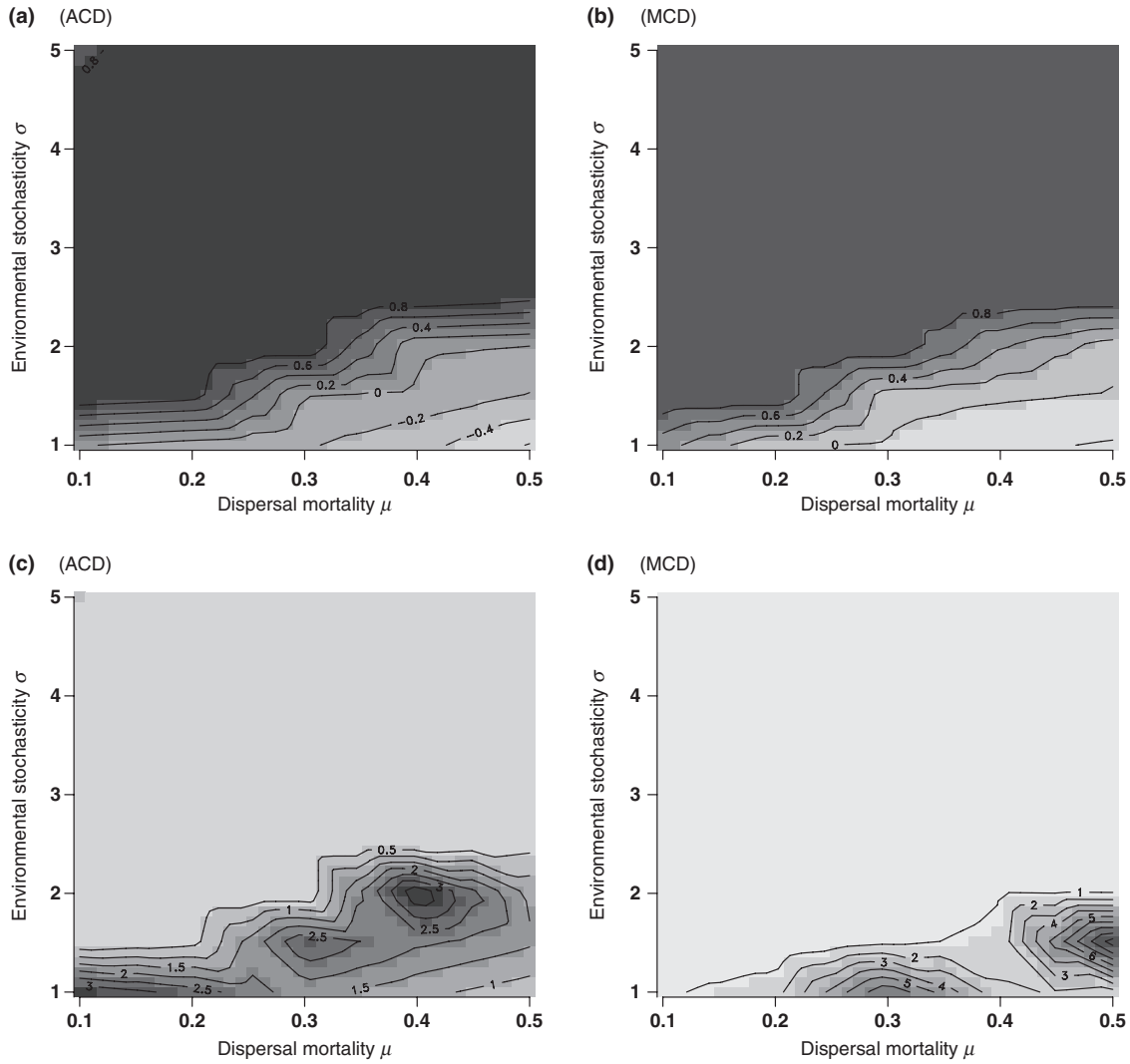

Fig. 5 Average slope values (a,b) and corresponding CV (c,d) of the body condition–dispersal reaction norms. (a,c) Body condition is determined during juvenile development with subsequent adult dispersal (ACD); (b,d) body condition is determined through maternal effects with subsequent natal dispersal (MCD). The *x*-axis gives dispersal mortality (μ), the *y*-axis environmental variability (σ).

with individuals in the better condition predominantly developing higher dispersal rates in connective metapopulations with high environmental stochasticity. When metapopulations are characterized by high dispersal mortality and low environmental stochasticity, individuals in low body condition develop a higher dispersal motivation. The study subsequently provides evidence for the evolution of body condition-dependent plasticity in dispersal.

At the first instance, our simulations confirm the general pattern of selection for increased dispersal in metapopulations with high environmental stochasticity and low dispersal costs. Individuals should disperse as long as their (inclusive) fitness expectations are higher away than in their natal habitat (Metz & Gyllenberg, 2001; Poethke & Hovestadt, 2002). Inversely, when expected dispersal costs are higher than expected (inclusive) fitness benefits (e.g. due to dispersal mortality) dispersal is disfavoured (e.g. Travis & Dytham, 1999; Travis *et al.*, 1999). Dispersal rates therefore increase with increasing environmental stochasticity or external extinction probability (possibilities of relaxed offspring competition in previously unoccupied or sparsely occupied patches), and decline as dispersal costs (dispersal mortality) increase.

Obviously, the first prerequisite for the evolution of body condition-dependent dispersal is the presence of sufficient variation in body condition within the metapopulation. Body condition was similarly regulated by local density as offspring survival, but

also dependent on the individual fecundity due to differences in habitat quality that can be reached within entire metapopulation. This is biologically meaningful when competition between offspring during development affects potential fecundity (e.g. Wise & Wagner, 1992; Wagner & Wise, 1996). Because body condition is relative to the maximal achievable fecundity within the entire metapopulation, low average values of body condition in metapopulations with high environmental stochasticity σ are obviously due to increased maximal Λ in the metapopulation. Evidently, high levels of σ are then responsible for high levels of individual variation in fecundity and related body condition. This variation is then predominantly between local populations. By contrast, when σ is low, variation in body condition is primarily determined by changes in stochastic variation in individual condition ($\sim\Lambda$) and density-dependent regulation, so variation within local populations.

For metapopulation conditions that select for high dispersal (high environmental stochasticity and low dispersal mortality), a positive effect of body condition on dispersal propensity emerged. Under these conditions, benefits for individuals with high potential fecundity are obviously higher because they may gain higher inclusive fitness in new patches which are at a lower density. The main benefit of dispersal in metapopulations with high environmental stochasticity is the increased probability to become a founder of a new population in unoccupied patches or to establish offspring in patches at low densities (e.g. Olivieri *et al.*, 1995; Ronce *et al.*, 2000; Poethke *et al.*, 2003). The most fecund founders (i.e. those in the best condition) will consequently experience the highest fitness benefits.

Dispersal in metapopulations with low environmental stochasticity and low dispersal mortality is driven by drift and reaction norms show high levels of variation with on average slightly positive slopes. Under these circumstances, variation in body condition is inherently low. However, as shown for metapopulations with low σ and high μ, condition-dependent dispersal strategies are theoretically possible, even when CV_c is low. High CV values of slopes indicate that drift mechanisms allow emergence of a wide array of alternative evolutionarily stable strategies.

Low dispersal rates emerge under conditions of low environmental stochasticity and high dispersal mortality, i.e. under conditions that induce kin competition (Poethke & Hovestadt, 2002; Bach *et al.*, 2006; Poethke *et al.*, 2007). Strong kin competition is responsible for high dispersal rates (Gandon, 1999; Gandon & Michalakis, 1999) and significantly increases dispersal rates in quite large populations ($K = 100$), despite high costs of dispersal (Poethke & Hovestadt, 2002; Poethke *et al.*, 2007). Dispersers consequently almost certainly die, but they die with high probability as well when remaining in the patch (no changes in K). Under kin competition, conditional strategies are expected according to theoretical work of Gyllenberg *et al.* (2008). Although the latter found predominantly positive conditional effects on dispersal under kin competition, our finding accords with Gyllenberg's modelled scenario in which negative reaction norms emerge when direct competition is weak because residents in (single-individual) patches are chosen at random. When populations 'need to send out' dispersers that are predestined to die during dispersal, subordinate individuals in worse condition are selected. This leads to rather unexpected increased dispersal rates under conditions of highest μ and lowest σ.

A high potential fecundity (λ) induces strong dispersal depression compared with lower λ values. This accords with the findings of Poethke & Hovestadt (2002), and is due to a decreased variation in population density between patches. Indeed as shown by the latter, high variation in local population density, independent of whether it is induced by environmental stochasticity, external local extinctions or variation in fecundity is the primordial selective force in the evolution of dispersal rates. However, despite the qualitative change in dispersal rate, differences in λ did not alter the evolution of general dispersal–body condition reaction norms. Similarly, between-habitat variation in local density is also increased when individual dispersal decisions are based on the condition of the mother, i.e. in the scenario of natal dispersal. This time lag in dispersal, which is a function of local conditions in the previous generation, enlarges the coefficient of variation in local density.

Timing of dispersal, i.e. natal vs. adult dispersal and the subsequent determination of body condition did not, however, affect the general evolution of reaction norms in metapopulations that select for high dispersal. In metapopulations with high dispersal mortality and low environmental stochasticity, the negative dispersal–body condition reaction norms disappeared under the MCD scenario and many alternative ES strategies emerged by drift (note the high CV on the slope r). This can be heuristically explained by the fact that a higher body condition is not anymore related to personally experienced environmental condition during life, but to those of the mother. Therefore, philopatric individuals in good body condition (having high fecundity) may also experience fitness loss through reduced survival and fecundity of their offspring when the habitat quality of the natal patch is lower compared with those experienced by the mother during the previous generation. It consequently does not pay anymore to send out only subordinate individuals. Contrary to the simulations with organisms that disperse in the adult life phase, no increased dispersal rates under conditions of high μ and low σ emerge under MCD.

In our simulations, body condition depends on habitat quality (λ), population density (s) and resembles fecundity (Λ). It is consequently regulated by metapopulation, patch and population-level processes. Population density has been shown to be an important source of information

and a subsequent strong driver of dispersal propensity, independent of the applied density–dispersal reaction norm (Travis *et al.*, 1999; Metz & Gyllenberg, 2001; Poethke & Hovestadt, 2002). In nature, individuals may perceive information on the local density directly or indirectly through effects on body condition. The emergence of conditional strategies in many metapopulation conditions is – not surprisingly – similar as for the earlier referred density-dependent dispersal strategies. However, although positive density-dependent dispersal always emerges under the implemented metapopulation conditions (Travis *et al.*, 1999), evolved body conditional strategies may be either positive or negative and point out that dispersers should not be a random sample from the population in diverse metapopulations. Moreover, under these body condition-dependent dispersal strategies, local metapopulation extinction rates are always (very close to) zero. Similar models, but with density-dependent emigration strategies, induced significant higher local extinction probabilities (Poethke *et al.*, 2003). The development of body condition-depended dispersal strategies therefore induces evolutionarily rescue mechanisms in spatially structured populations. Species that have evolved these strategies are therefore expected to be less prone to extinction when habitats become fragmented.

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