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Negative density-dependent dispersal emerges from the joint evolution of density- and body condition-dependent dispersal strategies

Celina B. Baines,[1,2,3,*] Justin M. J. Travis,[4] Shannon J. McCauley,[1,2] and Greta Bocedi[4]

[1] *Biology Department, University of Toronto Mississauga, Mississauga L5L 1C6, Canada*

[2] *Ecology and Evolutionary Biology Department, University of Toronto, Toronto M5S 3B2, Canada*

[3] *E-mail: celina.baines@gmail.com*

[4] *School of Biological Sciences, University of Aberdeen, Aberdeen AB24 2TZ, United Kingdom*

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Empirical studies have documented both positive and negative density-dependent dispersal, yet most theoretical models predict positive density dependence as a mechanism to avoid competition. Several hypotheses have been proposed to explain the occurrence of negative density-dependent dispersal, but few of these have been formally modeled. Here, we developed an individual-based model of the evolution of density-dependent dispersal. This model is novel in that it considers the effects of density on dispersal directly, and indirectly through effects on individual condition. Body condition is determined mechanistically, by having juveniles compete for resources in their natal patch. We found that the evolved dispersal strategy was a steep, increasing function of both density and condition. Interestingly, although populations evolved a positive density-dependent dispersal strategy, the simulated metapopulations exhibited negative density-dependent dispersal. This occurred because of the negative relationship between density and body condition: high density sites produced low-condition individuals that lacked the resources required for dispersal. Our model, therefore, generates the novel hypothesis that observed negative density-dependent dispersal can occur when high density limits the ability of organisms to disperse. We suggest that future studies consider how phenotype is linked to the environment when investigating the evolution of dispersal.

KEY WORDS: **Body condition, density-dependent dispersal, dispersal evolution, individual-based model, phenotype-dependent dispersal.**

Theory predicts that individuals should choose to disperse away from low quality sites in favor of reproducing at sites which maximize inclusive fitness, but must balance the potential benefits of dispersing to a new patch against the costs associated with dispersal (Hamilton and May 1977; Southwood 1977; Comins et al. 1980). Multiple aspects of the environment and the phenotype act simultaneously to determine the costs and benefits of dispersal. Therefore, individuals must integrate information from multiple sources to make optimal dispersal decisions (Clobert et al. 2009; Matthysen 2012). This multiple causation of dispersal has been observed in several empirical studies and is expected to be a common feature of dispersal behavior across taxonomic groups (Matthysen 2012). For example, Hanski et al. (1991) found that small shrews (*Sorex araneus*) were more likely to disperse when population densities were low, but when densities were high, dispersal rates were higher and no longer size biased.

*Current Address: Biology Department, McGill University, Stewart Biology Building, 1205 Docteur Penfield Avenue, Montreal, Canada, H3A 1B1.
E-mail: celina.baines@gmail.com

Population density is central to dispersal decision making because it strongly influences fitness (e.g., Clutton-Brock et al. 1987). Previous theoretical models predict that dispersal probability increases with population density above a threshold (Travis et al. 1999; Metz and Gyllenberg 2001; Poethke and Hovestadt 2002; Kun and Scheuring 2006) because of the fitness costs of competition (including kin competition; Ronce 2007). The empirical evidence is largely consistent with this prediction (see Bowler and Benton (2005), Harman et al. (2020) for reviews). However, the opposite effect (negative density-dependent dispersal) has also been observed (Kuussaari et al. 1996; Ims and Andreassen 2005). The cause of negative density dependence is still under debate (Matthysen 2005). Previous authors have hypothesized that it occurs when the relationship between fitness and density is positive as the result of Allee effects (Ims and Andreassen 2005), the benefits of group living (Bowler and Benton 2005; Kim et al. 2009), or the nonindependence of density and habitat quality (Gilbert and Singer 1973; Kuussaari et al. 1996; Roland et al. 2000; Rodrigues and Johnstone 2014).

We propose a novel hypothesis to explain the occurrence of negative density-dependent dispersal that is based on the idea that dispersal is influenced by multiple internal and external factors. Previous models have predicted that individuals disperse away from high-density patches when fitness is a decreasing function of density. However, if high density habitats create individuals with low dispersal ability, those individuals may not be able to use dispersal as a strategy to increase fitness (Benard and McCauley 2008). The ability to disperse and bear the associated costs is influenced by phenotypes including body condition (i.e. the size of an individual's energy reserves; Clobert et al. 2009). For example, high-condition individuals have more energy to invest in energetically costly dispersal activities, including locomotion (Cockbain 1961) and settlement (Bonte et al. 2011) than low-condition individuals. Theoretical models predict that when high-condition individuals incur lower costs or greater benefits from dispersal, positive condition-dependent dispersal evolves (Gyllenberg et al. 2008; Bonte and de la Pena 2009). If we consider body condition and density simultaneously, we expect that all individuals will have positive density-dependent dispersal strategies, but individuals in high body condition will evolve higher dispersal propensity which will manifest as a stronger dispersal response to density (increased dispersal rates out of high-density patches or lower density thresholds for dispersal). Our predictions must also account for the fact that body condition tends to be negatively associated with density (when density is a proxy for competition; e.g. Pettorelli et al. 2002). We hypothesize that individuals in high-density patches will have insufficient energy reserves for dispersal, potentially forcing the metapopulation to exhibit negative density-dependent dispersal.

In this article, we develop an individual-based model to test the hypothesis that the effect of population density on body condition modifies the dispersal response to density. In our model, body condition is a decreasing function of the density of the natal habitat, and populations evolve dispersal strategies that are conditional on both natal patch density and body condition. We then explore the consequences of the evolved dispersal strategy for realized dispersal in relation to density and condition.

The Model

THE LANDSCAPE

We model a sexual species with nonoverlapping generations that exists in a spatially explicit landscape composed of patches arranged in a 10×10 lattice with reflective boundaries. Each patch within the landscape has a finite amount of food resources, which is reset every generation. Resource availability varies spatially among patches. There is temporal, but no spatial, autocorrelation in resource availability. The amount of food present in each patch at generation 0 is randomly selected from a uniform distribution over the interval [0, 100], and then varies across generations, with temporal autocorrelation. Temporal variability was modeled such that the quantity of resources, R, in patch (x,y) at time t is given by:

$$R_{(x,y,t)} = \bar{R}\left(1 + \varepsilon_{(x,y,t)}\right), \tag{1}$$

where $\bar{R}$ is the mean resource availability of patches in the landscape across space and time and ϵ represents environmental noise given by:

$$\varepsilon_{(x,y,t+1)} = \kappa\varepsilon_{(x,y,t)} + \omega_t\sqrt{1-\kappa^2}, \tag{2}$$

where κ is the autocorrelation coefficient and ω is a random normal variable with mean 0 and standard deviation σ (Ruokolainen et al. 2009). For this study, σ was set to 0.8 and κ to 0.2.

The food resources present in each patch are divided into discrete parcels. The size of these parcels within each patch follows a uniform distribution over the interval [0.05, 0.2]. Juveniles born in each patch must compete for these resource parcels. Each juvenile acquires a number of parcels sampled from a Poisson distribution with mean equal to the number of expected parcels divided by the total number of juveniles. Using the number of *expected* parcels generates slight discrepancies: the total amount of resource obtained differs slightly from the resources actually available. However, this method reflects the random process of locating and competing for resources.

At each generation, the resource availability of each patch is calculated using equations 1 and 2. Individuals reproduce, offspring are born, and adults die. Juveniles compete for resources and then mature into adults. All individuals mature

simultaneously. Adults disperse or remain philopatric according to their dispersal phenotype. After dispersal, the new generation starts again with reproduction.

BODY CONDITION

Adult body condition is a sigmoid function of the amount of resources each individual obtains as a juvenile:

$$\rho_i = \frac{1}{1 + e^{-(r_i - \beta)\alpha}}, \quad (3)$$

where ρ_i is the body condition of individual i, r_i is the amount of resources individual i obtains as a juvenile, α and β are constants representing the maximum slope and inflection point of the curve, respectively. Condition, ρ_i, is bounded between 0 and 1. In this study, $\alpha = 8$, and $\beta = 0.5$. Juveniles that obtain no resource parcels die.

DISPERSAL

We assume that the species disperses actively, meaning they move under their own locomotion ability. Individuals make dispersal decisions immediately after they mature. The emigration probability of individual i in patch (x,y), $d_{i,x,y}$, is determined by a logistic function of both natal patch density and body condition:

$$d_{i,x,y} = \frac{D_0}{1 + e^{-\alpha_D[B_{x,y,t} - \beta_D] - \alpha_\rho[\rho_i - \beta_\rho] - \gamma B_{x,y,t}\rho_i}}, \quad (4)$$

where $B_{x,y,t}$ is the density of patch (x,y) at time t (calculated as the population size divided the by total amount of resources in that patch, at the beginning of that generation), and ρ_i is the individual's body condition. The remaining parameters control the dispersal response to density and body condition: α_D and β_D are the slope at the inflection point and the inflection point of the function of density on emigration probability, α_ρ and β_ρ are the slope at the inflection point and the inflection point of the function of body condition on emigration probability, γ represents the interactive effects of condition and density on dispersal, and D_0 is the maximum emigration probability. D_0 is constrained to values between 0 and 1. All other dispersal traits (α_D, β_D, α_ρ, β_ρ, and γ) can take any real value. If an individual disperses, it moves with equal probability to one of the eight patches neighboring its natal patch (nearest-neighbor dispersal). In addition to the full model in which individuals make dispersal decisions using information about both density and condition, we also explored special cases of equation 4 in which individuals made dispersal decisions using information about density only, condition only, or neither (formulae of dispersal functions in Supporting information Appendix S1).

Each parameter of equation 4 (α_D, β_D, α_ρ, β_ρ, γ, and D_0) represents a trait that can evolve. Each trait is controlled by a single diploid locus with continuous alleles. Individuals inherit one allele from each parent at each locus, and offspring phenotype is the sum of the parental alleles (additive genetic model). We assume full recombination among the loci. In each generation, each locus has a probability of mutating (10^{-2}) that is independent of the mutation probability of other loci. The size of each mutation is drawn from a normal distribution with mean, μ_m equal to zero and standard deviation, σ_m equal to 1 (α_ρ, α_D) or 0.1 (β_ρ, β_D, γ, D_0).

Dispersal imposes an energetic cost on active dispersers. We modeled this by reducing the body condition of dispersers by an amount c. This is an absolute cost and is independent of initial condition, although the proportional cost of dispersal decreases with increasing condition. This cost could represent investment into the production of dispersal structures, the cost of moving between patches, and/or the cost of settlement such as the building of a burrow, web, or nest. Individuals that attempt dispersal when the energetic cost, c, is greater than the size of their energy reserves (condition, ρ_i) cannot reproduce in their new patch (i.e., are functionally dead). There is no additional mortality risk imposed on dispersers.

REPRODUCTION

The number of offspring produced by each female i is sampled from a Poisson distribution with a mean μ_i given by:

$$\mu_i = \rho_i F, \quad (5)$$

where F represents the mean fecundity of a female in high condition ($\rho_i = 1$). In this study, F was set to 8. The primary sex ratio was 1:1. Each offspring is sired by a random male in the patch, hence, assuming complete promiscuity. Male mating success is weighted by condition such that the probability of a male i siring each offspring produced in his patch is:

$$m_i = \frac{\rho_i}{\sum_{j=1}^{n} \rho_j}, \quad (6)$$

where $\sum_{j=1}^{n} \rho_j$ is the sum of the body conditions of all males in the patch. The probability of siring an offspring is independent of the probability of siring other offspring. Dispersal therefore reduces reproductive success by lowering female fecundity and the ability of males to compete for mates.

SIMULATION EXPERIMENTS

At the beginning of each replicate simulation, each cell was initialized with a number of individuals equal to the amount of resources (i.e. assuming that on average each individual acquires one unit of resources). Initial individuals were split equally between males and females. Individuals in initial populations were assigned trait values that were randomly selected from normal distributions with mean = 0 and standard deviation = 1 for parameters α_D and α_ρ, mean = 0.5 and standard

Table 1. Description of variables and parameters used in the model. Results show simulations using default values (in bold), unless otherwise stated.

Variable	Description	Value
Landscape		
$R_{(x,y,t)}$	Quantity of resources in patch (x,y) at time t	Equation 1
$\bar{R}$	Mean resource availability of patches in the landscape across space and time	**50**
$\varepsilon_{(x,y,t)}$	Environmental noise in resource availability	Equation 2
κ	Temporal autocorrelation in resource availability	-0.5, -0.2, 0, **0.2**, 0.5
ω	Random normal variable with mean 0 and standard deviation σ	-
σ	Standard deviation of ω	**0.8**
$B_{(x,y,t)}$	Population density of patch (x,y)	$N_{(x,y,t)}/R_{(x,y,t)}$
Body condition		
ρ_i	Body condition of individual i	Equation 3
r_i	Amount of resources obtained by individual i	-
α	Slope at the inflection point of the function of r_i on ρ_i	**8**
β	Inflection point of the function of r_i on ρ_i	**0.5**
Dispersal		
$d_{i,x,y}$	Emigration probability of individual i in patch (x,y)	Equation **4**, S1, S2, S3
α_D	Slope at the inflection point of the function of density on emigration probability	Evolving trait
β_D	Inflection point of the function of density on emigration probability	Evolving trait
α_ρ	Slope at the inflection point of the function of body condition on emigration probability	Evolving trait
β_ρ	Inflection point of the function of body condition on emigration probability	Evolving trait
γ	Interactive effect of condition and density on dispersal	Evolving trait
D_0	Maximum emigration probability	Evolving trait, $0 \leq D_0 \leq 1$
c	Cost of dispersal	0, 0.05, **0.1**, 0.3
μ_m	Mean mutational effect	**0**
σ_m	Standard deviation of mutational effect	**1** (α_ρ, α_D) or **0.1** (β_ρ, β_D, γ, D_0)
Reproduction		
μ_i	Mean fecundity of female i	Equation 5
F	Mean fecundity of female in high condition ($\rho_i = 1$)	4, **8**, 12, 16
m_i	Probability that male i sires any given offspring produced in his patch	Equation 6

deviation 0.1 for β_D, β_ρ, and D0, and mean = 0 and standard deviation = 0.1 for γ. Simulations were run for 100,000 generations to reach evolutionarily stable strategies (Supporting information Fig. S4). For each combination of parameters, we present the final evolved dispersal strategy as a function of body condition and population density, by averaging the values of the evolved dispersal traits across 20 replicates in the final generation of the simulation. All the model variables and parameters are summarized in Table 1. The model was coded in C++ and the code is available in the GitHub repository (https://github.com/GretaBocedi/Body-condition-dependent-dispersal). Model outputs are deposited in the Dryad Digital Repository (https://doi.org/10.5061/dryad.5qfttdz2z).

REALIZED DISPERSAL

Body condition was determined mechanistically in our model by the amount of resources each individual obtained during the juvenile competition phase (see Methods: *Body condition*). In the simulated metapopulations, the result was that condition was a negative function of density (Fig. 1), and some combinations of natal patch density and condition did not occur. Therefore, the realized dispersal exhibited by a metapopulation (i.e., individual emigration status plotted against their body condition or the density they experience) may not reflect the evolved dispersal strategy (i.e., the phenotypes determining dispersal probability for every combination of local density and body condition). Realized dispersal exhibited by the metapopulation, however, is what is typically measured in empirical studies and is therefore important to consider when making theoretical predictions. We visualized realized metapopulation dispersal by plotting in R version 3.4.3 (R Core Team 2017) the predicted fit lines from general linear models with binomial error structures, using emigration status (emigrated or did not emigrate, a binary variable) as the response and

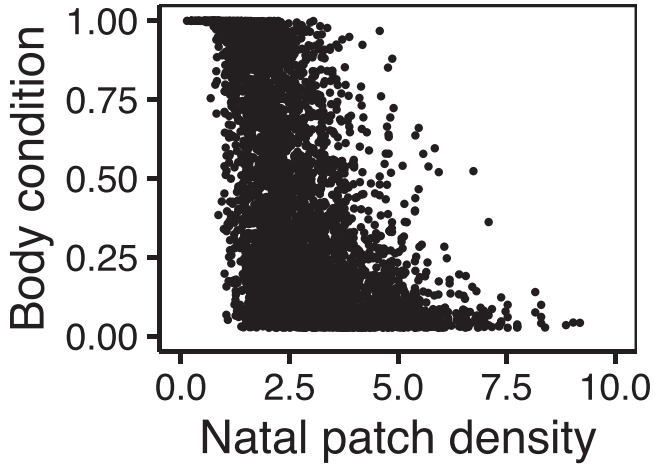

Figure 1. Individual body condition (measured before dispersal) as a function of natal patch density. Natal patch density is the population size divided by the amount of resources in that patch. Body condition is determined mechanistically in the model (see Methods).

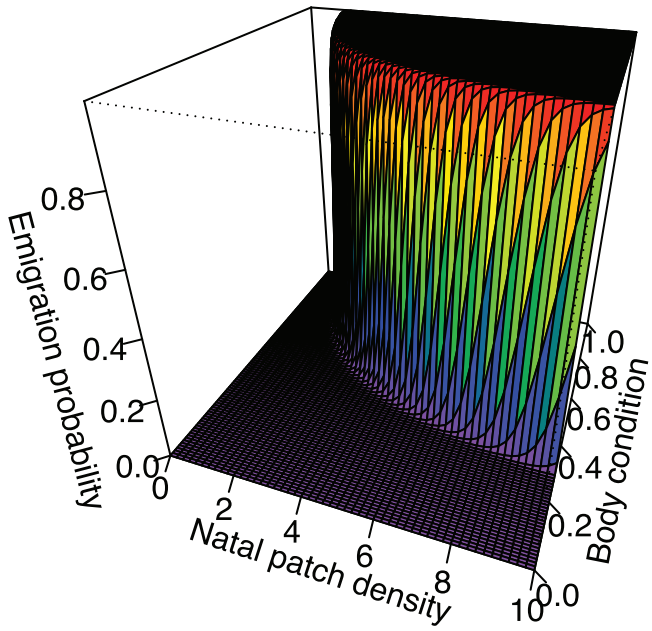

Figure 2. Evolved dispersal strategy in response to both natal patch density and body condition. Color corresponds to probability of emigration; purple = low emigration probability, red = high emigration probability. Parameter values given in Table 1.

body condition or the density of its natal patch as the predictor variable.

Results

EVOLVED CONDITION- AND DENSITY-DEPENDENT DISPERSAL STRATEGY

When individuals may base dispersal decisions on both condition and density and the cost of dispersal, c, is greater than zero, the evolved dispersal strategy is a steep, increasing function of both density and condition (Fig. 2). The effects of density and condition interact (strongly positive γ; Supporting information Fig. S4) such that dispersal probability drops close to zero when condition or density are very low. The condition threshold for dispersal decreases with increasing density (or, equivalently, the density threshold for dispersal decreases with increasing condition). Increasing the energetic cost of dispersal, c, from 0.05 to 0.3 decreased overall dispersal probability but did not alter the shape of the evolved dispersal function (Supporting information Fig. S5B-D). When there is no cost of dispersal, dispersal probability evolves to be a steep, increasing function of density only; emigration is very low in very low-density patches and high in all other patches (Supporting information Fig. S5A).

In the special case in which individuals make dispersal decisions based on condition alone, dispersal probability is a steep, increasing function of body condition when $c > 0$ and independent of condition when $c = 0$ (Supporting information Fig. S1). When individuals use information about density only, dispersal probability is independent of density, except when c is high. At the highest value of c, dispersal is a negative function of density because being in a high-density patch signals to individuals that they have low body condition, and therefore, that dispersal will use a large proportion of energy reserves and potentially be fatal (Supporting information Fig. S2). When individuals do not base dispersal decisions on body condition or density, dispersal probability decreases with increasing dispersal cost (Supporting information Fig. S3).

REALIZED DISPERSAL

When individuals base dispersal decisions on both condition and density and the energetic cost of dispersal is greater than zero, emigration status (i.e., whether an individual dispersed or not) is an increasing function of body condition when measured across the entire metapopulation (Fig. 3D). When plotted separately for discrete ranges of density, emigration status remained an increasing function of body condition within each density bin (Fig. 4A). When we plotted all individuals together, emigration status was a negative function of local density (Fig. 3H). When plotted separately for discrete ranges of body condition, we found that in each body condition bin, emigration status was an increasing function of density (Fig. 4B); however, since high-condition individuals tend to originate from low-density patches and have high emigration rates, and low-condition individuals tend to originate from high-density patches and have low emigration rates, emigration status became a negative function of density when measured across the entire metapopulation (Fig. 4B). Increasing the cost of dispersal from 0.05 to 0.3 decreased emigration probability and increased the dispersal bias toward high-condition

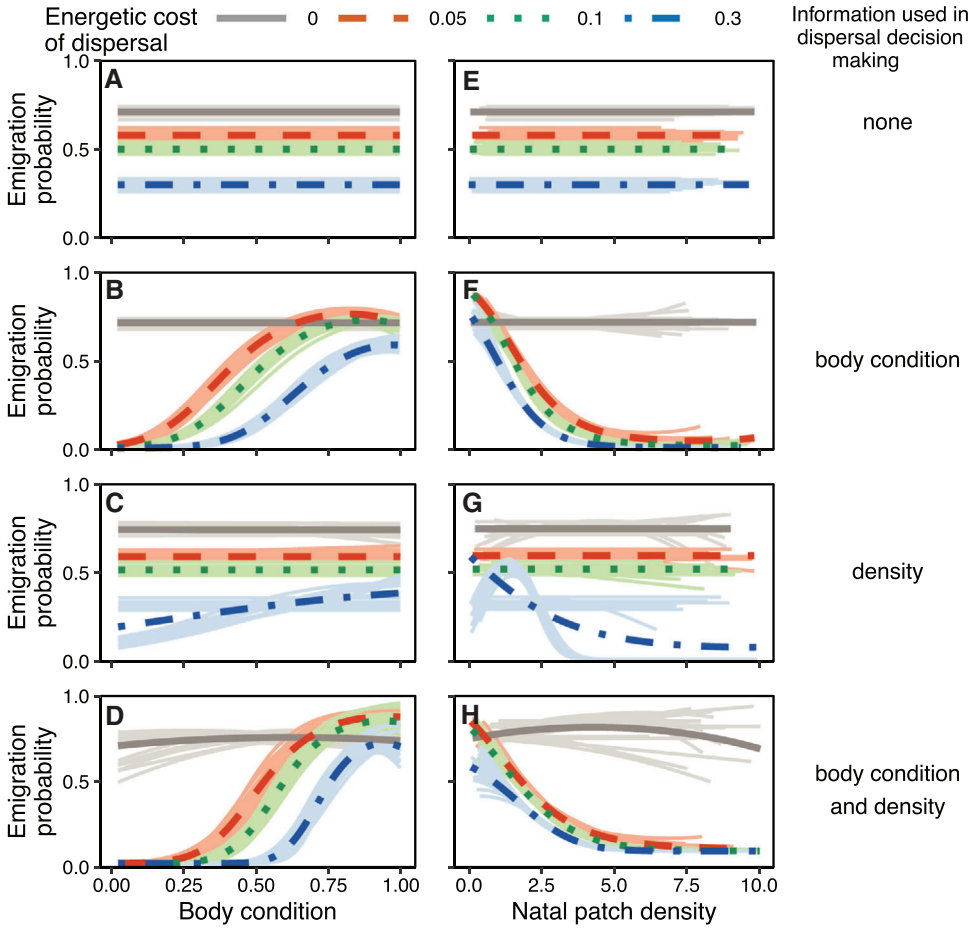

Figure 3. Realized emigration probability measured at the metapopulation level as a function of body condition (A-D) and natal patch density (E-H), for the main and special cases: dispersal is unconditional (A, E), individuals use information about condition only (B, F), density only (C, G), and condition and density (D, H). Increasing the energetic cost of dispersal decreased emigration probability. All panels: Fit lines are probabilities estimated from general linear models with a binomial error structure (formulae given in Supporting information Table S1). Light colored lines show individual replicates; dark lines show the average across replicates. Parameter values given in Table 1.

individuals and low-density patches (Fig. 3D, H). When there is no cost of dispersal, realized dispersal is independent of both body condition and density (Fig. 3D, H).

Realized dispersal was similar when individuals make dispersal decisions based on condition alone; realized dispersal probability was an increasing function of condition (for $c > 0$) (Fig. 3B). Surprisingly, because of the association between body condition and density, realized dispersal is correlated with density even when organisms do not use information about density when making dispersal decisions (Fig. 3F). When individuals make dispersal decisions based on density only, realized dispersal is independent of both density and condition except when the cost of dispersal was high (Fig. 3C, G). Under high costs, realized dispersal exhibits negative density dependence (Fig. 3G). This drives positive condition-dependent realized dispersal (Fig. 3C). When dispersal decisions are unconditional, a constant rate of dispersal evolves and realized dispersal is, as expected, independent of both density and condition (Fig. 3A, E). When there is no cost of dispersal, realized dispersal probability is always independent of body condition and density (Fig. 3).

The distribution of observed patch densities depended on the density dependence of realized dispersal. In cases in which negative density-dependent realized dispersal emerged (when individuals base dispersal decisions on density and body condition or body condition alone), the distribution of patch densities had a higher mean, wider interquartile range, and smaller peak than cases in which realized dispersal was density-independent (Supporting information Fig. S6).

SENSITIVITY ANALYSIS

We checked the sensitivity of our model outcomes (dispersal strategy and realized dispersal) to a range of assumptions. Neither the evolved dispersal strategy nor realized dispersal were qualitatively changed when patch resources ranged from 0 to 25

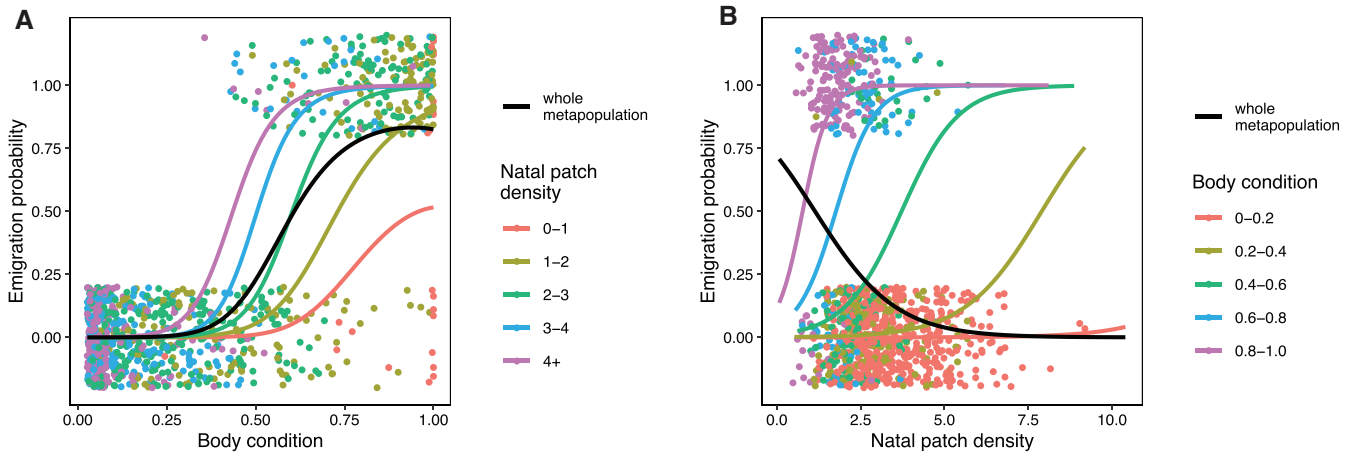

Figure 4. **(A) Realized emigration probability as a function of body condition. Colours represent subset ranges of natal patch density. The black line represents the overall emigration probability measured at the metapopulation level. Emigration probability is an increasing function of body condition in each density bin as well as at the metapopulation level. (B) Realized emigration probability as a function of natal patch density. Colours represent subset ranges of body condition. The black line represents the overall emigration probability measured at the metapopulation level. Emigration probability is an increasing function of density in each body condition bin, but the overall emigration probability measured at the metapopulation level is a negative function of density. Both panels: Each point represents a single individual. Points are 1000 individuals, randomly sampled from all replicates. Note that each individual either dispersed ($y = 1$) or did not disperse ($y = 0$), but points are vertically jittered to improve visibility. Fit lines are probabilities estimated from best-fitting general linear models with a binomial error structure (using all available data): individual emigration status (0/1) ~ (body condition)^2, and individual emigration status (0/1) ~ density. Black lines here are identical to the green lines in Fig. 3D and 3H. Parameters: $c = 0.1$, and Table 1.**

or 0 to 50, instead of 0 to 100. The evolved dispersal strategy and realized dispersal were also independent of resource parcel size, and initial trait values. Modeling body condition as a linear function of resources obtained by individuals produced no qualitative change. Likewise, modeling the cost of dispersal as a reduction in survival rather than a reduction in fecundity produced no qualitative change (Supporting information Appendix S2). Increasing fecundity led to a slightly higher emigration probability but had no qualitative effect on the shape of the evolved dispersal strategy or the relationship between realized dispersal and body condition or density (Supporting information Fig. S7-S9). Changing the direction or magnitude of temporal autocorrelation in resource availability had little effect on either the evolved dispersal strategy or realized dispersal (Supporting information Fig. S10-12). However, complete absence of temporal variability (i.e., constant environment) led to the evolution of a different dispersal strategy in which individuals disperse only at very low density (Supporting information Fig. S10-12). Reducing the mutation rate from 10^{-2} to 10^{-5} altered the dispersal strategy that evolved after 100,000 generations (Supporting information Fig. S13-S14). When mutation rates were low, dispersal probability was a shallow, increasing function of body condition and independent of density; increasing mutation rates increased the dispersal response to condition and density (Supporting information Fig. S13). Mutation rate had no qualitative effect on realized dispersal (Supporting information Fig. S15).

Discussion

We investigated the evolution of dispersal in response to the joint effects of density and body condition. We found that when dispersal evolution responds to density and condition simultaneously, the evolved dispersal strategy is a steep, increasing function of both variables. This result is consistent with the results of previous models which considered evolution of dispersal either in response to density or body condition separately (e.g., Travis et al. 1999; Kun and Scheuring 2006; Gyllenberg et al. 2008; Bocedi et al. 2012). Our results, however, demonstrate positive interactive effects that are apparent in the shape of the dispersal reaction norm to both density and body condition. This interactive effect results from the interplay of dispersal motivation (the willingness to initiate and complete dispersal) and dispersal capacity (the probability of successfully completing dispersal and reproducing in the settlement patch). Individuals in high-density patches have low fitness because of the cost of competition. Therefore, the motivation to disperse (or selection for dispersal) increases with increasing density. Since dispersal capacity depends on the individual having enough energy to deal with the costs of dispersal, capacity increases with condition. Previous authors (Southwood

1977; Benard and McCauley 2008) have argued that organisms should disperse when they have both sufficient motivation and capacity. Our results support this hypothesis from an evolutionary point of view; dispersal probability is close to zero when individuals have low capacity (i.e., low condition), regardless of their motivation to disperse (i.e., density). Moreover, though to a lesser extent, dispersal probability is low when individuals have low motivation (low density), regardless of their capacity (condition).

One goal of theoretical models is to predict empirical relationships between dispersal and environmental or phenotypic characters. Empirical studies typically measure realized dispersal (i.e., individual emigration status plotted against their body condition or the density they experience, e.g., Kuussaari et al. 1996; Barbraud et al. 2003), not dispersal strategies (i.e., the probability of emigration for every combination of density and body condition). It is therefore important to explore realized dispersal in our simulated metapopulations. When we plot realized emigration probability against density and condition, we observe, as expected, that emigration probability is an increasing function of condition. However, the relationship between dispersal and density is counter-intuitive: realized emigration probability is a negative function of density, even though individuals are following a positive density-dependent strategy. This occurs because of the negative association between condition and density emerging in our model. Because of competition for resources, high-density patches produce individuals in low condition who have low dispersal capacity (i.e., they pay a proportionally high cost of dispersal) and, therefore, have low dispersal probability, regardless of density. Conversely, low-density patches produce individuals in high condition, who have relatively high dispersal capacity. High-condition individuals in low-density patches have fairly high propensity to disperse; this may be driven by the inclusive fitness benefits achieved by risk spreading (den Boer 1968) and/or avoidance of kin competition (Gyllenberg et al. 2008). Population sizes range from 1 to 358 when maximum fecundity, $F = 8$ (the default in our model), so there is potential for moderate kin competition, especially when temporal variability exists that causes reductions in resource availability across generations. The result of these combined effects is that a negative relationship is generated between dispersal probability and density when measured at the metapopulation level. Increasing the cost of dispersal increases the dispersal bias toward high-condition individuals and low-density patches. It is important to note that although individuals behave on average adaptively, they may make dispersal decisions that result in them breeding in patches in which their reproductive output is lower than it would be in the median patch in the metapopulation. This results from the constraints imposed on dispersal capacity by low condition, and so disproportionately impacts individuals born into high-density patches.

There is some evidence for this kind of energetic constraint on dispersal strategies in nature. For example, Muraji et al. (1989) found that when the wing dimorphic insect, *Microvelia douglasi*, was reared at high densities, a greater proportion of individuals developed into winged adults. However, when juveniles were food limited, very few individuals developed into winged adults, and the effect of density on wing development disappeared (Muraji et al. 1989). This suggests that the effects of low food availability/high competition on phenotype limits the ability of individuals to disperse from low-quality sites. Unlike previous models which considered the evolution of dispersal in response to a single factor, our model can account for complex dispersal behavior such as that displayed by *M. douglasi*.

Interestingly, a relationship between realized dispersal and density emerged in our model when individuals did not use information about density to make dispersal decisions. When individuals make dispersal decisions based on body condition and not density, populations evolve steep, positive dispersal reaction norms to body condition (Supporting information Appendix S1: special case 1). Because high-condition individuals tend to be in low-density patches and vice versa, the elevated dispersal of high-condition individuals generates negative density-dependent dispersal. In fact, realized dispersal is almost identical in the main case and in the special case in which individuals use information about body condition only; information about density is more or less superfluous. Therefore, observations of relationships between dispersal and any factor cannot be used as evidence that organisms base their dispersal decisions on that factor. Researchers have only recently begun to explore the role of information use in dispersal (Clobert et al. 2009; Bocedi et al. 2012). How organisms gather and use information about multiple, potentially correlated, factors is still an open question. Our results suggest that having information about one factor can allow individuals to make adaptive dispersal decisions with respect to correlated factors, which may reduce the costs associated with gathering information (Clobert et al. 2009; Bocedi et al. 2012).

In the special case in which individuals use information about density only to make dispersal decisions (Supporting information Appendix S1: special case 2), realized dispersal patterns are different from the main case. Dispersal evolves to be mostly independent of density because selection for emigration away from competition is counteracted by selection against emigration of low-condition individuals. Dispersal probability instead evolves to a constant value that is ideal for high-condition individuals who have high reproductive success and are more visible to selection than low-condition individuals. Again, this result is dependent on the negative association between body condition and density. Our conclusions differ from previous models of

dispersal evolution which considered the effects of density independent of phenotype (e.g., Travis et al. 1999; Kun and Scheuring 2006). This highlights the importance of accounting for multiple factors in models of dispersal evolution.

Our model results provide important insights into an open question in the dispersal literature: the observation that a substantial amount of variability exists in density-dependent dispersal (Bowler and Benton 2005), despite the fact that theoretical models generally predict that emigration should be an increasing function of density (Travis et al. 1999; Kun and Scheuring 2006). Previous authors have explained negative density-dependent dispersal by seeking conditions under which negative density dependence evolves through natural selection. Current hypotheses for the causes of negative density-dependent dispersal include the benefits of group living, Allee effects, and positive associations between density and habitat quality (Kuussaari et al. 1996; Bowler and Benton 2005). To our knowledge, only one article has predicted the occurrence of both positive and negative density dependence under different conditions. Using a theoretical model, Rodrigues and Johnstone (2014) demonstrated that in temporally stable environments, selection acts to increase philopatry in high-quality/high-density patches because these sites will continue to be high quality in the future, resulting in negative density-dependent dispersal. In temporally variable environments, selection acts to increase dispersal out of high-quality/high-density patches (positive density-dependent dispersal) because of the risk that habitat quality will deteriorate, resulting in high levels of competition. Our model, in contrast, is the first to predict that ecological constraints may generate negative density dependence in contexts in which selection favours positive density-dependent strategies.

Negative density-dependent dispersal has implications for the distribution of individuals in space. In our study, negative density-dependent dispersal generated a distribution of patch densities with a higher mean, wider interquartile range, and smaller peak than the distribution generated when dispersal was density-independent. This was the result of several effects. First, there was a flow of individuals from low-density to high-density patches, which reduces the frequency of low-density patches and increases the frequency of high-density patches. Second, individuals were constrained from leaving high-density patches, further increasing the frequency of high-density patches and increasing the third quartile of population densities. Finally, while positive density-dependent dispersal may induce population synchrony and reduce spatial variance in some contexts (Bowler and Benton 2005), negative density dependence does not have this effect (Ims and Andreassen 2005). Negative density-dependent dispersal reduces the stability and persistence of metapopulations relative to other forms of density dependence (Bowler and Benton 2005; Harman et al. 2020), meaning the persistence of metapopulations may be influenced by the realized dispersal that emerges as the result of constraints on dispersal ability.

In our simulated metapopulations, dispersal was always an increasing function of condition, both at the level of the entire metapopulation and when broken down into subset ranges of density. Many empirical studies have supported this prediction for actively dispersing organisms (e.g., Meylan et al. 2002; Eraud et al. 2011; Baines et al. 2015). However, there are also several examples of negative and nonmonotonic condition-dispersal relationships (McMahon and Tash 1988; Tarwater and Beissinger 2012; Moore and Whiteman 2016). Our results cannot explain empirical examples of negative condition-dependent dispersal (Clobert et al. 2009). Authors have previously suggested that this occurs when competitive ability is an increasing function of condition, which gives low-condition individuals greater incentive to disperse, especially out of high-density patches (McCauley 2010; Baines et al. 2019). In some cases, this may be mediated by territoriality; low-condition individuals who cannot compete for territories when density is high will be incentivized to disperse (McCauley 2010). We tested this by altering our model such that competitive ability was an increasing function of body condition (Supporting information Appendix S2). The results of this alternative model did not differ from the results of the model presented above: dispersal was an increasing function of condition (Supporting information Appendix S2). This is due to the fact that the proportional costs of dispersal increase with decreasing condition, which imposes a constraint on the dispersal of low-condition individuals, even when dispersal costs are low. We therefore did not find support for the hypothesis that the effects of condition on competitive ability generates negative condition-dependent dispersal. However, altering the assumptions of our model may allow the evolution of negative condition-dependent dispersal in this or other scenarios. For example, if the costs of dispersal increase with body condition (e.g., because moving a large body is more expensive than moving a small body), individuals in low body condition may be more likely to disperse (as predicted by Gyllenberg et al. (2008)). Negative condition-dependent dispersal may also occur when organisms can behaviorally modulate dispersal costs according to their body condition. For example, owls (*Bubo bubo*) in poor condition travel in straighter paths than owls in high condition, which may decrease the costs imposed by dispersal (Delgado et al. 2010). Future studies should explore how differences in how dispersal costs are imposed and the level of behavioral plasticity in dispersal behavior may generate the observed variation in condition-dependent dispersal.

The results of our main model rely on a set of assumptions. There must be temporal variability in resource availability. Dispersal must impose a cost that reduces the fitness (reproduction or survival) of dispersers. And individuals must use information

about body condition to make dispersal decisions. As discussed above, information about density is superfluous. If any of these assumptions are violated, both the evolved dispersal strategy and realized dispersal are altered. We also make a series of assumptions about the determinants and effects of body condition that are necessary to obtain our results. We assume that adult body condition is a function of density experienced during juvenile development. One way to change this assumption is to allow individuals to dominate resource patches, rather than having resources divided into parcels for which all individuals compete. In this case, most patches would produce a small number of high-condition individuals and a varying number of low-condition individuals. If high-density patches produce high-condition individuals, the relationship between realized dispersal and density may flip direction. Changing the assumption that dispersal reduces body condition which then influences fitness (either through reproductive success or survival) would also change our results. Dispersal has varied costs (Bonte et al. 2012), but they may not be mediated by body condition. Alternatively, dispersal may reduce body condition, but condition may not have a strong effect on reproductive success (Wilder et al. 2016). Either of these scenarios would make body condition irrelevant to dispersal decisions; this would likely result in no relationship between body condition and dispersal, and positive density dependence in both the dispersal strategy and realized dispersal.

Our model predicts individual emigration decisions—the binary decision to depart from the natal patch or not. The simplest way to test our predictions would therefore be to use an experimental design that directly observes emigration. However, there are common designs for measuring dispersal that do not observe emigration but instead identify dispersers as those that immigrate into new patches or territories, or those that travel more than a set distance away from their natal site. These designs cannot distinguish between individuals who do not emigrate, and those who emigrate but die during dispersal. The latter may be excluded from the dataset or incorrectly identified as nondispersers (because they never show up in a new site). Assuming low-condition individuals have higher dispersal mortality, designs that observe immigration should find greater disparities in dispersal between low- and high-condition individuals than those that observe emigration. These designs may still be used to test our predictions, but they would require the researcher to estimate condition-dependent dispersal mortality and account for this in the analysis.

Empirical evidence has demonstrated that organisms integrate information about multiple aspects of their environment and their phenotype to make dispersal decisions. Yet, theoretical studies generally model the evolution of dispersal in response to a single factor in isolation from the broader ecological context. This represents a substantial gap in our understanding of dispersal and may explain why the predictions of dispersal models do not closely match empirical observations. In this study, we propose the novel hypothesis that negative density-dependent dispersal emerges as a result of a negative association between body condition and density. We argue that dispersal in natural systems will be best predicted by models that incorporate the interactive effects of environment and phenotype on dispersal. This has implications for understanding metapopulation dynamics including metapopulation persistence and the distribution of individuals in space.

AUTHOR CONTRIBUTIONS

CBB and SJM conceived of the study. All authors designed the study. GB wrote the model code. CBB and GB analyzed the results. CBB drafted the manuscript. JMJT, SJM, and GB critically revised the manuscript. All authors gave final approval for publication.

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DATA ARCHIVING

Model code are deposited in the GitHub Repository: https://github.com/GretaBocedi/Body-condition-dependent-dispersal. Model output are deposited in the Dryad Digital Repository (https://doi.org/10.5061/dryad.5qfttdz2z).

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Supporting Information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Figure S1. Evolved dispersal strategy in simulations in which individuals base dispersal decisions on body condition only, as a function of the energetic cost of dispersal.

Figure S2. Evolved dispersal strategy in simulations in which individuals base dispersal decisions on density only, as a function of the energetic cost of dispersal. The dispersal strategy is determined by calculating the mean of each dispersal trait (αD, βD, $D0$) across individuals in the final generation of the simulation and using equation [S2] to estimate dispersal probability as a function of natal patch density.
Figure S3. Evolved dispersal strategy in simulations in which dispersal decisions are unconditional, as a function of the energetic cost of dispersal.
Figure S4. Evolutionary trajectories of each of the six traits: the slope at the inflection point ($\alpha\rho$) and the inflection point ($\beta\rho$) of the function of body condition on emigration probability, the slope at the inflection point (αD) and the inflection point (βD) of the function of density on emigration probability, the interactive effect of condition and density on dispersal (γ), and the maximum emigration probability ($D0$).
Figure S5. Evolved dispersal strategy in response to both natal patch density and body condition, when the energetic cost of dispersal, $c = 0$ (A), $c = 0.05$ (B), $c = 0.1$ (C), and $c = 0.3$ (D).
Figure S6. Distribution of patch densities in the final generation of the simulations for the main model (body condition & dispersal case, in green) and each special case.
Figure S7. Evolved dispersal strategy when individuals base dispersal decisions on both body condition and density.
Figure S8. Distribution of trait values for each for each of the six dispersal traits in the final generation of the simulations, as a function of the parameter, F, mean fecundity of a female with body condition, $\rho i = 1$.
Figure S9. Realized emigration probability measured at the metapopulation level for different values of F, maximum fecundity, as a function of body condition (A) and natal patch density (B).
Figure S10. Evolved dispersal strategy when individuals base dispersal decisions on both body condition and density.
Figure S11. Distribution of trait values for each of the six dispersal traits in the final generation of the simulations, as a function of the parameter, κ, temporal autocorrelation in resource availability.
Figure S12. Realized emigration probability measured at the metapopulation level as a function of body condition (A, C) and natal patch density (B,D).
Figure S13. Evolved dispersal strategy when individuals base dispersal decisions on both body condition and density. Panels represent simulations with different mutation rates: A) 0.00001, B) 0.0001, C) 0.001, and D) 0.01.
Figure S14. Distribution of trait values for each of the six dispersal traits in the final generation of the simulations, as a function of the mutation rate.
Figure S15. Realized emigration probability measured at the metapopulation level for different values of mutation rate, as a function of body condition (A) and natal patch density (B). Both panels: Each point represents a single individual.
Figure S16. Illustration of how adult survival probability, si, was modeled. Survival probability increased with increasing body condition and decreased with increasing patch density.
Figure S17. Evolved dispersal strategy in simulations in which dispersal responds to body condition only, as a function of the energetic cost of dispersal.
Figure S18. Evolved dispersal strategy in simulations in which dispersal responds to natal patch density only, as a function of the energetic cost of dispersal.
Figure S19. Evolved dispersal strategy in simulations in which dispersal does not respond to body condition or density, as a function of the energetic cost of dispersal.
Figure S20. Evolved dispersal strategy in response to both natal patch density and body condition, when the energetic cost of dispersal, $c = 0$ (A), $c = 0.05$ (B), $c = 0.1$ (C), and $c = 0.3$ (D). These are the results of the modified model, in which body condition influences competitive ability, not reproductive success.
Figure S21. Realized emigration probability measured at the metapopulation level as a function of body condition (A-D) and natal patch density (E-H), for the main and special cases: dispersal is unconditional (A, E), individuals use information about condition only (B, F), density only (C, G) and condition and density (D, H).
Table S1. Top-ranked models based on BIC for each x variable (body condition or natal patch density), dispersal case (main case or one of the three special cases) and dispersal cost.