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Evolution of dispersal in spatially and temporally variable environments: The importance of life cycles

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Spatiotemporal variability of the environment is bound to affect the evolution of dispersal, and yet model predictions strongly differ on this particular effect. Recent studies on the evolution of local adaptation have shown that the life cycle chosen to model the selective effects of spatiotemporal variability of the environment is a critical factor determining evolutionary outcomes. Here, we investigate the effect of the order of events in the life cycle on the evolution of unconditional dispersal in a spatially heterogeneous, temporally varying landscape. Our results show that the occurrence of intermediate singular strategies and disruptive selection are conditioned by the temporal autocorrelation of the environment and by the life cycle. Life cycles with dispersal of adults versus dispersal of juveniles, local versus global density regulation, give radically different evolutionary outcomes that include selection for total philopatry, evolutionary bistability, selection for intermediate stable states, and evolutionary branching points. Our results highlight the importance of accounting for life-cycle specifics when predicting the effects of the environment on evolutionarily selected trait values, such as dispersal, as well as the need to check the robustness of model conclusions against modifications of the life cycle.

KEY WORDS: **Adaptive dynamics, dispersal, disruptive selection, hard selection, metapopulation, soft selection.**

Understanding why organisms disperse is a central question in evolutionary ecology (Clobert et al., 2001). Dispersal is an essential life-history trait that affects evolutionary processes, on both long-time scales (such as speciation or extinction, Leimar and Norberg, 1997; Kawata, 2002) and shorter time scales (e.g., species invasion, local adaptation, or shifts in geographic distribution in response to environmental changes, Pease et al., 1989; Burton et al., 2010; Boeye et al., 2013; Henry et al., 2013; Kubisch et al., 2014). Dispersal traits are known to be heritable in a wide range of organisms (Pasinelli et al., 2004; Saastamoinen, 2008; Charmantier et al., 2011) and to display some variability in the wild (Imbert et al., 1997; Hanski and Saccheri, 2006; Roff and Fairbairn, 2007): the traits can hence evolve in response to selection.

Theoretical studies have identified different mechanisms affecting the evolution of dispersal (reviewed in Bowler and Benton, 2005; Ronce, 2007; Duputié and Massol, 2013). Increased dispersal rates have been shown to evolve in order to avoid competition with related individuals (avoidance of kin competition, Hamilton and May, 1977; Frank, 1986), or to avoid mating with related individuals, when this has detrimental effects on fitness (inbreeding avoidance, Bengtsson, 1978; Perrin and Mazalov, 1999; Roze and Rousset, 2005); the latter mechanism will not be considered in this study, which focuses on clonally reproducing individuals. Dispersal can also evolve when local conditions vary spatially and temporally, whether this variation is due to stochastic population dynamics (Doebeli and Ruxton, 1997), or changes in patch quality (McPeek and Holt, 1992; Johst

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and Brandl, 1997; Mathias et al., 2001; Parvinen, 2002); the latter case is the focus of our article. Not all types of variation, however, favor the evolution of dispersal. Positive temporal autocorrelation in patch quality (Travis, 2001), or, more extremely, purely spatial variation in patch quality (i.e., the absence of temporal variability), has been shown to prevent the evolution of dispersal (Balkau and Feldman, 1973; Asmussen, 1983; Hastings, 1983; Cohen and Levin, 1991; Greenwood-Lee and Taylor, 2001) because, on average, dispersing individuals tend to emigrate out of high-quality patches toward low-quality patches (Holt, 1985). Conversely, when habitat quality fluctuates rapidly, higher dispersal rates are favored because they tend to help genotypes track favorable patches in the metapopulation and help recolonize extinct or nearly extinct patches (van Valen, 1971; Roff, 1975; Comins et al., 1980).

Most studies on the evolution of dispersal use a particular life cycle, but do not test the consequences of their choice. And yet, the order of events (reproduction, selection, density regulation, dispersal) in the life cycle has been shown to be of crucial importance in studies of local adaptation, because it influences the regime of selection (soft selection vs. hard selection, Levene, 1953; Dempster, 1955; Christiansen, 1975; Wallace, 1975; Ravigné et al., 2004; Débarre and Gandon, 2011; Massol, 2013), which, in turn, determines to what extent local adaptation polymorphisms can evolve or be maintained.

Similarly, the evolution of habitat selection is known to be affected by the selective regime or, equivalently, by the life cycle (Hedrick, 1990; de Meeus et al., 1993; Ravigné et al., 2009). In a study based on computer simulations, Johst and Brandl (1997) also highlighted the importance of the order of events in the life cycle for the evolution of dispersal strategies when the decision to disperse depends on local conditions ("conditional" dispersal). However, simple analytical predictions about the role of life cycle specifics on the evolution of unconditional dispersal (when the decision to disperse is made independently of the local conditions) in response to environmental variability are still lacking. In this article, using analytical methods, we show that unconditional dispersal strategies that evolve in response to environmental variability are also affected by the order of events in the life cycle, and we thereby clarify the role played by the life cycle in constraining or favoring the evolution of dispersal. Our focus is on the effect of spatiotemporal variability of the environment on the evolution of dispersal, and our analysis is done under the assumption of very large patch sizes. We also later run numerical simulations with small patch sizes to explore how the addition of kin competition affects our predictions.

Models

MODEL STRUCTURE

A metapopulation

We consider the evolution of an asexual species in a metapopulation consisting of an infinite number of patches, each containing a very large number of individuals (we thus neglect kin competition effects in our analysis; these effects are then introduced in numerical simulations, see Supporting Information). The temporal dynamics of the metapopulation follow a Wright–Fisher model: time is discrete, generations are synchronous, total density remains constant due to high fecundity and demographic regulation. In addition, generations are nonoverlapping. As typically done in island models (Wright, 1931), dispersal among patches is random: there is no habitat selection and all other patches are one dispersal step away. In the following, we will keep the notation and modeling framework introduced in Massol (2013) to study the evolution of traits in spatially and temporally variable metapopulations.

In the metapopulation, there are two types of patches, noted 1 and 2. The type of a patch indicates its quality, which affects the fecundity of its inhabitants. The proportion of type-1 patches in the whole metapopulation is denoted by ρ, and is constant across generations; however, the type of a given patch can change from one generation to the next, and φ denotes the temporal autocorrelation in patch state (notation is summarized in Table 1).

Within any generation, four events take place in a given order that defines the life cycle of the model. Each of these events affects the total densities of mutant individuals in the two types of patches and can be described by a matrix. If we denote by the column vectors $\mathbf{Y_B} = (y_{1B}\ y_{2B})^{\mathrm{T}}$ and $\mathbf{Y_A} = (y_{1A}\ y_{2A})^{\mathrm{T}}$ the total densities of mutants in habitats 1 and 2, before ($\mathbf{Y_B}$) or after ($\mathbf{Y_A}$) an event M, then the matrix $\mathbf{M}$ describing the event is such that $\mathbf{Y_A} = \mathbf{M} \cdot \mathbf{Y_B}$.

The different events in the life cycle

Reproduction, noted as event F (or matrix $\mathbf{F}$ when needed), corresponds to the asexual production of offspring by the adults in each patch, followed by the death of the parents. Individual fecundity only depends on the type of patch the parents live in, but not on the composition or density of the population within the patch. Each individual produces a number f_i of offspring in a patch of type i, so that the matrix $\mathbf{F}$ is given by

$$\mathbf{F} = \begin{pmatrix} f_1 & 0 \\ 0 & f_2 \end{pmatrix}. \quad (1)$$

Environmental change, noted as event E (or matrix $\mathbf{E}$), is assumed to be a stochastic process and occurs independently for each patch. It keeps the expected proportion of patches of type 1

Table 1. Model notation.

Notation	Meaning	Value
d	Dispersal rate	$[0; 1]$
c	Direct cost of dispersal	$[0; 1]$
f_i	Fecundity in type-i patches	$[0; +\infty[$
φ	Temporal autocorrelation in patch state	$[-\min(\frac{\rho}{1-\rho}, \frac{1-\rho}{\rho}); 1]$
β	Squared standardized geometric average of fecundity over two consecutive different patch states $\beta = f_1 f_2/[\rho f_1 + (1-\rho) f_2]^2$	$[0; +\infty[$
γ	Squared coefficient of variation of fecundity among patches $\gamma = \rho(1-\rho)(f_1 - f_2)^2/[\rho f_1 + (1-\rho) f_2]^2$	$[0; +\infty[$

equal to ρ while allowing for autocorrelation in patch state before and after environmental change (this autocorrelation is noted φ). With these assumptions, the matrix $\mathbf{E}$ is given by

$$\mathbf{E} = \begin{pmatrix} 1-(1-\rho)(1-\varphi) & \rho(1-\varphi) \\ (1-\rho)(1-\varphi) & 1-\rho(1-\varphi) \end{pmatrix}. \quad (2)$$

Dispersal, noted as event D (or matrix $\mathbf{D}'$), corresponds to the movement of individuals from their natal patch to their breeding patch. The trait under selection is the emigration probability, which is unconditional (i.e., only depends on the individual's type, but not on the local conditions). This trait, noted d, can take any value between 0 and 1. The prime $'$ indicates that we consider the movement of mutant individuals with a dispersal trait d'. We assume that dispersal is costly: a fraction c of dispersers dies during their journey. In our model, there is no habitat selection: the probability to land in a patch of type 1 is equal to the proportion of type-1 patches (ρ) in the metapopulation. Given these assumptions, the matrix $\mathbf{D}'$ is given by

$$\mathbf{D}' = \begin{pmatrix} (1-d') + d'(1-c)\rho & d'(1-c)\rho \\ d'(1-c)(1-\rho) & (1-d') + d'(1-c)(1-\rho) \end{pmatrix}. \quad (3)$$

Finally, density regulation, noted as event R (or matrix $\mathbf{R}$), is assumed to occur either independently in each patch (local regulation) or globally, at the metapopulation scale (global regulation).

With local regulation, the total density of individuals in each patch is reduced to the patch carrying capacity (the same for all patches), which is very large. This step does not change the local frequency of mutants, but changes their frequency in the metapopulation when preregulation densities differ among patches. To assess whether individuals with a mutant dispersal trait can invade, we follow the initial dynamics of mutant densities, that is, when the mutants are still rare. As a result, for these initial dynamics, regulation consists in dividing the number of mutants obtained after all other life cycle events in a given patch by the number of residents obtained after the same steps. The resulting regulation matrix $\mathbf{R}$ is given by

$$\mathbf{R} = \begin{pmatrix} r_1 & 0 \\ 0 & r_2 \end{pmatrix}, \quad (4)$$

where r_1 and r_2 depend on the precise life cycle (formulae for the r_i terms are presented in eq. A.2 in Supporting Information Material).

With global regulation, the total density of individuals in the metapopulation is kept constant from one generation to the next. This step does not change the frequency of mutants at the metapopulation scale (and hence is equivalent to not regulating at all). In practice, global regulation means dividing the mutant's total fecundity output over the whole metapopulation by the residents or, equivalently, using a regulation matrix $\mathbf{R}$ of the form $\mathbf{R} = r\mathbf{I}$, where $\mathbf{I}$ is the identity matrix and r is the inverse of the resident total fecundity output (see Supporting Information).

Different possible life cycles

Each event happens only once under the different life cycles. All individuals follow the same life cycle, that is, the same series of events between birth and death. Without loss of generality, when enumerating life cycles, we set regulation R as the last event. With local density regulation, six cycles can be obtained by permuting the remaining three events. However, some of these cycles are equivalent (Massol, 2013). First, because density regulation does not depend on the type of patch per se, but only on the local density of individuals, the events E and R commute, leaving only four different cycles. Second, because dispersal is unconditional and there is no habitat selection, the events D and E commute, leaving only two different life cycles (using a naming convention consistent with Débarre and Gandon, 2011): juvenile dispersal life cycles (E, F, D, R) (as in models by Bull et al., 1987; Taylor, 1992; Sasaki and de Jong, 1999; Ravigné et al., 2004) and adult dispersal life cycles (E, D, F, R) (as in Levene, 1953; Christiansen, 1975). These life cycles are illustrated in Figure 1(A, B).

Global regulation is equivalent to an absence of regulation in terms of mutant dynamics because neither of these options

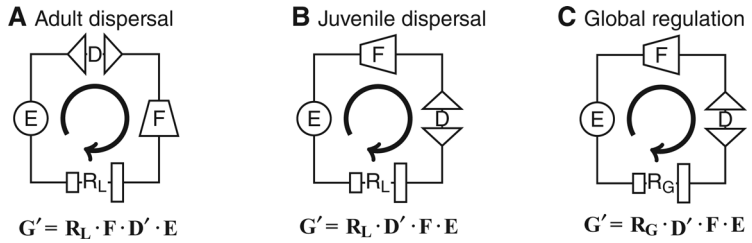

Figure 1. Graphical summary and expression of the next-generation matrices G′ of the three different life cycles. R_L represents local regulation; R_G, global regulation.

changes the frequency of mutants in the metapopulation. Thus, regulation commutes with all other events and the two different life cycles, (E, F, D, R) and (E, D, F, R), are equivalent when regulation is global. This life cycle will be referred to as global regulation life cycle (Dempster, 1955; Fig. 1C). When computing mutant fitness under this life cycle, one can either simply forget about event R, that is, replace matrix $\mathbf{R}$ by the identity matrix, or use the global regulation proposed above ($\mathbf{R} = r\mathbf{I}$ with r equal to the inverse of the resident fitness). Our aim is to compare the evolution of dispersal in these different life cycles, summarized in Figure 1.

FORMALIZATION OF THE FITNESS FUNCTION

The invasion fitness $W(d', d)$ of a rare mutant (with dispersal d') in a metapopulation entirely occupied by a single resident type (with dispersal d) is the dominant eigenvalue of the next-generation matrix $\mathbf{G}'$ (Diekmann et al., 1990), evaluated assuming that the mutants are still rare (Metz et al., 1992). This measure describes the invasion potential of individuals with a mutated dispersal trait. For each life cycle, the next-generation matrix $\mathbf{G}'$ is determined using a simple formalism based on the use of event matrices (Massol, 2013). If the vector $\mathbf{Y} = (y_1, y_2)^T$ represents the density of mutants in patches of type 1 and 2, respectively, after density regulation (if any), initial mutant dynamics can generally be represented by the following equation:

$$\mathbf{Y}_{t+1} = \mathbf{R}.\mathbf{C}'.\mathbf{Y}_t = \mathbf{G}'.\mathbf{Y}_t, \tag{5}$$

where $\mathbf{Y}_t$ is vector $\mathbf{Y}$ at generation t (after the tth regulation event), $\mathbf{C}'$ is the cycle matrix corresponding to mutant demographics until regulation (hence the prime), and $\mathbf{R}$ is the regulation matrix corresponding to the matching resident demographics. The next-generation matrix for the mutant densities is $\mathbf{G}' = \mathbf{R}.\mathbf{C}'$, and is detailed in Figure 1 for the different life cycles.

Since the next-generation matrices $\mathbf{G}'$ in our models are 2×2 matrices, we can write the invasion fitness simply as

$$W(d', d) = \frac{\mathrm{Tr}\left(\mathbf{G}'\right) + \sqrt{\mathrm{Tr}(\mathbf{G}')^2 - 4\det\left(\mathbf{G}'\right)}}{2}, \tag{6}$$

where Tr denotes the trace and det the determinant of the next-generation matrix.

In the following, we reduce the number of parameters in the model by using two compound parameters. We note that the average fecundity in the metapopulation is $\rho f_1 + (1-\rho) f_2$ and its variance is $\rho(1-\rho)(f_1 - f_2)^2$. We denote by β the squared standardized geometric average of fecundity over two consecutive different patch states

$$\beta = \frac{f_1\, f_2}{\left[\rho f_1 + (1-\rho) f_2\right]^2}, \tag{7}$$

and γ the squared coefficient of variation of fecundity among patches

$$\gamma = \frac{\rho(1-\rho)\,(f_1 - f_2)^2}{\left[\rho f_1 + (1-\rho) f_2\right]^2}. \tag{8}$$

The model is now parameterized by four parameters only: (1) the direct cost of dispersal c, (2) the temporal autocorrelation in patch quality φ, (3) a measure of the difference in quality among different patches β, and (4) a measure of the variance in patch quality γ. It is important to note that parameters β and γ do not account for the same measures of variability of patch quality. γ is a coefficient of variation: it is the spatial variance in fecundity in the environment (numerator), scaled by the square (arithmetic) average fecundity (denominator). There is variance in fecundity because there are two habitat types (in proportions ρ and $1-\rho$), and because fecundity differs among these habitats (f_1 and f_2). The parameter β deals only with this second component of environmental heterogeneity. β can also be interpreted as a comparison, over two generations (in the absence of dispersal and regulation), of the absolute fecundity of a lineage that would experience a change in patch quality (numerator), to the fecundity of a lineage experiencing the average environment (denominator). In the context of dispersal evolution, it is both intuitive and helpful to introduce parameters summarizing geometric and arithmetic averages of local fitness (Metz et al., 1983).

EVOLUTIONARY OUTCOMES

Analytical methods

We use the adaptive dynamics toolbox (Hofbauer and Sigmund, 1990; Dieckmann and Law, 1996; Geritz et al., 1998) to study

the evolutionary dynamics of the dispersal trait. The framework assumes that mutations are rare so that the number of coexisting types is small, and that mutants are phenotypically close to the residents. The evolutionary dynamics are initially determined by the selection gradient (first derivative of the fitness function), until either a singular strategy (trait value at which the selection gradient vanishes) or a boundary of the trait space ($d = 0$ or $d = 1$) is reached. A given singular strategy can only be reached by gradual evolution if it is convergence stable (CS), which occurs when the derivative of the selection gradient is negative at this point. This strategy is then the final evolutionary outcome only if it is also an evolutionarily stable strategy (ESS). A CS strategy is evolutionarily stable when the second derivative of the fitness function with respect to the mutant trait is negative; otherwise, that strategy is a branching point, and evolutionary diversification ensues. A Mathematica notebook detailing the analytical computations given in the text and in appendices is available on figshare at http://dx.doi.org/10.6084/m9.figshare.1195906.

Numerical simulations

We also run numerical simulations of the model, which relax the assumption of rare mutations, to confirm our analytical results. The simulations are coded in R, and follow the different life cycles used in the analysis; the codes are available on figshare at http://dx.doi.org/10.6084/m9.figshare.1180191.

Phenotype space is divided into 51 possible dispersal phenotype classes (d_i, i in $\{1, \ldots, 51\}$), taking values between 0 and 1. With a probability $\mu = 0.01$, an individual offspring is a mutant; its parent's phenotype being d_i, the mutant's phenotype is d_{i+1} or d_{i-1} with equal probabilities (except at the boundaries 0 and 1, which are absorbing). At a given time step, multiple dispersal phenotypes potentially coexist in the population.

Simulating infinite local population sizes. The population is subdivided in $n = 25$ patches, and we follow the proportions of individuals in each phenotype class in each patch. To avoid the persistence of phenotype classes at extremely low frequencies, after the regulation event, all classes that are at a frequency below $1/N$ are set equal to 0—where N, equal to 10^5 in the simulations, represents the carrying capacity of each patch—and the remaining class frequencies are then renormalized. The order and description of the different events of the three life cycles are otherwise identical to the analytical part.

Simulating finite, small local population sizes. Our analysis assumes that local population sizes are very large, but we checked the robustness of our results when local population sizes are small. In this other set of simulations, patch sizes were set equal to $N = 10$. In the life cycles with local density regulation, the regulation event consists in randomly sampling N individuals among all the offspring present in each patch. In the life cycle with global regulation, the total size of the population is Nn, n being the number of patches in the metapopulation, but local population sizes may vary. We first draw the postregulation local population sizes (which depend on the preregulation relative abundances), and then sample individuals locally. The results of these simulations are described in the Results section, and illustrated in Figures S1 and S2.

Results

ADULT DISPERSAL LIFE CYCLE

With large patch sizes

The adult dispersal life cycle is an example of soft selection: the output of each patch is independent of its genetic composition. In this case, the invasion fitness of mutants with a dispersal trait d' appearing in a population with a dispersal trait d is given by:

$$W_L(d', d) = \frac{1 - cd'}{1 - cd}. \tag{9}$$

When there is no direct cost of dispersal ($c = 0$), equation (9) reduces to $W_L(d', d) = 1$: there is no selection for or against modified values of the dispersal trait. This is because population regulation occurs right after reproduction, thereby removing all differences in densities between patches, and with this any advantage in having a modified emigration rate. In other words, the adult dispersal life cycle behaves as if there were no differences among patches from the viewpoint of a potential migrant. Because of recurrent mutation, all dispersal trait values will end up being present in the population, which is why the simulation results in Figure 2(A) appear all white (actually, in a very light shade of gray), meaning that all trait values are present.

When dispersal is costly ($c > 0$), mutants with a lower dispersal trait are always selected for: the dispersal trait evolves toward its lowest possible value, $d = 0$, regardless of the other parameters (Massol, 2013) and the only effect at play is the direct cost of dispersal: as confirmed by the simulations, evolution leads to total philopatry ($d = 0$; Fig. 2D).

With small patch sizes

When patch sizes are small, dispersal is a way to escape kin competition. In the absence of cost, high values of the dispersal trait evolve (Fig. S1A).

When dispersal is costly, evolution leads to an intermediate value of d, where the benefits (avoiding kin competition) and costs (the direct cost of dispersal) of dispersal compensate each other (Fig. S1D), in line with classic models of this situation (using results from Frank, 1986, the predicted dispersal is approximately 0.39, as shown in Fig. S1D).

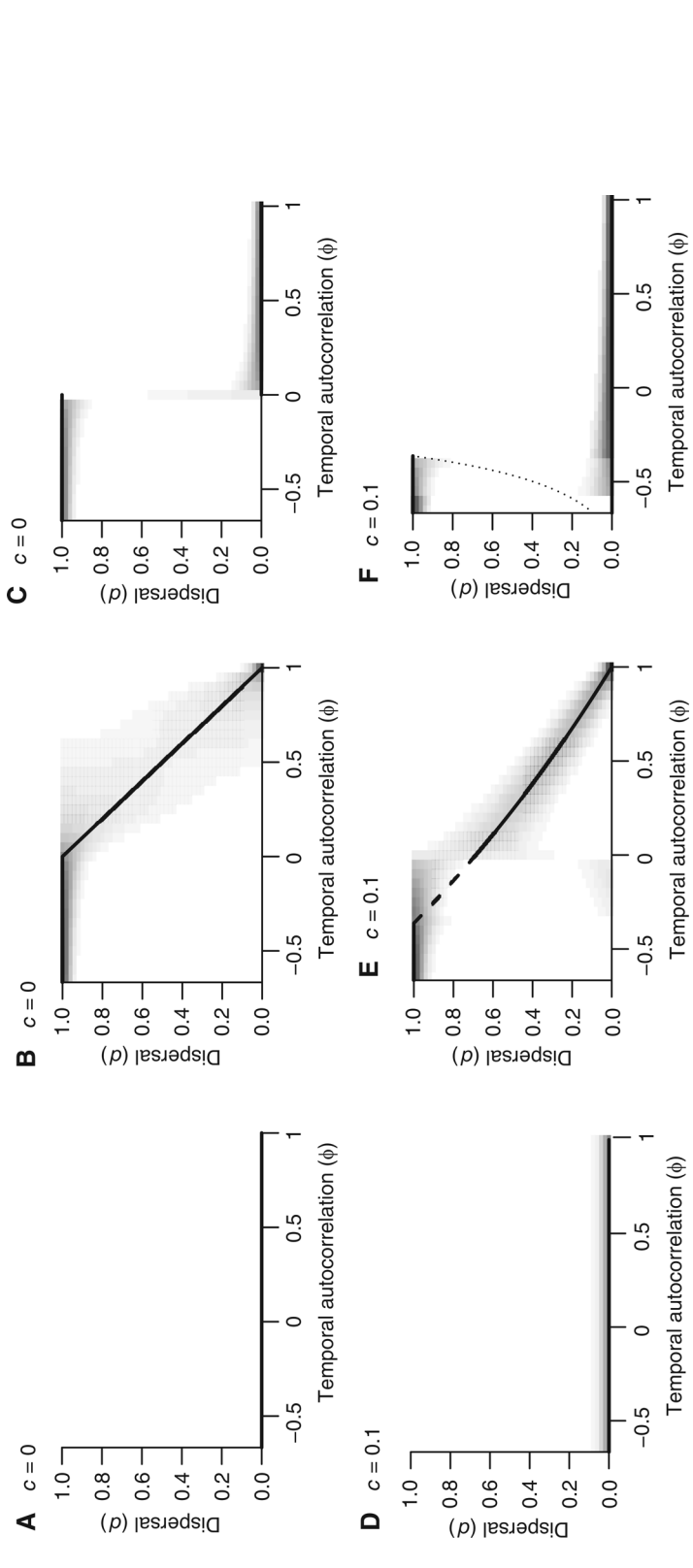

Figure 2. Evolution of dispersal under the three different life cycles (columns), for different values of the direct cost of dispersal c (first line, no cost; second line $c = 0.1$). Variation in singular dispersal strategy (ordinates, d) with temporal autocorrelation in patch state (abscissas, φ). Parameter values: $\rho = 0.4$, $c = 0.1$, $f_1 = 1$, $f_2 = 4$. Solid lines indicate ESS, dashed lines represent branching points, and dotted gray lines represent evolutionary repellers. Note that, because $\rho = 0.4$, $\varphi > -0.66$ (hence, the horizontal axis starts at -0.66 and not -1). Background gray levels indicate the results of simulations in very large populations with no stochasticity in recruitment.

Admittedly, this life cycle is of limited interest when habitat qualities vary because local density regulation, which happens right after offspring production, removes all productivity differences among patches. We now turn to the other life cycles, in which the outputs of the patches vary.

JUVENILE DISPERSAL LIFE CYCLE

With large patch sizes

Under the juvenile dispersal life cycle, the expression for fitness is cumbersome, but the selection gradient, indicating the direction of selection, reads as follows:

$$\frac{\partial W_R}{\partial d'}(d,d) = (1-c)\,d\,\frac{\left(\frac{1-\varphi-d}{d}\right)\gamma-\left(\frac{c}{1-c}\right)\left(\frac{1-d}{d}\right)(1-\varphi)\beta-c}{(1-c)d(1-d)\gamma+(1-c\,d)\,[(1-c)d+(1-d)(1-\varphi)\beta]}. \quad (10)$$

The denominator in equation (10) is always positive, so the direction of selection is given by the sign of the numerator. The numerator has three different components.

The first component (first term in the numerator in 10) is the balance between benefits and costs of dispersing when habitat is of heterogeneous quality, $(\frac{1-\varphi-d}{d})\gamma$. This first term is the only one left when there is no cost of dispersal (when $c = 0$). When $d < 1-\varphi$ (a condition that is always met when the environment is temporally negatively autocorrelated), this effect of habitat heterogeneity is beneficial to the evolution of dispersal. However, when $d > 1-\varphi$ (when the environment is positively autocorrelated and dispersal is high), this component becomes a net cost of dispersal. In both cases, the intensity of this benefit or cost is proportional to γ: the magnitude of this first term is greater when there is more spatial heterogeneity. It is also worth pointing out that this transition from benefit to cost of dispersal as dispersal increases (or equivalently, the existence of intermediate CS strategies) is a hallmark of frequency-dependent selection in such a simple model (see, e.g., Result 1 in Heino et al., 1997, where it is shown that intermediate CS strategies necessitate environmental feedback that is more than one-dimensional, hence frequency-dependence).

The second component, always negative, is the indirect cost of dispersal due to temporal habitat fluctuations, $-(\frac{c}{1-c})(\frac{1-d}{d})(1-\varphi)\beta$. It is proportional to β, the measure of environmental heterogeneity involving geometric mean fitness (see eq. 7). This indirect cost increases with c, the direct cost of dispersal, and with the negative autocorrelation of environmental fluctuation $(-\varphi)$. Finally, the third component is the direct cost of dispersal, $-c$, which is obviously detrimental to the evolution of dispersal.

Total dispersal ($d = 1$) is selected for when $\varphi < \varphi_1$ (Fig. 2E and Figs. S3–S5 and S9–S11); this is also the case when patch sizes are small (Figs. S1 and S2). The threshold φ_1 is given by:

$$\varphi_1 = -\frac{c}{\gamma}. \quad (11)$$

The quantity φ_1 is always negative or null (the latter when $c = 0$). This means that total dispersal is favored when changes in habitat type become more likely. The threshold φ_1 increases (i.e., is easier to fulfill) when the spatial variance in fecundity in the environment, γ, increases (Fig. S9).

Conversely, total philopatry ($d = 0$) is selected for only when the cost of dispersal is higher than a threshold value c_R, which is the value of c that exactly balances costs and benefits of dispersal at $d = 0$ in equation (10) (Fig. 3 and Fig. S1 – S3 and S7 – S9):

$$c_R = \frac{\gamma}{\beta+\gamma}. \quad (12)$$

This threshold value of the cost does not depend on φ, the temporal autocorrelation in patch states (Fig. S9). It tends toward zero as the environment becomes more uniform ($\gamma \to 0$, Fig. S2), and c_R tends toward one as the environment becomes more heterogeneous ($\gamma \to \infty$, Fig. S2) while keeping β finite (i.e., through increasing $\rho(1-\rho)$ rather than changing f_2/f_1). When there is no variance in patch quality, that is, $\gamma = 0$, then $\beta = 1$ and we recover selection for total philopatry as in adult dispersal life cycles. Note that total dispersal and total philopatry cannot be selected at the same time because $\varphi > -1/(\beta+\gamma)$ (see Supporting Information for proof).

When $\varphi > \varphi_1$ and $c < c_R$, singular dispersal strategies take intermediate values (i.e., neither 0 nor 1) equal to:

$$d_R^* = \frac{(1-\varphi)\,[(1-c)\gamma - c\beta]}{(1-c)(c+\gamma)-c(1-\varphi)\beta}. \quad (13)$$

Without a direct cost of dispersal ($c = 0$), this reduces to $d_R^* = 1-\varphi$, meaning that increased autocorrelation in patch state selects for more philopatry. These intermediate dispersal strategies are always CS (see Supporting Information for a proof). It can also be shown that singular strategies d^* are ESS if and only if $\varphi > 0$ (Supporting Information and Fig. S7 – S9). When $\varphi_1 < \varphi < 0$ and $c < c_R$, the singular strategy d^* is an evolutionary branching point: branching occurs and the population diversifies; simulations show that the population reaches a polymorphic state, with individuals either fully philopatric, or total dispersers (Figs. 2E and 3B). There is no branching when there is no direct cost of dispersal ($c = 0$) because $\varphi_1 = 0$ in this case.

When $\beta = 0$ (fecundity is 0 in one of the patch types), all intermediate strategies are neutrally stable (neither ESS nor branching point). In the absence of variation in fecundity among patches ($f_1 = f_2$, leading to $\gamma = 0$ and $\beta = 1$), selection for philopatry is in accordance with dispersal strategies predicted for generalist species (Cheptou and Massol, 2009; Massol and Cheptou, 2011).

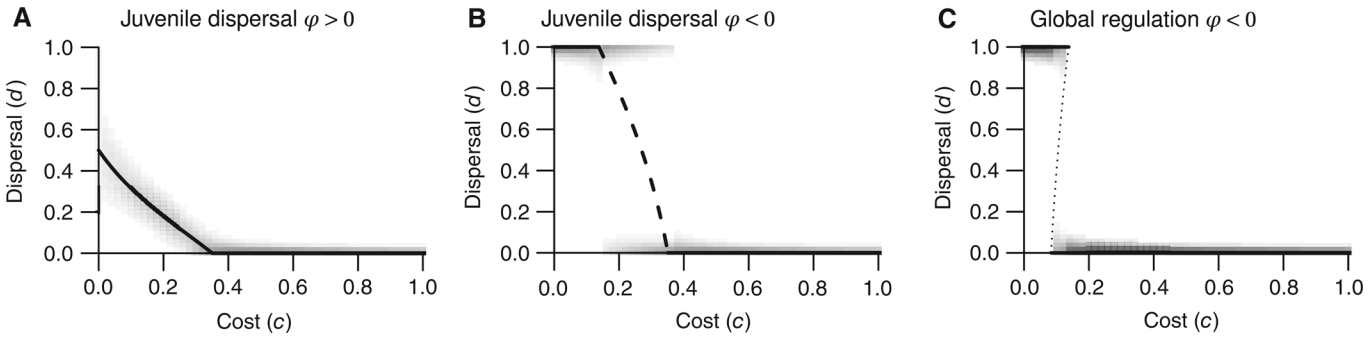

Figure 3. Variation in singular dispersal strategy (ordinates, *d*) with the direct cost of dispersal (abscissas, *c*). (A, B) Evolution of dispersal under juvenile dispersal life cycles. (C) Evolution of dispersal under global regulation life cycles. Parameter values: $\rho = 0.4$, $f_1 = 1$, $f_2 = 4$, (a) $\varphi = 0.5$ or (b, c) $\varphi = -0.5$. Solid lines indicate ESS, dashed lines represent branching points, and dotted gray lines represent evolutionary repellers. Background gray levels indicate the results of simulations in very large populations with no stochasticity in recruitment.

With small patch sizes

When patch sizes are small, kin selection adds another pressure toward higher values of d: the ESS values of d are therefore higher than with infinite population size (compare Fig. S1B, E to Fig. 2B, E), but predictions match under conditions favoring total dispersal, because $d = 1$ is the highest possible value for d. The other main change is the absence of clear branching in the simulations with small patch sizes. As our analytical treatment did not deal explicitly with patches of finite size, interpretation of this last result is speculative at best, but it is remarkable that finite patch size has also been shown to impede evolutionary branching of competitiveness and local adaptation traits (Day, 2001; Ajar, 2003). Here, kin competition might sufficiently select for dispersal that the existence of fully philopatric individuals might not be possible (Figs. S1b, e and S2a, b).

GLOBAL REGULATION LIFE CYCLE

With large patch sizes

Under the global regulation life cycle, only two ESS are possible (Figs. 2C, F, and 3C): total philopatry ($d = 0$) or total dispersal ($d = 1$). Equation (A.7b; in Supporting Information) indicates that total dispersal is an ESS when $\varphi < \varphi_1$ (Figs. S6–S8 and S12–S14), φ_1 being the same as in equation (11). Total philopatry is an ESS for large values of c (Supporting Information, eq. A.7a; the precise value of the threshold c_D is given by equation A.8).

When c is not too high and φ is mildly negative and obeys certain conditions (Supporting Information), total philopatry and total dispersal are simultaneously possible ESS (Figs. 2F, 3C, and Figs. S6–S8 and S12–S14). Under these conditions, an evolutionary repeller exists between the two ESS (its precise value d_D^* is given in the Supporting Information equation A.9). Initial dispersal rates above d_D^* lead to total dispersal, whereas initial dispersal rates below this threshold lead to total philopatry. When $c = 0$ (Fig. 2C), we recover predictions from McNamara and Dall (2011), that is, that the ESS goes from total dispersal to total philopatry with no bistable state as φ increases.

With small patch sizes

Kin competition plays a much weaker role when density regulation is global; the ESS levels of dispersal in the simulations with small patch sizes are therefore closer to the analytical predictions (see Fig. S1C, F, and S2C).

Discussion

We explore the evolution of unconditional dispersal strategies in an environment that varies in both time and space, depending on the order of events in the life cycle. By dispersal, we refer to the probability of emigrating away from the natal patch. We find that the evolution of dispersal strongly depends on the regime of selection and on whether density regulation is local or global. There is no differential local adaptation in our model: we focus on demographic consequences of patch heterogeneity on the evolution of dispersal. We derive our analytical predictions under the assumption of very large patch sizes, hence in the absence of kin competition. Additional numerical simulations conducted with small patch sizes (10 individuals per patch) helped investigate how kin competition changes our predictions. Kin competition adds a pressure toward higher values of d, so that the ESS values in these models are higher than predicted. Moreover, our simulations show that small patch sizes hinder branching of dispersal, in line with conclusions on the effect of small patch sizes on the evolution of local adaptation and competitiveness (Day, 2001; Ajar, 2003).

HETEROGENEITY OF PREDICTIONS UNDER DIFFERENT LIFE CYCLES

In the adult dispersal model, the output of each patch is constant, thereby annihilating demographic differences among patches. A modified dispersal trait is then at best neutral (when $c = 0$),

or detrimental (when $c > 0$). Thus, consistently with other results from the literature (e.g., Hovestadt et al., 2014), the adult dispersal life cycle always selects for zero dispersal because it obliterates variance in fitness expectations among the different patches.

When the output of each patch depends on its composition (juvenile dispersal and global regulation life cycles), total dispersal is selected for when the temporal autocorrelation in patch type is negative and below a threshold φ_1. When the temporal autocorrelation in patch state is above this threshold, the evolved dispersal strategy depends on whether density regulation is a local or global process.

When density regulation occurs locally (juvenile dispersal), different outcomes are possible (Bocedi et al., 2012). When temporal autocorrelation in patch state is negative ($\varphi_1 < \varphi < 0$) and there is a direct cost of dispersal ($c > 0$), evolutionary branching of dispersal occurs: some of the individuals are then totally philopatric ($d = 0$) while the others always disperse ($d = 1$). When temporal autocorrelation in patch state is positive, the dispersal ESS is intermediate ($0 < d < 1$) and decreases with the autocorrelation parameter, in agreement with results of Travis (2001).

Under global regulation, there is no intermediate dispersal strategy: total philopatry is selected for as soon as either temporal autocorrelation in patch state or dispersal costs are high enough. Without a direct cost of dispersal, the switch between total philopatry and total dispersal happens at $\varphi = 0$, a result consistent with previous findings (McNamara and Dall, 2011). When dispersal is costly ($c > 0$), however, there is a range of parameters at which both total dispersal and total philopatry are simultaneously stable. Which strategy is attained depends in this case on the initial conditions.

Explaining why an intermediate dispersal ESS emerges under juvenile dispersal life cycle, but not under global regulation life cycle, is based on analyzing the consequences of the absence of local regulation. Assuming global regulation means that population densities can amply vary between patches that have recently experienced good and bad habitat qualities, and that there is no penalty associated with being in a crowded patch. When φ is mildly positive and $\gamma > 0$ (i.e., when we would expect an intermediate dispersal ESS under the juvenile dispersal life cycle), total philopatry is selected in the global regulation life cycle because (1) population densities accumulate in patches that are currently of good quality and, thus, (2) dispersing means that, on average, a migrant individual is more likely to leave a good patch for a bad patch than the other way round (see also Hastings, 1983). Conversely, in the case of juvenile dispersal life cycle, individuals do not "accumulate" in currently good patches, and thus dispersal between patches belonging to different habitat types is not biased.

DEMOGRAPHIC VERSUS LOCAL ADAPTATION EFFECTS ON THE EVOLUTION OF DISPERSAL

Importantly, our study differs from population genetics studies investigating coevolution of dispersal and local adaptation, that is, where there is an additional locus controlling adaptation to the local patch (e.g., Balkau and Feldman, 1973; Kisdi, 2002; Billiard and Lenormand, 2005; Blanquart and Gandon, 2011; Drown et al., 2013; Blanquart and Gandon, 2014). There is no such locus in our work: emigration probability is the only trait that is variable and under selection; within a patch, all individuals have the same fecundity. We are therefore exploring the importance of demographic effects for the evolution of dispersal, a perspective in line with studies in which habitat quality is not subjected to the effect of a local adaptation locus (e.g., Asmussen, 1983; Hastings, 1983; Bull et al., 1987; Cohen and Levin, 1991; McPeek and Holt, 1992; Johst and Brandl, 1997; Mathias et al., 2001; Parvinen, 2002; McNamara and Dall, 2011).

DIFFERENT TYPES OF ENVIRONMENTAL HETEROGENEITY

In a simulation model, Johst and Brandl (1997) argued that the order of events in the life cycle influenced the evolution of conditional dispersal strategies, that is, when the emigration probability depends on the local conditions, but not of unconditional dispersal strategies (when emigration only depends on the genotype of the individual, but neither on the type of patch it inhabits, nor on the local density of individuals). Even though emigration is unconditional in our model, we do observe a strong effect of the order of events in the life cycle on the evolution of dispersal. The reason for this difference lies in the type of heterogeneity that is modeled. In our model, fecundities differ among patch types, but all patches have otherwise the same, constant, carrying capacity. In Johst and Brandl's (1997) model, fecundities in the different patch types are identical, but carrying capacities differ; in addition, the different patches can be unsaturated, and finally, reproduction and density regulation are coupled (Johst and Brandl used a Ricker model): there are therefore only three effective events in the life cycle. Because regulation is habitat type-dependent, environmental change and regulation do not commute in Johst and Brandl's model, while they do in our model. Dispersal and environmental change still commute when dispersal is unconditional, but do not otherwise. Therefore, there are actually no life cycle differences in Johst and Brandl's model when dispersal is unconditional, which explains why they find that life cycle differences only matter when dispersal is conditional.

Doebeli and Ruxton (1997) showed that while unconditional dispersal is either neutral or counterselected when the metapopulation is at demographic equilibrium, intermediate dispersal rates can be maintained under nonequilibrium dynamics (a situation that can be induced when there is a direct cost of dispersal). In

their model, evolutionary branching can even occur if the patches are ecologically different. Because we assumed that the total size of the metapopulation is constant, these effects of nonequilibrium dynamics are absent from our model; yet, we show that intermediate dispersal rate and evolutionary branching can occur nonetheless. Similarly, Mathias et al. (2001) found evolutionary branching of dispersal strategies in the absence of cost of dispersal and with no temporal autocorrelation in patch types. Here again, this is because local densities are variable in Mathias et al.'s (2001) model, and because the carrying capacities of the patches change with patch type; in our model, these effects are absent because carrying capacities are constant and the same for all patch types.

LIMITATIONS OF THE MODELLING FRAMEWORK

Although widely used in population genetics (the iconic island model, Wright, 1931), our assumptions of an infinite number of patches and of a fixed total size of the population are arguably artificial. As described in the previous section, different modes of density regulation, and in particular, spatiotemporal variations in carrying capacities instead of fecundities, can lead to different predictions.

Our analysis is done under the assumption of large patch sizes in order to focus on the effects of environmental heterogeneity on the evolution of dispersal, thus excluding the role of kin competition. Numerical simulations performed with small patch sizes (Figs. S1 and S2) identify the additional contribution of kin competition: higher levels of dispersal evolve and evolutionary branching is hindered. Finite and equal patch sizes are known to hinder diversification (Day, 2001; Ajar, 2003), but previous models have also shown that asymmetric patch sizes can help recover branching (Massol et al., 2011). Whether it would be the case in our model as well remains to be investigated.

Individuals reproduce clonally in our model; the role of inbreeding avoidance on the evolution of dispersal (Bengtsson, 1978) or the existence of dimorphism of dispersal in sexual species (Hovestadt et al., 2014) are hence out of the scope of our study (and often, the addition of sexual reproduction does not have a substantial effect on the predictions, Parvinen and Metz, 2008; Aguilée et al., 2015). Still, the evolution of dispersal can be affected by sexual reproduction in other, less direct ways (e.g., assortative mating diminishing the potential for dispersal polymorphism, Fronhofer et al., 2011).

Like most models on the evolution of dispersal, we model global dispersal. Including isolation by distance, for instance using a stepping stone model (Gandon and Rousset, 1999) or more generally a network structure (Henriques-Silva et al., 2015) would be substantially more complex with spatial and temporal heterogeneity in habitat quality, because we would need to also define and take into account spatiotemporal correlations in habitat qualities.

EMPIRICAL REPRESENTATIVES OF MODELED LIFE CYCLES

Faced with the variety of predictions coming out of the different life cycles modeled in the present study, an important question is to identify the typical features of a system (i.e., type of organism, regulation mechanism, etc.) leading to life cycles that would most closely resemble the adult dispersal, juvenile dispersal, or global regulation models. Density regulation corresponding to the establishment of offspring and their maturation into adult individuals, the distinction between the two life cycles with local regulation in semelparous organisms boils down to whether adults or juveniles disperse (Débarre and Gandon, 2011). The life cycle with global regulation, by contrast, corresponds to the situation in which regulation occurs at the metapopulation scale, irrespectively of whether adults or juveniles disperse.

Our model assumes discrete and nonoverlapping generations. It is difficult to extrapolate the distinction between adult and juvenile dispersal without first assessing what would change if adults were to survive from one time step to the next. Assuming that adults and juveniles are regulated together at each generation, the models presented in this study could also represent iteroparous organisms provided that (1) adults do not necessarily settle in a given patch for their whole life and (2) the age structure of adults has no impact on the values of their fecundity or survival. In this case, the fecundity parameters f_i would instead represent the sum of adult survival and fecundity (as in classic population genetics models). When adults settle in a single patch for their whole lifetime, the models presented here only work if the time step considered equals a typical organism lifespan and the time scale of environmental change is larger than this typical lifespan. Following this time rescaling, the f_is need to be reinterpreted as lifetime fecundities, that is, the sum of fecundities at ages discounted by survivorship, potentially integrating many rapid fluctuations of the environment within a given time step.

A pending question is now whether data on dispersal in natural systems corroborate or not the predictions of our models. As already stated in the literature (Johnson and Gaines, 1990; Duputié and Massol, 2013), it is quite difficult to find empirical tests of theory on the evolution of dispersal for at least two reasons: (1) techniques to measure dispersal in the field (e.g., from neutral genetic marker assignment or mark capture–recapture models) have come to fruition only recently; (2) parameters considered in theoretical models of the evolution of dispersal are seldom measured in the field. Still, qualitative results obtained on species commonly used to study dispersal and its evolution might evidence blatant agreement or disagreement between our model and existing data.

What we call "adult dispersal" in this article corresponds to dispersal occurring in the already regulated phase of the life cycle. This can arise, for example, in long-lived mobile organisms such as vertebrates (although in their case it is likely that dispersal will

also be conditional, i.e., that the decision to disperse will also depend on the local conditions). In such a case, our model predicts that the spatial and temporal variability in fecundity does not select for more dispersal or, in other words, that selection for dispersal must be looked for in other factors, that is, kin competition (see results in patches of finite size, Figs. S1 and S2) and inbreeding avoidance. In Johnson and Gaines' (1990) review on the subject of dispersal evolution in vertebrates, their Table 2 highlights the fact that almost all empirical tests in mammals and birds had, at the time, focused on inbreeding avoidance, mate competition, and resource competition, not environmental variability (by which we mean, as in the remainder of the article, variations of the environmental factors that are extrinsic to the population). While this does not constitute a test of our model's predictions per se, it tends to confirm that environmental variability could be less relevant, as a cause of dispersal evolution, in organisms with "adult dispersal."

By contrast, "juvenile dispersal" in our work corresponds to dispersal happening before local density regulation. Local regulation of juveniles seems to be a widespread phenomena when space is the limiting resource, for example, in marine species with larval dispersal and a sessile adult stage or in plants: in sessile animals and plants, there is indeed some evidence of intermediate dispersal rates, as predicted by our model, for example, through heterocarpy in plants (Imbert et al., 1997; Hall et al., 2011) or poecilogony in marine invertebrates (Hoagland and Robertson, 1988; Ellingson and Krug, 2006; Krug, 2007).

A global regulation life cycle is a priori more difficult to imagine. However, when limiting factors are shared among patches, such a situation becomes possible, for example, if predators of the modeled species forage at a spatial range that encompasses all prey patches (McCann et al., 2005). Other possibilities of shared limiting factors may include a common pool of resources (e.g., a single aquatic system in which different populations of seabirds might go fishing), diseases or parasites affecting all the patches with the same intensity, or access to limiting mutualists that forage among all patches (e.g., limiting pollinators in a metapopulation of plants). One could also think of anadromous fish species in which most of the population regulation occurs at sea before the final spawning migration, at which point adults could choose to get back to their ancestral river or not (thus dispersing to other patches to reproduce). Given the known limiting factors of such fish species during their spawning stage (i.e., not food), the difference in patch fecundities could be attributable to, for example, differences in predation rates or disease prevalence among rivers, while regulation at sea (when all individuals are in the same pool), in between birth and the spawning migration, would ensure global regulation. In some cases, global regulation could be a transient phase linked to exponential growth of the metapopulation and mechanisms of local regulation could begin to take effect after this temporary period.

Conclusion

Our model and its comparison with past literature on the evolution of dispersal under spatiotemporal variability (McPeek and Holt, 1992; Johst and Brandl, 1997; Mathias et al., 2001; McNamara and Dall, 2011) highlight the fact that apparently small changes in model structure, such as the timing of events, the type of density regulation (global/absent or local), and the type of environmental heterogeneity (productivity *vs.* carrying capacity) can drastically alter the evolution of dispersal. Our results call for an exploration of the robustness of conclusions of already published models to these types of changes before any general claim can be made on how temporal and spatial heterogeneity affect the evolution of dispersal.

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DATA ARCHIVING

Codes for simulating the model are available on figshare at http://dx.doi.org/10.6084/m9.figshare.1180191. A Mathematica notebook detailing the analytical computations given in the text and in appendices is available on figshare at http://dx.doi.org/10.6084/m9.figshare.1195906.

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Supporting Information

Additional Supporting Information may be found in the online version of this article at the publisher's website:

Figure S1: Evolution of dispersal under the three different life cycles (columns), for different values of the direct cost of dispersal c (first line, no cost; second line $c = 0.1$).
Figure S2: Variation in singular dispersal strategy (ordinates, d) with the direct cost of dispersal (abscissas, c).
Figure S3: Evolutionary outcomes under juvenile dispersal life cycles.
Figure S4: Evolutionary outcomes under juvenile dispersal life cycles.
Figure S5: Evolutionary outcomes under juvenile dispersal life cycles.
Figure S6: Evolutionary outcomes under global regulation life cycles.
Figure S7: Evolutionary outcomes under global regulation life cycles.
Figure S8: Evolutionary outcomes under global regulation life cycles.
Figure S9: Evolutionary outcomes under juvenile dispersal life cycles.
Figure S10: Evolutionary outcomes under juvenile dispersal life cycles.
Figure S11: Evolutionary outcomes under juvenile dispersal life cycles.
Figure S12: Evolutionary outcomes under global regulation life cycles.
Figure S13: Evolutionary outcomes under global regulation life cycles.
Figure S14: Evolutionary outcomes under global regulation life cycles.