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Interim Report **IR-01-043**

Invasion Fitness and Adaptive Dynamics in Spatial Population Models

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IIASA STUDIES IN ADAPTIVE DYNAMICS NO. 55

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Contents

Abstract

Disentangling proximate and ultimate factors of dispersal and assessing their relative effects requires an appropriate measure of fitness. Yet there have been few theoretical attempts to coherently define fitness from demographic “first principles”, when space-related traits like dispersal are adaptive. In this chapter, we present the framework of adaptive dynamics and argue that *invasion fitness* is a robust concept accounting for ecological processes that operate at the individual level. The derivation of invasion fitness for spatial ecological scenarios is presented. Spatial invasion fitness involves the effect of neighbors on a focal individual, mediated by coefficients analogous to relatedness coefficients of population genetics. Spatial invasion fitness can be used to investigate the joint evolution of dispersal and altruism—two traits that both have a direct influence on, and are strongly responsive to, the spatial distribution of individuals. Our deterministic predictions of dispersal and altruism evolution based on spatial invasion fitness are in good agreement with stochastic individual-based simulations of the mutation-selection process acting on these traits.

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Invasion Fitness and Adaptive Dynamics in Spatial Population Models

Régis Ferrière
Jean-François Le Galliard

1. Introduction

Even in homogeneous habitats, *spatial fluctuations* of population size arise inevitably as a result of demographic stochasticity, and *spatial correlations* build up from the imperfect mixing of individuals, induced by their limited range of dispersal (Tilman & Kareiva, 1998; Dieckmann *et al.*, 2000). As a consequence, selective forces acting on the life-history traits of individuals are neither uniform nor independent across space. Dispersal propensity (in the broad sense of natal dispersal and breeding dispersal) is therefore a pivotal component of the individuals' phenotype, for it is both a target of selection and a primary factor in spatial fluctuations and correlations in the selective regime (Ferrière *et al.*, 2000).

Since the seminal work of Hamilton & May (1977), we know that the avoidance of competition with related individuals is an important factor in explaining the evolution of dispersal. It has recently been argued that dispersal probabilities evolving under the sole effect of kin competition provide a null model against which to assess the relative importance of alternative selective forces, as predicted by more elaborate kin selection models (Ronce, 1999). In kin selection theory based on diallelic, haploid genetics, the commonly used measure of fitness is *invasion fitness*, that is, the per capita growth rate of a mutant when rare. For pairwise interactions involving an "actor" and a "recipient", the definition of invasion fitness involves the *relatedness* of the recipient to the actor (Grafen 1979), for which the correct definition is the probability that the recipient is a mutant (Day & Taylor, 1998). However, this assumes that the altered phenotype of a mutant has no effect on that probability and therefore does not change relatedness. Obviously, this does not hold true when the phenotypic traits under consideration, like dispersal, modify the distribution of individuals across space. Furthermore, modelling a mutant's initial rarity requires some care in spatial models (Rousset & Billiard, *manuscript*), for the population size is locally finite everywhere, and the initial number of mutants may not be regarded locally as infinitesimal.

The purpose of this chapter is to provide a modeling framework that allows us to investigate the evolutionary dynamics of adaptive, continuous traits, while accounting explicitely for both the reciprocal effects of these traits on the spatial distribution of individuals, and for the effects of the spatial heterogeneity of selective pressures on the traits' evolutionary dynamics. In section 2, we provide a general argument that the notion of invasion fitness is appropriate to capture "first" demographic principles

operating at the level of individuals, and to describe the long-term evolutionary dynamics of adaptive life-history traits (Metz *et al.*, 1992, 1996; Dieckmann & Law, 1996; Geritz *et al.* 1997, 1998). We then present, in section 3, van Baalen and Rand's (1998) extension of the notion of invasion fitness to spatially heterogeneous populations. *Spatial invasion fitness* is derived from first demographic and behavioral principles operating at the levels of individuals and their nearby neighbors. In non-spatial populations, where individuals are assumed to be constantly well-mixed and interactions occur at random between them, invasion fitness can be obtained as the Malthusian growth rate of a simple birth-and-death process (Ferrière & Clobert, 1992; Metz *et al.*, 1992; Ferrière & Gatto, 1995). In contrast, when interactions develop locally and dispersal is limited to neighborhoods, the process of mutant growth should be modelled by keeping track of spatial statistics that describe local population structures beyond global densities. The theory of *correlation equations* (Matsuda *et al.*, 1992; Morris, 1997; Rand, 1998) provides the appropriate mathematical tools. Under certain assumptions about habitat structure and the model's mathematical properties, invasion fitness can then be obtained as the dominant eigenvalue of a matrix (van Baalen & Rand, 1998), just as one would recover the population growth rate of a simple Leslie model (Caswell, 1989). In the spatial setting, the matrix involved contains demographic parameters that depend upon the local spatial structure of the population.

In section 4 we use this framework to investigate the joint evolution of dispersal and altruistic behavior. The evolution of dispersal and the evolution of altruism have been the focus of two rather independent lines of research that trace back to the seminal work of Hamilton (1964). Yet there are serious reasons for trying to merge these lines. With limited dispersal, individuals are likely to interact with relatives, and kin selection models would then predict altruism to evolve. Yet neighbors do not only interact socially; they compete with each other as well. Thus, clustering of relatives may not be sufficient for sociality to evolve. A dose of dispersal might be needed, so that a locally successful strategy can be exported throughout the resident population. Co-adaptive changes in dispersal and social behavior may thus be expected. A detailed exploration of these questions is to be found in Ferrière & Le Galliard (in prep.) and Le Galliard et al. (a and b, in prep.).

2. Adaptive dynamics and the concept of invasion fitness

We will first introduce the basics of a general and coherent mathematical theory of Darwinian evolution which aims at describing the evolutionary dynamics of adaptive, continuous traits. This *adaptive dynamics theory* (founding papers are Metz *et al.*, 1992, Metz *et al.*, 1996; Dieckmann & Law, 1996; Geritz et al., 1997) satisfies three important requirements:

- Adaptive dynamics are modelled as a macroscopic description derived from microscopic mechanisms. Selective pressures are set by ecological mechanisms operating at the "microscopic" level of individuals.
- Adaptive dynamics incorporate the stochastic elements of evolutionary processes, arising from the random process of mutation and from the extinction risk of initially small mutant populations.

- Adaptive dynamics describe evolution as a dynamical process, identifying potential evolutionary endpoints, and among them those which, indeed, are attractors for the traits' dynamics.

In this section, we present a brief overview of the principles of adaptive dynamics modelling, to show that a consistent measure of fitness arises naturally from the description of microscopic processes underlying ecological interactions (the reader should refer to Metz *et al.*, 1992, Marrow *et al.*, 1992, and Dieckmann & Law, 1996, for a more thorough treatment). In the following sections, we shall see how to derive this fitness measure for a class of spatial population models where the individual probability of dispersal is one of the adaptive traits under consideration.

The canonical equation of adaptive dynamics

We consider a closed population of a single species. Individuals are characterized by a suite of adaptive, quantitative traits which define their *phenotype*. They reproduce and die at rates that depend upon their phenotype and their environment, including external factors as well as their own congeneric population. Haploid inheritance is assumed, and there is a non-zero probability for a birth event to produce a mutant offspring, that is, an individual that differs from its parent in one of the traits. Individuals interact with each other, and the process of selection determines changes in the abundance of each phenotype through time.

Direct individual-based models accounting for the stochasticity of birth, death, and mutation events could be run to study how the distribution of phenotypes present in the population evolves through time. The theory of adaptive dynamics was developed as an alternative to intensive computer calculations, to provide a handy, deterministic description of the stochastic processes of mutation and selection.

Adaptive dynamics models rest on two basic principles (Metz *et al.*, 1996): mutual exclusion, "in general two phenotypes x and x' differing only slightly cannot coexist indefinitely in the population"; and time scale separation, "the time scale of selection is much faster than that of mutation". Thus, one may regard the adaptive dynamics as a trait substitution sequence. Each step occurs at a rate equal to the probability $w(x'|x)$ per unit time for a specific phenotype substitution, say x' substituted to x. The so-called *canonical equation* of adaptive dynamics then describes how the mean of the probability distribution of trait values in the evolving population changes through time. If we keep using x to denote this mean, the canonical equation reads (Dieckmann & Law, 1996):

$$\frac{d}{dt}x = \int (x'-x)\cdot w(x'|x)\,dx' \tag{2.1}$$

where the integral sum is taken over the whole range of feasible phenotypes.

Following on the traditional view of the evolutionary process as a hill-climbing walk on an adaptive landscape (Wright, 1931), we seek to recast the canonical equation into the form

$$\frac{d}{dt}x = \eta(x)\cdot \frac{\partial}{\partial x'} W(x',x)\bigg|_{x'=x} \tag{2.2}$$

where the coefficient $\eta(x)$ would scale the rate of evolutionary change, and $W(x',x)$ *would rigorously define the measure of fitness of individuals with trait value* x' *in the environment set by the bearers of trait value x.* This mathematical derivation ought to be underpinned by a biologically consistent description of the mutation-selection process.

Mutant invasion rate as a measure of fitness

To recast the canonical equation (2.1) into the form of equation (2.2), we first expand $w(x'|x)$ as the product of a mutation term and a selection term. To keep notations simple, we shall restrict ourselves to the case where phenotypes are characterized by a single trait. The mutation term is the probability per unit time that the mutant enters the population. It involves four multiplicative components: the per capita birth rate $b(x)$ of phenotype x, the fraction $\mu(x)$ of births affected by mutations, the equilibrium population size $\hat{n}_x$ of phenotype x, and the probability of a mutation step size $x'-x$ from phenotype *x*. The selection term is the probability that the initially rare mutant gets to fixation. Under the assumption that the population is *well mixed*, we can neglect the effects of the mutant density on the demographic rates of the mutant and resident populations. Let us denote the per capita birth and death rates of the rare mutant in a resident population of phenotype x by $b(x',x)$ and $d(x',x)$. Then, the difference $b(x',x)-d(x',x)$ measures the mutant invasion rate, that is, the per capita growth rate of initially rare mutants, hereafter denoted by $s(x',x)$. The theory of stochastic birth-and-death processes (e.g. pages 39-41 in Renshaw, 1991) shows that the probability that the mutant population escapes initial extinction starting from size 1 is zero if $s(x',x)<0$, and is approximately equal to $s(x',x)/b(x',x)$ otherwise.

Altogether we obtain

$$w(x'|x)=\mu(x)\cdot b(x)\cdot \hat{n}_x \cdot M(x,x'-x)\cdot \frac{[s(x',x)]_+}{b(x',x)} \tag{2.3}$$

where $\hat{n}_x$ denotes the equilibrium population size of phenotype x. The quantity $[s(x',x)]_+$ is equal to $s(x',x)$ if $s(x',x)>0$, and to zero otherwise; this means that only advantageous mutants, with a positive invasion rate, have a non-zero chance of getting established. Up to first order in the mutation step size $x'-x$ we have also

$$\frac{s(x',x)}{b(x',x)} \approx \frac{1}{b(x)}\cdot (x'-x)\cdot \left.\frac{\partial s}{\partial x'}\right|_{x'=x} \tag{2.4}$$

where we have used $s(x,x)=0$ since the population of phenotype *x* is at demographic equilibrium. If we assume the mutation process to be symmetric, and denote the variance of the mutation distribution *M* by $\sigma^2(x)$, we can insert equation (2.3), together with equation (2.4), into equation (2.1) and compute the integral to obtain (Dieckmann & Law, 1996)

$$\frac{d}{dt}x=\left[\mu(x)\cdot\frac{\sigma^2(x)}{2}\cdot\hat{n}_x\right]\cdot\left.\frac{\partial s}{\partial x'}\right|_{x'=x} \tag{2.5}$$

which precisely conforms to equation (2.2).

According to this deterministic approximation of adaptive dynamics, the evolutionary rate $\eta(x)$ of equation (2.2) is given by the bracketed product which encapsulates the influence of mutation. Most importantly, this derivation identifies the mutant invasion rate $s(x', x)$ as the appropriate measure of fitness denoted by $W(x', x)$ in equation (2.2). Therefore, we call $s(x', x)$ the mutant *invasion fitness*.

Invasion fitness, ESS, CSS, and evolutionary branching

The *selection derivative* (Marrow *et al.*, 1992), $\partial s/\partial x'\big|_{x'=x}$, determines the direction of adaptive change. When the selection derivative is positive (or negative), an increase (or a decrease) of the trait value x will be advantageous in the vicinity of the resident trait value. Phenotypes that nullify the selection derivative are called *evolutionary singularities* and represent potential end points for the evolutionary process. Yet careful inspection of stability properties of evolutionary singularities is required before conclusions can be drawn about the adaptive dynamics in their vicinity (Geritz *et al.*, 1998):

- If invasion fitness reaches a local maximum at an evolutionary singularity, then this singularity is an *evolutionarily stable strategy* (ESS), in the classical terminology of evolutionary biology.
- An ESS need not be attainable: if the selection derivative increases near the ESS, any evolutionary trajectory starting nearby will actually be repelled away from the ESS. In this case, the ESS also is an *evolutionary repellor*.
- Conversely, a singularity may attract evolutionary trajectories and yet correspond to a fitness minimum. In this perhaps most remarkable case, selection is initially stabilizing and drives the population to a point where ecological interactions turn the selective regime into a disruptive one, and dimorphism evolves. This phenomenon is known as *evolutionary branching*. The canonical equation for adaptive dynamics provides an approximate model for evolutionary trajectories heading to a branching phenotype, but obviously fails to capture the population's further evolutionary dynamics.

3. Spatial invasion fitness in homogeneous habitats

One conclusion to be drawn from the previous section is that the derivation of invasion fitness must be underpinned by an ecological model for the population dynamics. The definition of a fitness measure as a function of space-related traits therefore requires that spatial structure and local interactions are both incorporated into the underlying ecological model.

Spatial population models

Spatial models fall into two main categories, depending on the continuous versus discrete structure of the habitat. Traditional models for continuous space (*reaction-diffusion models;* see Okubo, 1980) run into serious biological inconsistencies, like the assumption that infinitely many "nano-individuals" may live in arbitrarily small areas. It is only recently that two new types of mathematically sound and biologically consistent

models have been derived. *Hydrodynamics limit models* are spatially explicit; akin to reaction-diffusion equations, they involve correction terms that account for local interactions and dispersal (Durrett & Levin, 1994). *Moment equations* are spatially implicit; they describe the dynamics of the statistical moments of the distribution of individuals in space (Bolker & Pacala, 1999; Dieckmann & Law, 2000). For modelling spatial population processes over discrete space, there is a long tradition of *metapopulation models* (Levins, 1969; Hanski & Gilpin, 1997; Hanski, 1999, and references therein). Classical models of metapopulations are not truly spatial in the sense that they do not involve the notion of neighborhood; dispersal is global, and all dispersing individuals, irrespective of their location, are mixed into a common pool before being redistributed to patches.[1] *Stepping-stone models* (Kendall, 1948; Kingman, 1969; Renshaw, 1986) assume that a set of finite populations is distributed on a regular lattice of patches. Dispersal takes place between neighboring patches. In the field of population genetics, stepping-stone models usually assume that all patches are saturated to their carrying capacity (Malecot, 1948; Kimura, 1953; Malecot, 1975). *Lattice models* (Matsuda *et al*., 1992; Morris, 1997; Rand, 1998) have been developed recently as another tool for modelling population dynamics in discrete space. Lattice models prescribe the possible locations of individuals on a network of sites, each site hosting at most one individual. There is no saturation assumption: all sites need not be occupied. Local interactions and local dispersal occur between any site and its neighborhood of connected sites. Like moment equations, lattice models are spatially implicit, and they aim at describing neighbor-range spatial correlations.

When it comes to deriving a measure of invasion fitness from these ecological models, operational results are scant. So far, no rigorous invasion criterion has been able to be established for models of hydrodynamics limits or moments. Invasion fitness in metapopulations has been worked out by Olivieri *et al.* (1995) and, in greater generality, by Metz and Gyllenberg (*in press*). However, as we have already pointed out, such models do not account for limited dispersal, and therefore address spatial processes in a rather special way. The study of interacting populations, using stepping-stone models, remains very limited. Only lattice models have led to a rigorous mathematical definition of invasion fitness in space (van Baalen & Rand 1998), and it is models of this type that we shall consider further in the rest of this chapter.

Modeling the spatial dynamics of population lattices

The population is distributed over an infinite network, or lattice, of connected sites (Fig. 1). A site contains at most one individual. Interactions (social, competitive, parasitic, etc.) may occur only between individuals that inhabit connected sites, and movement may occur only from a given site to a connected site. This has the important consequence that the spatial scale is the same for dispersal and interactions. For simplicity, we shall assume that each site is connected to the same number (n) of neighboring sites (e.g. a regular lattice). Each site is in one of a limited number of possible states: empty, or occupied by an individual of one out of N possible types. The

[1] For the sake of completeness, we should mention the so-called *two-patch* or *n-patch models* frequently used (possibly overused) to describe local population regulation by means of simple nonlinear density-dependence (like the Ricker map). For examples and corresponding references, see chapter 3 in Hanski (1999). Unfortunately, as they treat the densities of local populations as continuous variables, they have to rely on the rather unsatisfactory premise that local population size is infinite.

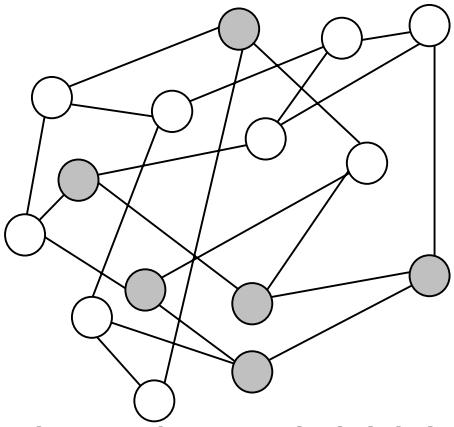


Figure 1. Example of random lattice. Each site is randomly linked to a fixed number *n* of other sites. Here $n = 3$. Dark circles are occupied sites; open circles are empty sites.

configuration of the whole lattice is given by the states of all sites. The lattice configuration changes as a result of two types of events potentially affecting any site during any short time interval: birth or immigration of an individual from a neighboring site, and death or emigration of the individual occupying a site. In general, dispersal (emigration-immigration) is not restricted to the newborn class.

We aim at describing the temporal dynamics of the frequencies of sites that are empty and sites that are occupied by any given phenotype (Matsuda *et al.*, 1992; Rand, 1998). The probability that the state of a site changes depends not only on its current state but also on the state of neighboring sites, for two different reasons. On the one hand, dispersal and birth are local events whose realization is conditional on the availability of empty sites in the neighborhood. The likelihood that an individual in a given site moves or exports its offspring is proportional to the frequency of empty sites in its neighborhood. On the other hand, local interactions with neighbors will affect the birth rate and death rate of any focal individual. For example, individuals might negatively affect each other's birth rate through local competition for food. In this case, the birth rate could be seen as a decreasing function of the number of neighbors.

Therefore the *frequency of sites in state i* among all sites in the lattice, p_i, must depend on the neighborhood structure, as described by a second-order statistic for the distribution of the configurations of all pairs of nearest-neighbor sites. The dynamics of pair configurations depend in turn on the state of triplets including the pairs' neighbors, and so on. A full description of the lattice dynamics eventually requires an infinite hierarchy of statistics, each one describing the spatial structure on a particular scale (sites, pairs, triplets and so on) in relation to the next one (Morris, 1997). To make a model tractable, one has to choose a particular scale of description, and make appropriate approximations to close the exact, infinite system at that scale. This means that the frequencies of configurations beyond the chosen spatial scale are estimated from the frequencies of configurations up to that scale. No mathematical procedure is currently available to systematically identify the scale at which the system should be closed and the closure procedure that should be applied in order to obtain the best approximation of the dynamics of the infinite-dimensional model. This will depend on the particular model under consideration and on the biological motivation guiding the analysis (Morris, 1997; Dieckmann & Law, 2000).

Our aim is to describe the dynamics of lattices at the most local scale, that of pairs of nearest neighbors. Pair-dynamics models can account for the effect of spatial correlations which arise at a local scale and vanish quickly, although they are not concerned with the development of large-scale spatial structures. It should be noticed

that, at least for regular lattices, one may straightforwardly recover the frequencies of sites in the various states (i.e., the p_i's) simply by adding the appropriate pair frequencies. Pair-dynamics models offer a handy compromise between the need to incorporate and describe some of the spatial complexity of the population dynamics, and the aim of deriving useful analytical results on population equilibrium and invasion conditions. The pair-dynamics approach has been used to construct appropriate correlation equations for plant dynamics models (Harada & Iwasa, 1994; Satō & Konno, 1995), spatial games (Morris, 1997; Nakamaru *et al.*, 1997), social interactions (Matsuda *et al.*, 1992; Harada *et al.*, 1995; van Baalen & Rand, 1998), and epidemic models (Keeling, 1996; Morris, 1997). In the case of a spatial game on a regular lattice, however, Morris (1997) showed that the pair-dynamics description could fail dramatically. Then, moving up to the triplet dynamics is often sufficient to obtain a substantial improvement in the closure accuracy.

From individuals to pair dynamics and correlation equations

We define p_{ij} as the frequency of pairs of nearest-neighbor sites, one being in state *i*, and the other in state *j*. Such a pair is denoted by (i, j), and the frequency p_{ij} is calculated over all pairs[2] in the lattice. We shall take four heuristic steps in order to derive the so-called *correlation equations*—that is, a set of nonlinear differential equations that describe the lattice dynamics at the spatial scale of pairs. The four steps are:

1. Write the rates of local events for anchored pairs. We call *anchored pair* one that contains a given site *z* occupied by an individual in a specified state *i*. By definition, *local pair events* affect anchored pairs, and are triggered by a site event at the anchored site *z* (see Fig. 2). Four local events have to be considered (see paragraph below for details).
2. Average the rates of local events for anchored pairs calculated at Step 1 over all sites *z* in state *i*.
3. Calculate the rate of change of the frequency of all (i, j) pairs by bookkeeping all possible transitions of anchored pairs that may create or destroy an (i, j) pair.
4. Apply an appropriate closure procedure designed to approximate all statistics involving triplets in terms of statistics for pairs.

(See Morris, 1997, and Rand, 1998, for a rigorous account of all mathematical details involved).

[2] Note that the pairs are symmetric, which implies $(i, j) = (j, i)$.

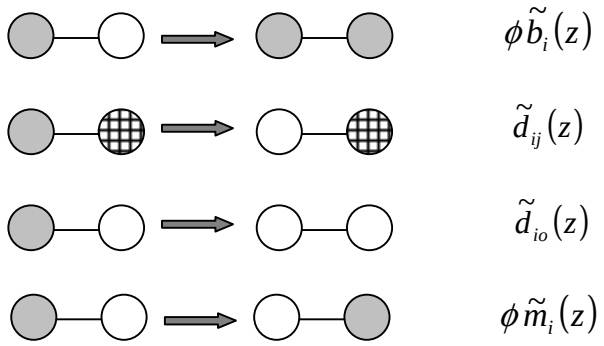


Figure 2. The four local pair events and their rates. Open circles are empty sites. Each dark circle is occupied by a type *i* individual. Hatched circles are in state *j*. See text for notations and explanations.

Step 1. Transition rates for anchored pairs. We define the anchored pair $(i \in z; j \in z')$ to be the pair spanning the sites located at *z* and *z'*, and hosting a type *i* individual in site *z* while site *z'* is in state *j*. We consider the four local events that can affect such a pair as a result of an individual event occuring at *z* (Fig. 2): a birth event at *z* when *j* is the empty state; two mortality events affecting the *i* individual at *z*, differing in the presence or absence of an individual at *z'*; a dispersal event from *z* to *z'*, assuming *z'* to be empty. The individual birth rate, death rate, and dispersal rate involve three additive components: an intrinsic, baseline rate that may depend on the individual's phenotype, an interaction term that measures the effect of neighbors, and a cost term that depends on the individual's phenotype. To calculate the rate of local events, we must introduce the number $n_{k:ij}(z)$ of neighboring sites in state *k* next to the *z* site of an anchored pair $(i \in z; j \in z')$. We simply add the contributions to the event rate affecting the *i* individual at *z* resulting from all possible configurations of the neighborhood of site *z*. The per-capita rate of the birth and dispersal local events should be scaled by ϕ, the inverse neighborhood size. This reflects the fact that a birth or dispersal event affecting, at a given rate, a focal individual that belongs to *n* pairs, will affect any of these pairs at a rate *n* times slower; in contrast, a death event at *z* will concommitently affect all *n* pairs containing *z*. Altogether, this yields the following rates for each of the transitions depicted in Fig. 2:

$$\phi\tilde{b}_i(z) = \phi\left(b_i(z) + \sum_{k\in N} E^b_{ik}(z) n_{k:io}(z) - C^b_i(z)\right) \tag{3.1a}$$

$$\tilde{d}_{ij}(z) = d_i(z) + E^d_{ij}(z) + \sum_{k\in N} E^d_{ik}(z) n_{k:ij}(z) + C^d_i(z) \tag{3.1b}$$

$$\tilde{d}_{io}(z) = d_i(z) + \sum_{k\in N} E^d_{ik}(z) n_{k:io}(z) + C^d_i(z) \tag{3.1c}$$

$$\phi\tilde{m}_i(z) = \phi m_i \tag{3.1d}$$

Notice that, for the sake of simplicity, we have assumed that the intrinsic dispersal rate $m_i(z)$ of any focal individual was merely equal to the intrinsic dispersal rate. There is, however, no conceptual predicament entailed by extending the model and making dispersal conditional on the neighborhood composition (Rand, 1998).

Step 2. Averaging transition rates for anchored pairs over the lattice. Assuming that the lattice is homogeneous, we can take the intrinsic rates, the interaction effects and the costs of interaction to be independent of the location *z* of any focal individual, and set $b(z) \equiv b$, $d(z) \equiv d$, $m(z) \equiv m$, $E^b_{ij}(z) \equiv E^b_{ij}$, $E^d_{ij}(z) \equiv E^d_{ij}$, $C^b_i(z) \equiv C^b_i$ and $C^d_i(z) \equiv C^d_i$.

Transition rates for anchored pairs given by equations (3.1) are still influenced by the local configurations of the lattice, through the neighborhood-structure terms $n_{k:ij}(z)$, which depend on the location *z*. *Local fluctuations* caused by demographic stochasticity induce spatial variations in the neighborhood structure. If we would know at any time the state of every site *z*, then we could calculate each $n_{k:ij}(z)$ and obtain all transition probabilities for each anchored pair. However, the large number of sites makes this endeavor hopeless. Instead, we aim at deriving average transition rates for anchored pairs across the lattice. We first compute an average measure of the neighborhood structure, $\overline{n}_{k:ij} = \sum n_{k:ij}(z) / |i|$, calculated as the total number $|i|$ of sites in state *i* becomes is very large; the sum is taken over all sites *z* that host a type *i* individual belonging to an (i, j) pair. Likewise, we define $q_{k:ij}$ as the average proportion of sites in state *k* in the neighborhood of a site in state *i* within a (i, j) pair; in other words, $q_{k:ij}$ is the conditional probability of having a site in state *k* in the vicinity of a site in state *i*, given that one of the latter's neighboring sites is in state *j*. Since a focal site in an anchored pair is connected to $(n-1)$ sites outside that pair, we have $\overline{n}_{k:ij} = (n-1)q_{k:ij}$. This averaging procedure applied to all local pair-events rates, equations (3.1), eventually yields the following average rates:

$$\phi \overline{b}_i = \phi\left(b_i + \sum_{k\in N} E^b_{ik}\,(n-1)\,q_{k:io} - C^b_i\right) \tag{3.2a}$$

$$\overline{d}_{ij} = d_i + E^d_{ij} + \sum_{k\in N} E^d_{ik}\,(n-1)\,q_{k:ij} + C^d_i \tag{3.2b}$$

$$\overline{d}_{io} = d_i + \sum_{k\in N} E^d_{ik}\,(n-1)\,q_{k:io} + C^d_i \tag{3.2c}$$

$$\phi \overline{m}_i = \phi\, m_i \tag{3.2d}$$

Step 3. Pair transition rates and equations for pair dynamics. To compute the transition rates for all possible pairs, we have to complete the bookkeeping of all local pair events that may create or destroy any given pair, and use the average rates given by equations (3.2). This is done in Box 1 for one particular type of pair, in the case of a lattice where there are three possible states for a site: empty, or occupied by one of two types. Once all pair transition rates are available, it is straightforward to assemble a system of differential equations that govern the temporal dynamics of pair frequencies. It turns out that the combinations of rates that enter these equations can be simplified by making use of the following composite rates (van Baalen & Rand, 1998):

- $\alpha_{ij} = (1-\phi)(\overline{b}_i + \overline{m}_i)q_{i:oj}$ is the rate at which type *i* enters a pair (o, j) with $j \neq i$,
- $\beta_i = \phi\,\overline{b}_i + (1-\phi)(\overline{b}_i + \overline{m}_i)q_{i:oi}$ is the rate at which type *i* enters a pair (o,i),
- $\delta_{ij} = \overline{d}_{ij} + (1-\phi)\overline{m}_i\, q_{o:ij}$ is the rate of loss of type *i* from (i, j) pairs.

We shall refer to these equalities as equations (3.3a), (3.3b), and (3.3c), respectively. It is also convenient to introduce the auxiliary parameter $\alpha'_{ij} = (1-\phi)(\overline{b}_i + \overline{m}_i)$.

Box 1 – Derivation of pair dynamics

We consider a dimorphic population with two types of individuals, *x* and *y*. We perform the bookkeeping of all possible transitions and their rates that may create or destroy (x,o) pairs. The frequency of this pair is affected by six potential events, which can be grasped easily by mere graphical depiction (Fig. B1; also see van Baalen & Rand, 1998). The rate of each transition is computed by summing the appropriate average rates of local pair events.

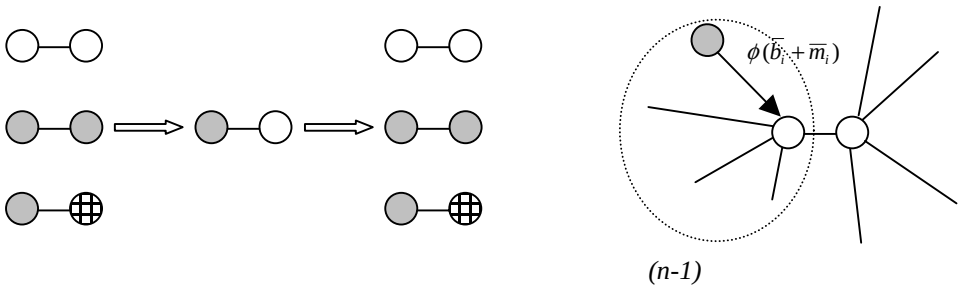


Figure B1. How local pair events affect the pair (x,o). (**a**) All possible transitions that may create and destroy the focal pair (in the middle). (**b**) An example of a local pair event showing how a pair (x,o) can be created from a pair (o,o): reproduction or dispersal occurs in an anchored pair that belongs to the neighborhood of one of the empty sites of the focal pair. This happens at rate $\phi\left(\bar{b}_x + \bar{m}_x\right)$ for each of the $(n-1)q_{x:oo}$ possible anchored pairs under consideration.

Pairs (x,o) are created by:

- the transition from (o,o), as illustrated in Fig. B1. There are, on average, $(n-1)q_{x:oo}$ anchored pairs (x,o) whose empty site belongs also to a pair (o,o); the empty pair (o,o) will be turned into an (x,o) pair by reproduction at the local pair-event rate $\phi\bar{b}_x$, and by dispersal at the rate $\phi\bar{m}_x$.
- the transition from (x,x), either due to death at rate $\bar{d}_{xx}$ or to movement towards a neighboring site. In the latter case, there are $(n-1)q_{o:xx}$ anchored pairs that may undergo the corresponding transition, each at an average local pair-event rate $\phi\bar{m}_x$.
- and the transition from (x,y), which is calculated in a similar way.

Pairs (x,o) are destroyed by:

- the transition to (o,o), due to death at rate $\bar{d}_{xo}$ or to dispersal. Again, we calculate the number of anchored pairs where this transition may take place to be $(n-1)q_{o:ox}$, and for each of them the transition occurs at the rate $\phi\bar{m}_x$.

- the transition to (x,x), due to reproduction within this pair at rate $\phi\bar{b}_x$ or to a reproduction or dispersal event involving an x neighbor. The latter transition involves $(n-1)q_{x:ox}$ (x,o) anchored pairs, which are affected by a local birth event at rate $\phi\bar{b}_x$ and by a local dispersal event at rate $\phi\bar{m}_x$.
- likewise, the transition to (x,y) involves $(n-1)q_{y:ox}$ anchored pairs (y,o), undergoing local birth at rate $\phi\bar{b}_y$ and local dispersal at rate $\phi\bar{m}_y$.

Collecting all these transition rates together, and using the notation $\bar{\phi}=(n-1)\phi$, we finally obtain the following rate of change for the pair frequency p_{xo}:

$$\begin{aligned}\frac{dp_{xo}}{dt} = {} & \left(\bar{b}_x+\bar{m}_x\right)\bar{\phi}\,q_{x:oo}\,p_{oo}+\left(\bar{d}_{xx}+\bar{\phi}\,\bar{m}_x\,q_{o:xx}\right)p_{xx}+\left(\bar{d}_{yx}+\bar{\phi}\,\bar{m}_y\,q_{o:yx}\right)p_{xy} \\ & -\left(\phi\bar{b}_x+\bar{d}_{xo}+\bar{\phi}\,\bar{m}_x\,q_{o:xo}+\bar{\phi}\left(\bar{b}_x+\bar{m}_x\right)q_{x:ox}+\bar{\phi}\left(\bar{b}_y+\bar{m}_y\right)q_{y:ox}\right)\end{aligned} \quad \text{(B1.1)}$$

Step 4. Closing the system. The equations for pair frequencies obtained at Step 3 involve the conditional probabilities $q_{k:ij}$. This implies that the system is not closed: The frequencies of pairs depend on the frequencies of triplets, and to avoid a cascade of dependency on even more complex configurations, the frequencies of configurations involved beyond pairs have to be approximated from the pairs. Finding an accurate approximation amounts to solving the "closure problem" posed by the dynamical system under concern.

The general form of such a *pair approximation* can be written as $q_{k:i}$, the probability that there is a site in state *k* next to a site in state *i*, plus an error term capturing an estimation bias due to local fluctuations (Morris, 1997). Different pair approximations have been developed, reflecting different ways of correcting for the neighborhood structure (Matsuda *et al.*, 1992; van Baalen, 2000), the lattice regularity (Morris, 1997), and the distribution of local fluctuations (Morris, 1997). Ad hoc corrections accounting for the population clustering pattern have also been proposed (Satō *et al.*, 1994). In general, we can safely assume that an infinite random lattice, or a more regular lattice with weak aggregation, will produce a small bias. The *standard pair-approximation* (Matsuda *et al.*, 1992) precisely equals the bias to zero and therefore reads $q_{k:ij}\cong q_{k:i}$. It has been challenged against individual-based simulations in a number of models corresponding to various biological situations (Matsuda *et al.*, 1992; Harada & Iwasa, 1994; Satō & Konno, 1995; Kubo *et al.*, 1996; Nakamaru *et al.*, 1997). The match is often very good, but sometimes devastatingly bad. In such cases, moving up the description level to the spatial scale of triplets can suffice to improve matters substantially (Morris, 1997). Satō *et al.* (1994), Harada *et al.* (1995), Ellner *et al.* (1998), Morris (1997), and van Baalen (2000) have investigated the alternative path of deriving better pair approximations.

Here, we shall content ourselves with the standard pair approximation and apply it to equations (3.2) and (3.3). This yields

$$\phi\bar{b}_i=\phi\left(b_i+\sum\nolimits_{k\in N}E^b_{ik}\,(n-1)\,q_{k:i}-C^b_i\right) \quad \text{(3.4a)}$$

$$\bar{d}_{ij} = d_i + E^d_{ij} + \sum_{k\in N} E^d_{ik}\,(n-1)\,q_{k:ij} + C^d_i \tag{3.4b}$$

$$\bar{d}_{io} = d_i + \sum_{k\in N} E^d_{ik}\,(n-1)\,q_{k:i} + C^d_i\ , \tag{3.4c}$$

and

$$\alpha_{ij} = (1-\phi)(\bar{b}_i + \bar{m}_i)q_{i:o} = \alpha_i \tag{3.5a}$$

$$\beta_i = \phi\bar{b}_i + (1-\phi)(\bar{b}_i + \bar{m}_i)q_{i:o} \tag{3.5b}$$

$$\delta_{ij} = \bar{d}_{ij} + (1-\phi)\bar{m}_i\ q_{o:i} \tag{3.5c}$$

One can insert these approximate expressions into the system of differential equations for pair frequencies written with exact pair transition rates; see equation (B1.1). If there is a single phenotype x in the population (resident phenotype), the dynamics of pairs obey the following system of so-called *correlation equations* (Rand 1998):

$$\begin{pmatrix} dp_{ox}/dt \\ dp_{xx}/dt \end{pmatrix} = \begin{pmatrix} \alpha' x\ \ q_{o:o} - (\beta_x + \delta_{xo}) & \delta_{xx} \\ 2\beta_x & -2\delta_{xx} \end{pmatrix} \begin{pmatrix} p_{ox} \\ p_{xx} \end{pmatrix} \tag{3.6}$$

The equilibrium state of the population, fully characterized by $q_{o:x}$ and $q_{o:o}$, may then be found by solving the system $dp_{ox}/dt = 0$ and $dp_{xx}/dt = 0$.

Spatial invasion fitness

We now have the modeling machinery in place to tackle the calculation of invasion fitness, that is, a measure of the population growth rate of a mutant (phenotype y) introduced at low frequency in the resident population where only phenotype x is present. When two strategies x and y are represented in the population, there are six possible types of pairs. A simple bookkeeping procedure is applied to all possible transitions of these pairs, and the rates defined by equations (3.3) are used to construct a system of correlation equations (3.7):

$$\begin{pmatrix} dp_{ox}/dt \\ dp_{xx}/dt \\ dp_{oy}/dt \\ dp_{yx}/dt \\ dp_{yy}/dt \end{pmatrix} = \begin{pmatrix} \alpha' x\ \ q_{o:o} - (\beta_x + \alpha_y + \delta_{xo}) & \delta_{xx} & 0 & \delta_{yx} & 0 \\ 2\beta_x & -2\delta_{xx} & 0 & 0 & 0 \\ 0 & 0 & \alpha' y\ q_{o:o} - (\beta_y + \alpha' y\ q_{y:o} + \delta_{xo}) & \delta_{xy} & \delta_{yy} \\ 0 & 0 & \alpha_x + \alpha' y\ q_{x:o} & -(\delta_{xy} + \delta_{yx}) & 0 \\ 0 & 0 & 2\beta_y & 0 & -2\delta_{yy} \end{pmatrix} \begin{pmatrix} p_{ox} \\ p_{xx} \\ p_{oy} \\ p_{xy} \\ p_{yy} \end{pmatrix}$$

The mutant rate of growth, denoted by $s(y,x)$, can be obtained by summing up the last three equations of system (3.5):

Box 2. A numerical recipe to compute spatial invasion fitness in lattice models

The expression of mutant population growth rate depends on the spatial statistics $q_{o:y}$, $q_{x:y}$, and $q_{y:y}$, which a priori vary over time. Yet the so-called *relaxation property* of the system entails that the statistics $q_{o:y}$, $q_{x:y}$, and $q_{y:y}$ converge very fast to equilibrium values, compared to the slow growth or decline of the system variables p_{oy}, p_{xy}, and p_{yy} (Matsuda *et al.* 1992, our simulations). Therefore, to obtain a measure of spatial invasion fitness, we may write an auxiliary system of differential equations for the variables $q_{o:y}$, $q_{x:y}$, and $q_{y:y}$ only, solve it for equilibrium, and insert the result into equation (4.1).

The numerical derivation of this auxiliary system relies on the initial rarity of the mutant in the resident population. This, by definition, means: $q_{y:o} = 0$. This property allows us to write a closed model for the mutant pair dynamics, using the 3×3 lower-right block **M** of the transition matrix which appears in equation (3.5):

$$\frac{d\vec{p}_y}{dt} = \mathbf{M}(\vec{q}_y)\,\vec{p}_y \text{ with } \vec{p}_y = (p_{oy}, p_{xy}, p_{yy}) \qquad \text{(B2-1)}$$

Using the relations $dp_y/dt = s(y,x)\,p_y$ and $\vec{p}_y = p_y\vec{q}_y$, we can further transform this system into

$$\frac{d\vec{q}_y}{dt} = \left[\mathbf{M}(\vec{q}_y) - s(y,x)\,\mathbf{I}\right]\vec{q}_y \qquad \text{(B2-2)}$$

(**I** is the 3×3 identity matrix.) At equilibrium, $d\vec{q}_y/dt = 0$, and the spatial statistics $\vec{q}_y$ are obtained by solving (numerically, or analytically in the simplest cases) the nonlinear system $M(\vec{q}_y)\vec{q}_y = \lambda\vec{q}_y$, which involves four unknowns ($q_{o:y}$, $q_{x:y}$, $q_{y:y}$, and the corresponding eigenvalue λ) and three equations, along with the constraint $q_{o:y} = 1 - q_{x:y} - q_{y:y}$. Solving for λ at the same time yields the numerical value of the spatial invasion fitness $s(y,x)$ (4.1).

$$\frac{dp_y}{dt} = \frac{dp_{oy}}{dt} + \frac{dp_{xy}}{dt} + \frac{dp_{yy}}{dt} = s(y,x)\,p_y \qquad (3.8)$$

which, after some algebra, simplifies into:

$$s(y,x) = \bar{b}_y q_{o:y} - \bar{d}_y \qquad (3.9)$$

where

$$\bar{b}_y = b_y + (n-1)E^b_{yx}q_{x:y} + (n-1)E^b_{yy}q_{y:y} - C^b_y \qquad (3.10)$$

$$\bar{d}_y = d_y + n E^d_{yx} q_{x:y} + n E^d_{yy} q_{y:y} + C^d_y \tag{3.11}$$

Rearranging terms, we obtain the final expression:

$$\begin{aligned} s(y,x) = &\left[\left(b_y - C^b_y\right) q_{o:y} - d_y - C^d_y\right] \\ &+ \left[(n-1) E^b_{yx} q_{o:y} - n E^d_{yx}\right] q_{x:y} + \left[(n-1) E^b_{yy} q_{o:y} - n E^d_{yy}\right] q_{y:y} \end{aligned} \tag{3.12}$$

This expression bears an interesting relationship to the notion of *direct* or *neighbor-modulated fitness* (Hamilton, 1970; Frank, 1998). Direct fitness is defined by summing the fitness effects on an individual caused by all the phenotypes of neighbors (including the individual itself). Likewise, spatial invasion fitness is obtained by adding the effects on a focal mutant of a resident or mutant neighbor, weighted by the probability that the focal individual is neighbored by a resident or a mutant individual.

Further analysis based on spatial invasion fitness as defined by (3.12) requires that we solve equation (3.7) for $q_{y:y}$, $q_{x:y}$, and $q_{o:y}$. This can be done numerically by following the algorithmic recipe outlined in Box 2, or even analytically in the simplest cases (Matsuda *et al.*, 1992).

4. Application: coadaptation of dispersal and altruism

Empirical work has stressed the importance of spatial structure and spatial processes for the evolution of dispersal (Hanski, *this volume*; Ims and Hjermann, *id.*; Ronce *et al.*, *ibid.*). Coadaptation of other life-history components is also expected to have a decisive influence on the evolution of dispersal, because of physiological and/or genetic correlations (Ronce *et al.*, *this volume*; Roff & Fairbairn, *id.*) or behavioral alternatives (see Lambin *et al.*, *this volume*, for a discussion of the joint adaptation of dispersal, competition, and cooperation). There is an urgent need for theory to incorporate these empirical facts.

The purpose of this section is to take a step forward in that direction. We make use of the framework of lattice population models to investigate the joint evolution of dispersal and social behavior, while accounting explicitly for local interaction and dispersal processes. More specifically, our main objectives are (i) to identify selective pressures acting on these traits, (ii) to make predictions about their relative effects on the direction of evolution, and (iii) to relate them quantitatively to basic individual and interaction traits. The material presented here provides a short review of analyses expounded in Le Galliard (1999), Le Galliard *et al.* (*in prep.*), and Ferrière and Le Galliard (*in prep.*).

Model assumptions

We focus on two adaptive components of the individual's phenotype: dispersal and altruism (Table 2). The former trait is measured by the dispersal rate *m*. The altruistic trait is measured by the total investment in altruism *u* and the amount *u/n* of help an actor individual may distribute over its neighborhood. This amount additively affects any recipient's intrinsic birth rate. Note that this is a simplified description of altruism, because individuals will have a total potential amount *u* to give and will always give the same amount of help per neighbor, whatever the number of receivers. In the biological realm, this would mean the absence of any kind of strategical distribution of altruism.

Table 1. Variables and parameters of the lattice model

N	set of all phenotypes present in the population
z	generic notation for the location of a site in the graph
i, j, k	generic notations for different site states
p_i	frequency of sites in state *i* among all sites (*site frequency*)
p_{ij}	frequency of (i, j) pairs among all pairs of sites (*pair frequency*)
$q_{i:j}$	probability that, next to a site in state *j*, there is a site in state *i* (*aggregation coefficient*)
$q_{i:jk}$	probability that, next to a site in state *j* in a (j,k) pair, there is a site in state *i*
n	number of neighboring sites to any given site (constant)
$n_{k:ij}(z)$	number of sites in state *k* in the neighborhood of a type *i* at site *z*, in a (i, j) pair
ϕ	probability of randomly generating a connection with[or "probability of making a connection at random with", or "random probability of…"] all sites which could potentially be connected to that site ($\phi = 1/n$).
$b_i(z)$	intrinsic per capita birth rate at location *z*
$d_i(z)$	intrinsic per capita death rate at location *z*
$m_i(z)$	intrinsic per capita dispersal rate of type *i* at location *z*
$E_{ij}^b(z)$	additive effect (competition, cooperation) on the per capita birth rate of a type *i* individual located at *z*, induced by interaction with a type *j* individual located in the neighborhood
$E_{ij}^d(z)$	additive effect (competition, cooperation) on the per capita death rate of a type *i* individual located at *z*, induced by interaction with a type *j* individual located in the neighborhood
$C_i^b(z)$	cost of type *i* strategy, decreasing the birth rate of a type *i* individual located at *z*
$C_i^d(z)$	cost of type *i* strategy, decreasing the death rate of a type *i* individual located at *z*

Both traits are costly to the bearer. A linear model for the cost of dispersal *m* is assumed, whereas the cost of altruism scales algebraically with the amount of total investment *u* (Table 2). The total cost is substracted from the intrinsic birth rate. The costs of dispersal and altruism are paid unconditionally, irrespective of the movement actually performed by the individual and the average amount of help actually given to the neighborhood. A representative biological instance would be an organism where both dispersal and altruism imply an initial ontogenetic shift towards a fixed physiological or morphological state that would determine the individual lifetime investment of dispersal and altruism. This state would permanently impact the birth rate. An example of this might be the case of a dispersal structure (O'Riain *et al*., 1996).

Table 2. Specific variables and parameters of the model

b	intrinsic per capita birth rate (b=2.0)
d	intrinsic per capita death rate (d=1.0)
m	intrinsic per capita dispersal rate (*adaptive trait*)
u	intrinsic per capita altruism rate (*adaptive trait*)
κu^{γ}	cost of altruism, decreasing the birth rate
νm	cost of disperal, decreasing the birth rate

Starting from the general model presented in section 3, we make two simplifying assumptions on our way to derive the measure of spatial invasion fitness: the intrinsic birth and death rates are independent of the phenotype, and costs and benefits impact the birth rate only.

Referring to notations introduced in Table 1, this means: $b_i \equiv b$, $d_i \equiv d$, $E_{ij}^d \equiv 0$, $C_i^d \equiv 0$. We use the notation $C(u,m)$ to designate the total cost associated with altruism u and dispersal rate m, $C(u,m) = \kappa u^{\gamma} + \nu m$ (Table 2). Parameters κ and ν measure the sensitivity of the costs of altruism and dispersal. The parameter γ further indicates how the sensitivity of the cost of altruism varies with the degree of altruism. A high value of γ means that the cost of altruism increases slowly with the degree of altruism when the degree of altruism is low, and becomes more sensitive to altruism as the degree of altruism increases.

Adaptive dynamics of dispersal and altruism

Spatial invasion fitness s follows from the general model of equation (3.10) and is given here by

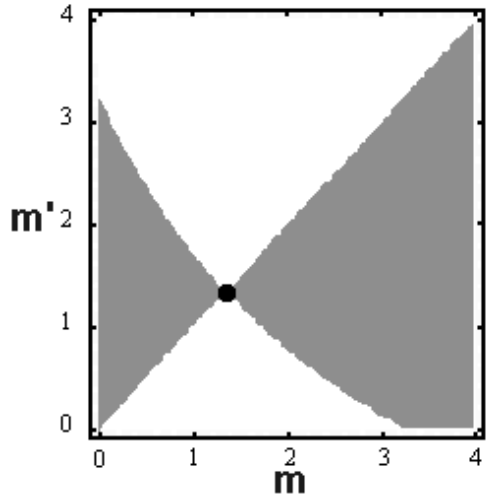
$$s \equiv \left(b + u(1-\phi)\, q_{x:y} + u'(1-\phi)\, q_{y:y} - C(u',m')\right) q_{o:y} - d \tag{4.1}$$

where $x = (u,m)$ denotes the resident phenotype; $y = (u',m')$, the mutant phenotype. The canonical equation (2.5) reads

$$\frac{d}{dt}\begin{pmatrix} u \\ m \end{pmatrix} = \begin{pmatrix} \eta . \frac{\sigma^2}{2} . p_x . \left.\frac{\partial s}{\partial u'}\right|_{u'=u} \\ \eta . \frac{\sigma^2}{2} . p_x . \left.\frac{\partial s}{\partial m'}\right|_{m'=m} \end{pmatrix} \tag{4.2}$$

where η and σ^2, respectively, denote the mutation rate and the mutation step variance, which we assume to be the same for both traits and independent of the current phenotypic mean. Making use of the facts that the resident population is at equilibrium and that the mutant is little different from the resident, a first-order approximation of spatial invasion fitness reads

$$s / q_{o:y} \cong \left[d / q_{o:x}^2 - (1-\phi)u\right]\left(q_{o:y} - q_{o:x}\right) + (1-\phi)\, q_{y:y}\,(u'-u) - \left[C(u',m') - C(u,m)\right] \tag{4.3}$$



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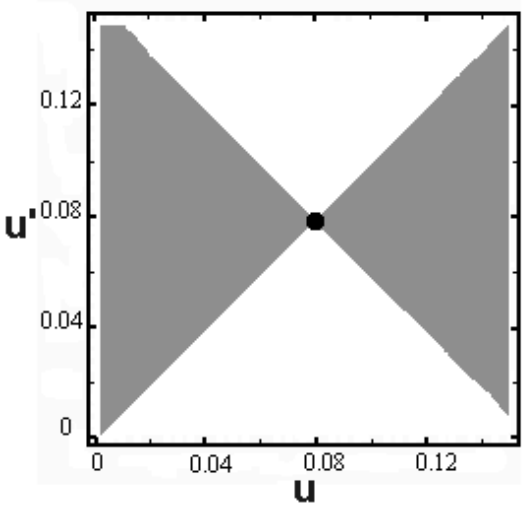


(b)

Figure 3. Pairwise invasibility plots when either the altruism trait or the dispersal trait is fixed. Spatial invasion fitness (see equation [4.1]) is positive in the grey region. (**a**) Evolution of dispersal for fixed altruism ($u = 0.1$). (**b**) Evolution of altruism for fixed dispersal ($m = 0.5$). In both cases, there is a unique evolutionary singularity which is attracting and evolutionarily stable. Parameter values: $n = 4$, $b = 2.0$, $d = 1.0$, $\gamma = 2.0$, $\kappa = 1.0$, $\nu = 0.1$.

(see Ferrière & Le Galliard, *in prep*., for details). This expression clearly identifies three components of selection operating on dispersal and altruism. The first term in the right-hand side of equation (4.3) quantifies the pressure for opening free space in an individual's neighborhood. It is stronger under more crowded conditions (*i.e*., when $q_{o:x}$ is low), and increases with the intrinsic death rate *d*: When mortality is low, there is little selective advantage to be gained from opening space by reducing altruism or increasing dispersal. Also, this pressure is opposed if the resident degree of altruism, *u*, is sufficiently high, since then it pays off to interact with more neighbors, regardless of their altruism phenotype (resident or mutant). The second term in equation (4.3)

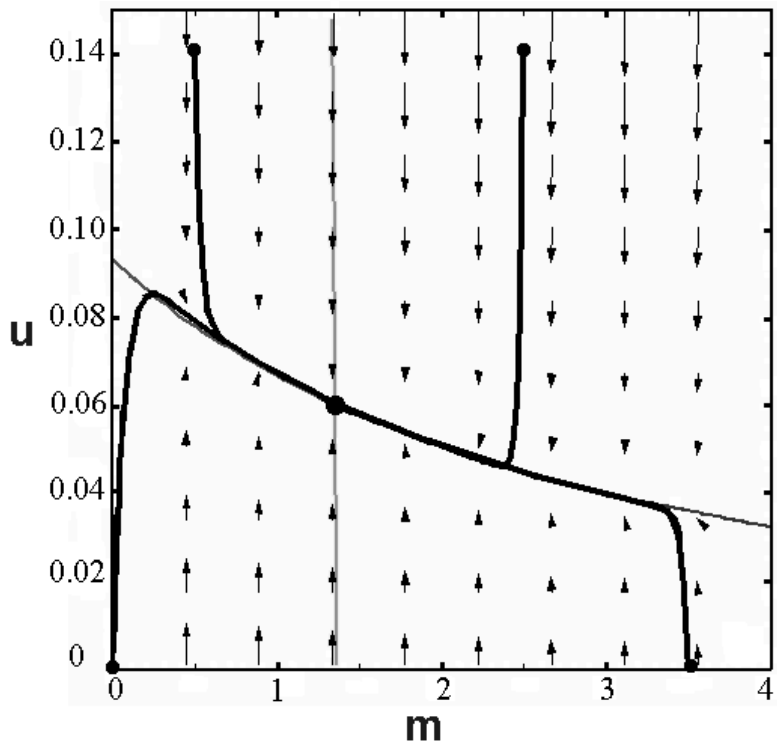
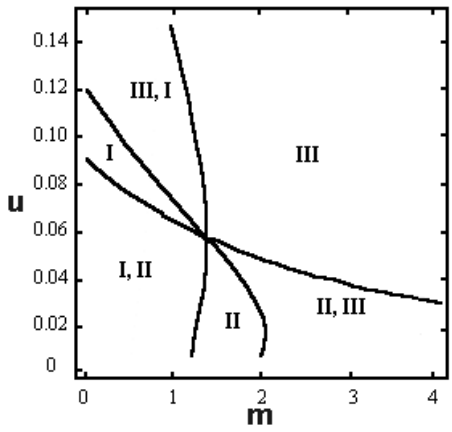


Figure 4. Co-adaptive dynamics of dispersal and altruism. Predictions from the canonical equation (4.2). Thin lines are evolutionary isoclines. The four thick lines are examples of evolutionary trajectories, starting from four different ancestral states. The crossing point of the isoclines gives the singularity, which is attracting and evolutionarily stable. Parameter values: same as in Fig. 3.

expresses the pressure for increased altruism ; it is stronger under more aggregated mutant conditions. The third term measures the pressure for reducing the direct costs of dispersal and altruism. By following the numerical recipe for the calculation of aggregation coefficients and spatial invasion fitness (Box 2), one can obtain explicit analytical expressions for $q_{y:y}$ and $q_{o:y}$. It is thus possible to write each component of selection as a function of individual parameters.

In general, when the evolution of one trait alone is considered, the adaptive dynamics of the trait are monotonous and converge to a point attractor. This attractive point corresponds to a singularity of the adaptive dynamic, that is, a point where the selection derivative vanishes. A mutant appearing around this phenotype value is actually counterselected and cannot invade (Fig. 3). The pattern of stabilizing selection is well explained by the relative effects of conflicting pressures. Focusing on the case of dispersal, we can see that at low dispersal, the predominant selective pressure is induced by local competition for space; reduced aggregation is favored, and this selects for higher dispersal rates. As dispersal increases the intensity of the opposed selective pressure induced by the cost of dispersal also raises. An intermediate equilibrium value is reached at which both pressures exactly compensate each other. Numerical analysis of the dispersal rate at this attractor suggest that its value is mainly sensitive to the parameter ν, which scales the cost of dispersal.



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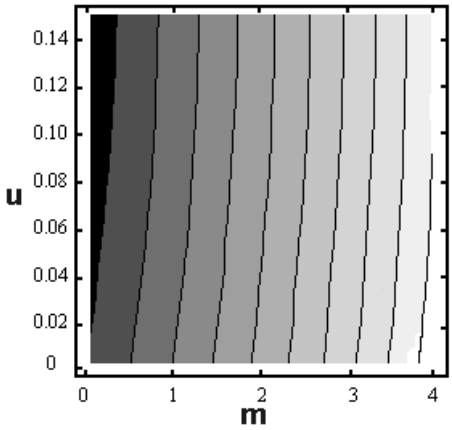


(b)

Figure 5. (**a**) Zero-contour lines of the components of selection along adaptive trajectories. In each of the six delineated regions, positive pressures are indicated. Component I: pressure for reducing local competition for space; component II: pressure for increased altruism under aggregated conditions; component III: pressure for reducing the direct costs of dispersal and altruism. (**b**) Spatial aggregation, shown as a contour plot of the aggregation coefficient $q_{x:x}$ for a pure population of phenotype *x*. Parameter values: same as in Fig. 3.

We now consider the coadaptive dynamics of dispersal and altruism. The selective gradient respective to either trait vanishes along the corresponding isocline (Fig. 4), which is the set of evolutionary singularities obtained for this trait, for each possible value of the other trait. Both isoclines cross at the singularity of the coadaptive dynamics, denoted by (m^*, u^*).

When the cost of altruism is high and very sensitive to a change in the degree of altruism, the singularity is always a stable node (Le Galliard *et al.*, in prep.). The dispersal rate still converges monotonously to the singularity, but explaining the adaptive dynamics in the two-dimensional trait space now requires that we consider how the three selective pressures interplay. This can be done by identifying the sign of each selection component evaluated locally in the direction of adaptation (Fig. 5). For example, one can interpret the four trajectories (1-4) depicted in Fig. 4 in this way: (1) Starting from low dispersal and low altruism, mutants that invest more in altruism and

dispersal are initially favored (selection components I and II are positive); being more altruistic is advantageous because the level of aggregation is high; being slightly more mobile is also beneficial for it yields more free space in one's neighborhood. In a second phase of the dynamics, mutants dispersing more are selected for (selective component I is positive); this reduces spatial aggregation and therefore promotes invasion by less altruistic phenotypes. (2) Initially, dispersal is low and altruism is high. Only the first phase of the adaptive dynamics differs: Here, the adaptive dynamics begin with the reduction of the cost of altruism and the reduction of local competition for space (components III and I are positive). (3) Starting with a high dispersal-low altruism phenotype, selection favors an increase in altruism and a decrease in dispersal: At low altruism, mutants with lower dispersal rates pay a significantly reduced cost (component III is positive), and the benefit of more altruism in a population that develops more aggregation dominates the cost of increased local competition (component II is positive). (4) Finally, when ancestral dispersal and altruism are high, the selective pressure for reduced costs dominates (component III is positive) and drives the system all the way down to the singularity where both traits stabilize.

Revisiting Hamilton's Rule

Hamilton (1964) formulated his famous rule according to which, if an actor expresses a behavior that costs him C offspring and increases by B the number of individuals related to the actor, this behavior is selected for if $B\ r > C$. There has been much debate over the interpretation of the fitness costs C, benefits B, and relatedness r which make Hamilton's rule work, and by which this rule can be generalized for more complex ecological scenarios.

Defining and measuring relatedness in spatially structured populations is a longstanding problem of population genetics (Malécot, 1948; Rousset & Billiard, *manuscript*). The spatial invasion condition provides a natural definition of relatedness as a measure of phenotypic correlation between neighbors (Frank, 1998; van Baalen & Rand, 1998). When altruistic and selfish individuals are identical in their basic demographic rates (b, d, and m), altruists with phenotype $y = (u', m)$ can invade non-altruists with phenotype $x = (0, m)$ if

$$u' \cdot (1 - \phi) \cdot q_{y:y} > \kappa \cdot u'^{\gamma} \tag{4.4}$$

that is, we have recovered a variant of Hamilton's rule in which $B \equiv (1-\phi)u'$, $= \kappa \cdot u'^{\gamma}$, and the coefficient of relatedness r is given by

$$r = q_{y:y} \tag{4.5}$$

As already mentioned, $q_{y:y}$, and therefore r, can be computed from the invasion matrix (Box 2). This coefficient r estimates how much of an altruist's environment consists of other altruists, an interpretation that is consistent with Day & Taylor (1998). The precise interpretation of B, C, and r in Hamilton's rule, however, is dependent on the details of demographic processes operating in the population. For example, van Baalen and Rand (1998) note that if the cost of altruism is incurred as an increased mortality rate instead of a decreased birth rate, for zero dispersal the invasion condition of altruists in a selfish population becomes

$$u'\cdot(1-\phi)\cdot q_{y:y} > (b/d)\cdot\kappa\cdot u'^{\gamma}. \qquad (4.6)$$

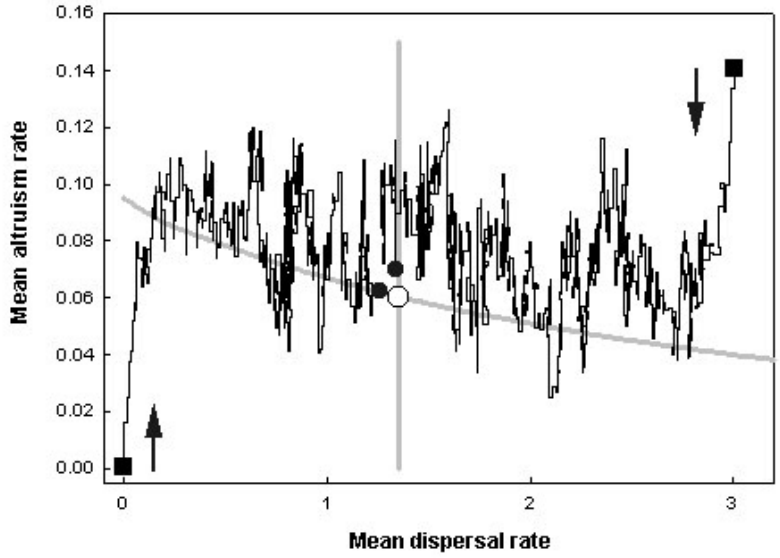
This provides another version of the Hamilton's rule where the cost C is recovered as the cost of altruism corrected for intrinsic birth and death rates. Other variants of the spatial Hamilton's rule, where relatedness similarly depends on local demographic processes, have been established by Ferrière & Michod (1995, 1996) for the invasion of cooperation in a spatial iterated Prisoner's Dilemma.

How kin selection models handle relatedness is usually problematic (Day & Taylor, 1998; Rousset & Billiard, *manuscript*). This is not to suggest that kin selection is not the ultimate cause of the evolution of altruism in viscous populations, as Hamilton originally asserted it is (1964), but that measuring inclusive fitness as defined in kin selection models may not correctly predict the evolutionary dynamics of social traits when selection is density-dependent. Using spatial invasion fitness, Hamilton's principle is recovered as an emergent property of the model. This backs up Nunney's (1985) statement that group selection acts when there is positive preferential association of common phenotypes, but that kin selection is the only form of group selection that is able to maintain altruism.

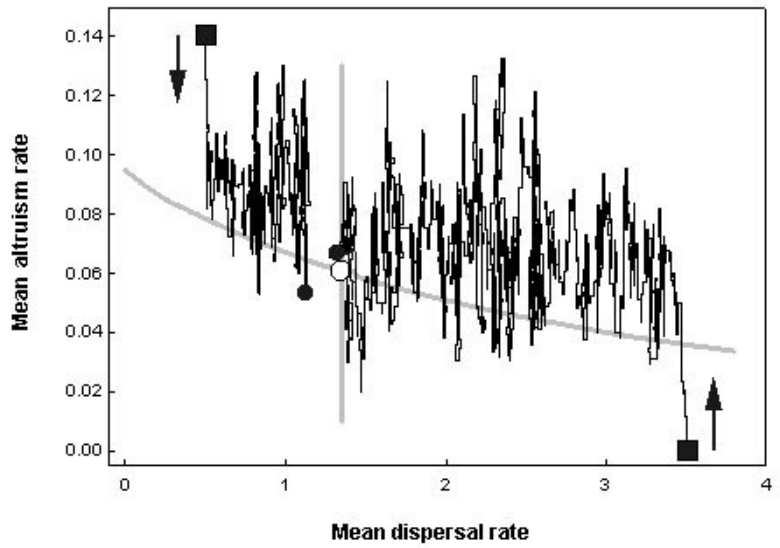
Does spatial invasion fitness rightly predict evolutionary dynamics?

Although our coevolutionary model of dispersal and altruism incorporates salient features of the ecological and evolutionary processes (including density-dependence, demographic stochasticity, and evolutionary feedback), it remains underpinned by several critical simplifications. We assume an infinite lattice size, and describe the dynamics of local densities by making use of the standard pair approximation (Morris, 1997). The derivation of the fitness measure relies on the small frequency of mutants when they appear and on the relaxation approximation that they instantaneously build up a characteristic invasion structure that may serve as a vehicle for their potential spread (Dieckmann & Law, 2000). Furthermore, the deterministic description of the adaptive dynamics gives an approximation for the mean path of the stochastic mutation selection process (Dieckmann & Law, 1996), which itself already entails averaging over an infinite number of realizations.

Notwithstanding all this, the properties of stochastic simulations are remarkably well captured by the deterministic predictions (Fig. 6; see Le Galliard *et al.*, *in prep.*, for a more thorough comparison). The positions of the isoclines and the attracting singularity (m^*, u^*) remain nearly unchanged. Overall trends of stochastic trajectories are correctly predicted by the deterministic model. Wilder fluctuations in trait values, involving the repeated rise and fall of altruism, are observed nearer to the singularity, as the selection gradient tends to weaken there. In our case, these complex regimes in the degree of altruism, which have received some attention elsewhere (Doebeli & Knowlton, 1999), are best explained by genetic drift in regions of low selection pressure across the phenotypic space.



(a)



(b)

Figure 6. Mean trajectory of ten individual-based simulations of dispersal and altruism evolutionary dynamics. Thick grey lines are isoclines predicted by the canonical equation (4.2) (see Fig. 4). Black squares indicate initial states. (**a**) Simulations of two trajectories starting at $(m,u)=(0,0)$ and $(m,u)=(3,0.14)$, respectively. (**b**) Simulations of two trajectories starting at $(m,u)=(0.5,0.14)$ and $(m,u)=(3.5,\ 0.0)$, respectively. The stochastic trajectories, although rather jerky near the convergence state at the isoclines intercept (open circles), hit rather close to it (black circles) after following closely the deterministic path predicted by spatial invasion fitness (see Fig. 4). For both traits, the mutation rate is 10^{-2} and the mutation variance is 10^{-2}. Time at the end of simulations: 500,000 time units. Lattice size: 900 sites.

5. Concluding Remarks

Defining invasion fitness for spatial ecologies is no trivial matter. Starting from demographic and behavioral processes operating at the individual level and locally between close neighbors, the invasion exponent of a simple system of correlation equations for the dynamics of a mutant population provides a tractable solution to this problem. The notion of spatial invasion fitness allows one to *derive*, rather than postulate, an explicit relationship between distinct components of selection on the one hand, and the characteristics of the individuals and their interactions on the other. Numerical simulations of individual-based models confirm that the resulting spatial invasion fitness correctly predicts the dynamics of the stochastic mutation-selection process. On the empirical side, Rainey and Travisano's (1998) experiments on the evolution of polymorphism in bacteria have shown that invasion fitness measured in spatially heterogeneous populations successfully predicts the maintenance of morph diversity. In contrast, the destruction of local structures developed in the course of population growth alters the phenotypes' invasion fitnesses and modifies the eventual phenotypic composition of the population.

The mathematical derivation of spatial invasion fitness proceeds by averaging over space the transition rates of pairs. This amounts to looking at the local structure of the mutant population as homogeneously replicated across the whole (infinite) lattice. The non-homogeneous distribution of the pairs containing mutants, induced by the finite size of the mutant population and the non-typical clustering pattern that may develop at the earliest stage of invasion, may also require us to incorporate correction terms to our measure of spatial invasion fitness. There may be an interesting parallel to be drawn with the theory of evolutionary games in continuous space. In this context, the initial clustering of mutants entails that fitness should be defined not from space averages of individual traits, but as the speed at which the front of a mutant cluster moves forward and propagates mutants through space (Hutson & Vickers, 1992; Ferrière & Michod, 1995, 1996; Ellner *et al.* 1998).

We have used the notion of spatial invasion fitness to model the joint adaptive dynamics of dispersal and altruism. Even without further corrections for more subtle spatial effects, spatial invasion fitness appears to give very consistent predictions on how these two behavioral traits coevolve. The analysis of this particular model underlines three important and general achievements of adaptive dynamics based on the notion of spatial invasion fitness. First, it unravels the interplay of the ecological (spatial) dynamics of a population and the evolutionary dynamics of the individual traits. Spatial self-structuring shapes the selective pressures, which in return may alter the aggregation pattern. Here we have seen that a high degree of spatial aggregation is not a prerequisite for, but rather a consequence of, the joint evolution of altruism and dispersal. Second, this analysis underlines important transient effects. A state of high dispersal or high altruism may be maintained transitorily, up to the point where the direction of selection changes or even reverts. In general, this means that variations, under the same environmental conditions and for the same species, of adaptive traits may be explained by different ancestral states and by the observation of populations at different points in time in their evolutionary history. Finally, this approach allows us to separate out distinct components of the selective regime and to express these components in terms of individual traits and characteristics of the population aggregation structure. In practice, there is potential here to predict how the selective

pressures should equilibrate to produce patterns observed empirically, and how dispersal-related traits may respond to the experimental manipulation of each component of the selective regime.

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