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THE ADAPTIVE DYNAMICS OF NICHE CONSTRUCTING TRAITS IN SPATIALLY SUBDIVIDED POPULATIONS: EVOLVING POSTHUMOUS EXTENDED PHENOTYPES

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Niche construction, by which organisms modify the environment in which they live, has been proposed to affect the evolution of many phenotypic traits. But what about the evolution of a niche constructing trait itself, whose expression changes the pattern of natural selection to which the trait is exposed in subsequent generations? This article provides an inclusive fitness analysis of selection on niche constructing phenotypes, which can affect their environment from local to global scales in arbitrarily spatially subdivided populations. The model shows that phenotypic effects of genes extending far beyond the life span of the actor can be affected by natural selection, provided they modify the fitness of those individuals living in the future that are likely to have inherited the niche construction lineage of the actor. Present benefits of behaviors are thus traded off against future indirect costs. The future costs will generally result from a complicated interplay of phenotypic effects, population demography and environmental dynamics. To illustrate these points, I derive the adaptive dynamics of a trait involved in the consumption of an abiotic resource, where resource abundance in future generations feeds back to the evolutionary dynamics of the trait.

KEY WORDS: Consumer-resource system, extended phenotype, inclusive fitness, niche construction, social evolution, spatial population subdivision, spite.

Adaptations, by which organisms appear to fit their particular environment in form and function, have captured the imagination of humans for millennia (Orzack and Sober 2001; Vincent and Brown 2005). But organisms not only adapt to the demands of their environment, but they also modify it and construct it from the elements of the world in which they reside (Lewontin 2000). Although adaptations are often regarded as the phenotypic variants reaching the highest fitness among a set of alternatives in a given environment (Reeve and Sherman 1993), it has also been stressed that adaptations, by modifying the environment, participate in a feedback between the selective pressures and the adaptations themselves (Odling-Smee et al. 1996, 2003). The view that organisms construct their niches leads to a picture in which organisms and their environment are tightly intertwined, both depending on the histories of the organism and the environment (Lewontin 2000). A classical example of this process is the case of the earthworms described by Darwin (1883), which through their burrowing change the structure and chemistry of the soil, with the consequence that some earthworm adaptations, such as epidermis structure or the amount of mucus secreted are likely to have coevolved with earthworm niche construction.

The example of the earthworms illustrates the central principle of niche construction theory. Namely, that a niche-constructing gene, whose phenotypic effect modifies the environment, may generate a selective pressure on other genes (loci) in later generations (Odling-Smee et al. 1996, 2003; Laland et al. 2001). But what about the selective pressure on the niche constructing gene itself? To what extent can the phenotypic effects changing the

fitness of individuals living in future generations be subject to natural selection when the actor reproduces now? This question has received little attention so far. However, organisms, merely by existing, use resources and produce wastes that change the structure of their local environments, which may directly modify the reproductive rate of individuals living in later generations. A simple example is given by prey–predator dynamics (Begon et al. 1996; Murdoch et al. 2003), where the amount of preys consumed in one generation directly feeds back on the growth rate of predators in the next generation. Hence, the phenotypic effects of genes underpinning prey consumption directly influence the fitness of these genes in adjacent and/or later generations.

The situation in which the expression of a phenotype affects future generation through carry-over effects across generations has been termed "ecological inheritance" (Odling-Smee et al. 2003). Ecological inheritance occurs whenever individuals encounter features of their environment that have been altered and/or modified by other individuals living in previous generations. Examples of such alterations may be found in the extensive variety of organisms that construct nests, burrow systems, paths, dams, nurseries, mounds, biofilms, and return detritus to their habitat. For instance, monogynous and polygynous social insects exhibit temporal succession of reproductive individuals within the same colony (Gadagkar et al. 1993; Peeters 1993; Gotwald 1995; Evans 1996; André et al. 2001), thus reproducing in nests and mounds assembled by previous generations. But structures persisting beyond the life span of the organism inhabiting them are also numerous in birds (Hansell 1984; Skutch 1987), and are well exemplified by the complex burrow systems of black-tailed prairie dogs extending over several kilometers (Hoogland 1995) or beaver dams inherited for up to hundreds of years (Odling-Smee et al. 2003).

Lasting environmental modifications can be considered as part of an individual's extended phenotype (Dawkins 1982), because they affect the fitness, not only of the individual producing them, but also of individuals living in future generations. The answer to the question of whether such intertemporal phenotypic effects are subject to natural selection is clear for the case in which evolution takes place in a panmictic population. Here, any genetic variant causing its bearer to leave more offspring in the next generation than an alternative variant will be selected to increase in frequency. This is true whatever the phenotypic effects of the variant on the demographic and the natural environment of subsequent generations (Rousset and Ronce 2004), and a beneficial variant can go to fixation even if it ultimately causes the population to go extinct (i.e., "evolutionary suicide," see Matsuda and Abrams 1994; Gyllenberg and Parvinen 2001; Parvinen 2005). Accordingly, a gene coding for the construction of an artifact (e.g., nest, burrow), will be selected for only if it increases the fitness of its bearer. However, spatial population subdivision changes the patterns of selection on traits modifying the demographic and the natural environment (Rousset and Ronce 2004). Indeed, limited dispersal results in gene lineages associated in space so that individuals bearing identical genes tend to interact together, which results in a kin selection pressure on evolving traits. But limited dispersal results in gene lineages associated not only in space but also in time (Epperson 1999, 2003). Hence, an individual expressing a niche construction trait will presumably change the fitness of whole sets of relatives living in future generations. This will lead to a kin selection pressure affecting the evolution of such traits. It has indeed been observed that spatial population subdivision may increase selection on traits resulting in ecological inheritance, such as the construction of a nest or the provision of a public good, whenever they are not completely erased or depleted from one generation to the next (Lehmann 2006; Silver and Di Paolo 2006; Wakano 2007).

The aim of this article is to analyze the selective pressure on a niche construction phenotype in an arbitrary spatially subdivided population. By building on previous results from inclusive fitness theory (Rousset and Billiard 2000; Rousset 2003; Lehmann 2006), I present a game theoretic model for selection on niche construction phenotypes. This model allows to compute both candidate (potential) evolutionary stable niche construction strategies and their associated stable ecological states (e.g., Vincent and Brown 2005). In this game theoretic framework, the phenotype expressed by an individual can have any positive (or negative) effect on the fitness of all other individuals, living in present and future generations, of the population. Hence, the niche construction traits can affect the environment to which the phenotype is exposed in subsequent generations, which generates a feedback on its own dynamics. This defining characteristic of the model allows it to capture, in particular, the selective pressure on traits resulting in inceptive perturbations, defined as those processes in which an organism initiate a change in their selective environment by physically modifying its surroundings (e.g., construction of a nest or a burrow, consumption of a resource, emission of a detritus, Odling-Smee et al. 2003, table 2.1). By allowing for ecological inheritance, the model relaxes the assumption that individuals can only affect the fitness of their contemporaries, which underlies most evolutionary modeling in subdivided populations, the so-called "spatial," "group selection" or "evolutionary graph theory" models (e.g., Eshel 1972; Hamilton 1975; Aoki 1982; Taylor 1992b; van Baalen and Rand 1998; Irwin and Taylor 2001; Perrin and Lehmann 2001; Le Galliard et al. 2003; Ohtsuki et al. 2006).

The results of the model show that a niche construction trait can evolve when the fitness cost to the actor is offset by the fitness benefit of the trait to all individuals living in future generations, each weighted by the probability that it has inherited the niche construction gene of the actor. Hence, present direct costs are

traded off against future indirect benefits (or vice versa). The long-term fitness costs and/or benefits are determined by the sometimes complicated interplay of phenotypic effects, population demography, and environmental dynamics. To illustrate this point, I analyze below the adaptive dynamics of a trait involved in the consumption of an abiotic resource, whose abundance in future generations feeds back on the evolutionary dynamics of the trait.

Model

LIFE CYCLE

Suppose the population occupies a homogeneous finite discrete space: a circular lattice in one dimension and a torus in two dimensions (e.g., Maruyama 1970; Malécot 1975; Nagylaki 1983; Taylor 1992b; Epperson 1999; Gandon and Rousset 1999; Rousset and Billiard 2000). Each of the n_d nodes of the lattice consists of both a deme with N haploid individuals of a focal species and an environment (symbols are summarized in Table 1). The environment could for instance be a biotic or an abiotic resource, an artifact (e.g., nest, burrow), or a waste product. Individuals reproduce by using and/or affecting their local environment and the environments at different locations in the habitat. The environment at each lattice point is thus modified by the behavior of the individuals of the population. Consequently, the state of the environment at a given point in time and position in space is likely to depend on the phenotypes of the individuals in the population over a number of generations. For simplicity, the environment is assumed to take continuous values and to be of the same type (same variable) at each lattice point. For tractability, the dynamics of the environment is assumed to send it into a stable fixed point.

The sequence of life cycle events of the focal species occurs in the following order. (1) Each adult individual of a focal generation produces a very large number of juveniles, the exact number depending on its own genotype, on the genotypes of its neighbors, and eventually on the genotype of all individuals living in previous generation through their effect on the environmental state of the focal generation. After reproduction, all adults die. (2) Each juvenile disperses independently of each other with probability $m_{\mathbf{k}}$ to deme $\mathbf{k}(\sum_{\mathbf{k}} m_{\mathbf{k}} = 1)$, where $\mathbf{k} \equiv (x, y)$ is the coordinate (single or pair) of a deme relative to the natal deme of a disperser, so that $\mathbf{k} = \mathbf{0} \equiv (0, 0)$ stands for the natal deme. The dispersal

Table 1. List of symbols

Symbol	Definition
N	Deme size
n_{d}	Number of demes
$m_{\mathbf{k}}$	Probability that a juvenile disperses to deme **k** relative to its natal deme
σ^2	Variance of the axial distance of dispersal of an individual. For an isotropic dispersal model it is obtained by measuring dispersal along one dimension only ($\sigma^2 = \sum_k k^2 m_k$, see Rousset 1997)
$z_\bullet$	Phenotype of a focal individual living in a focal deme in a focal generation
$z_{\mathbf{k},t}$	Average phenotype of an individual living in deme **k** relative to the focal deme at t generations prior to the focal generation
$\mathbf{z}$	Vector collecting the phenotypes of all actors in the population in present and past generations. It is given by $\mathbf{z} \equiv (\mathbf{z}_0, \ldots, \mathbf{z}_t, \ldots)$, where the elements are indexed over time and given by $\mathbf{z}_t \equiv (z_{\mathbf{0},t}, \ldots, z_{\mathbf{k},t}, \ldots)$
$\mathbf{z}_{\mathbf{k}}$	Vector with elements $\mathbf{z}_{\mathbf{k},t}$ indexed over time and obtained by circular permutation of the elements of $\mathbf{z}_t$ with first element $z_{\mathbf{k},t}$ [e.g., for a one dimensional lattice $\mathbf{z}_{1,t} = (z_{1,t}, z_{2,t} \ldots, z_{0,t})$]
$Q_{\mathbf{k},t}$	Probability that a gene sampled in a focal individual is identical with a homologous gene sampled in an individual living in deme **k** relative to the focal deme at t generations prior to the focal generation
w	Fitness of a focal individual defined as its expected number of offspring reaching adulthood
$b(z_\bullet, \mathbf{z})$	Fecundity of a focal individual defined as its expected number of offspring produced before density-dependent competition
$b(z_{\mathbf{k},0}, \mathbf{z}_{\mathbf{k}})$	Average fecundity of an individual living in the focal generation at distance **k** from the focal deme
s	Effect of a focal individual on its fecundity
$s_{\mathbf{k},t}$	Effect of a focal individual on the fecundity of the whole set of individuals living in deme **k** relative to the focal deme at t generations posterior to the focal generation
$P_{\mathbf{k},t}$	Probability that an individual, residing in deme **k** relative to the focal deme at t generations posterior to the focal generation, has inherited a gene from an individual breeding in the focal deme
ω^2	Home range of the focal species
a	Attack rate of the focal species on the resource
r	Natural rate of replenishment of the resource
μ	Natural rate of depletion of the resource
α	Effect (positive or negative) of resource consumption on the replenishment rate r

distribution is assumed to be symmetric and identical for all demes (i.e., isotropic dispersal so that the dispersal probabilities are the same to (x,y), (-x,y), (x,-y), and (-x,-y)). (3) Density-dependent competition occurs and exactly N juveniles reach adulthood in each deme.

SPATIAL AND TEMPORAL HOMOGENEITY

To investigate the effect of natural selection on niche construction under the life cycle just described, I introduce a one locus niche constructing trait z. The evolution of this trait is assumed to follow a gradual, step-by-step transformation caused by the successive invasion of mutant alleles having different phenotypic effects than resident alleles fixed in the population. Under this evolutionary scheme, the phenotype of individuals bearing a mutant allele (say A) can be written as $z_A = z_a + \delta$, where z_a is the phenotype of individuals bearing a resident allele (a) and δ is the phenotypic deviation caused by the mutation. By successive allele replacement, the population may eventually converge to some candidate evolutionary stable state $z_a = z^\star$ (i.e., singular point), which is either immune to any deviation δ of the phenotype of the resident or results in a branching point (e.g., Eshel 1996; Geritz et al. 1997, 1998; Ajar 2003; Rousset 2004; Vincent and Brown 2005).

To evaluate the adaptive dynamic of the trait z, we need a fitness function that allows us to establish the strength of selection on a mutant allele. The direct fitness w of an adult individual is defined here as its expected number of offspring that reach adulthood (expected number of recruited offspring, e.g., Hamilton 1964; Rousset and Billiard 2000; Rousset 2004). Phenotypic effects on fitness are assumed to be spatially and temporally homogeneous. Spatial homogeneity means that the fitness of each individual in the population can be expressed as a function of its own phenotype, of the phenotype of the other individuals in its deme, of the phenotypes of individuals one step further on the lattice, of the phenotypes of individuals two step further on the lattice and so on. Temporal homogeneity means that the fitness of each individual can be expressed as a function of the phenotypes of the individuals of its generation, of the phenotype of individuals one time step further in the past, of the phenotype of individuals two time steps further in the past and so on.

With the assumptions of spatial and temporal homogeneity, the absolute position of individuals in both space and time has no specific effect on fitness, and the direct fitness of a focal individual bearing a mutant allele can be expressed as

$$w \equiv w(z_\bullet, \mathbf{E}(\mathbf{z})), \tag{1}$$

where $z_\bullet \equiv z_A$ is the phenotype of the focal individual, and $\mathbf{z} \equiv (\mathbf{z}_0, \mathbf{z}_1, \ldots, \mathbf{z}_t, \ldots)$ is a vector consisting itself of vectors indexed over time $\mathbf{z}_t \equiv (z_{\mathbf{0},t}, z_{\mathbf{1},t}, \ldots, z_{\mathbf{k},t}, \ldots)$, where $z_{\mathbf{k},t}$ is the average phenotype of individuals living in deme $\mathbf{k}$ (relative to the focal deme) at t generations prior to the focal generation. Hence, $t = 0$ stands for the focal generation and the vector $\mathbf{z}$ collects the phenotypes of all individuals living in the focal and in previous generations. All these individuals might affect the fitness of the focal individual through their impact on the environment at each lattice point. The environmental states at each lattice point relative to the focal deme are collected in the vector

$$\mathbf{E}(\mathbf{z}) \equiv (\mathcal{E}(\mathbf{z}), \ldots, \mathcal{E}(\mathbf{z}_\mathbf{k}), \ldots), \tag{2}$$

where the function $\mathcal{E}$ determines the environment in a deme as a function of the phenotypes of all individuals living in the population in present and past generations. Different values of the arguments of the function will give the environment in the different demes such that $\mathcal{E}(\mathbf{z})$ stands for the environment in the focal deme, whereas $\mathcal{E}(\mathbf{z}_\mathbf{k})$ is the environment in deme $\mathbf{k}$, where $\mathbf{z}_\mathbf{k} \equiv (\mathbf{z}_{\mathbf{k},0}, z_{\mathbf{k},1} \ldots, \mathbf{z}_{\mathbf{k},t}, \ldots)$ and $\mathbf{z}_{\mathbf{k},t}$ is a vector obtained by circular permutation of the elements of $\mathbf{z}_t$ with first element $z_{\mathbf{k},t}$ [e.g., for a one dimensional lattice $\mathbf{z}_{1,t} = (z_{1,t}, z_{2,t}, \ldots, z_{0,t})$, $\mathbf{z}_{2,t} = (z_{2,t}, z_{3,t}, \ldots, z_{0,t}, z_{1,t})$, see example below]. This notation emphasizes that the environments in different demes as "viewed" from the focal deme differ only to the extent that the phenotypic values of the individuals affecting them may differ, and this difference is only accounted for by the spatial separation between demes, a direct consequence of the assumptions of spatial and temporal homogeneity. The model described here can be interpreted as the extension with extended phenotypic effects in time of the seminal inclusive fitness models for geographically subdivided populations (Taylor 1992b; Gandon and Rousset 1999; Rousset and Billiard 2000; Rousset 2006).

INCLUSIVE FITNESS EFFECT

A convergence measure of stability of the trait z can be obtained by evaluating the change in the probability of fixation of a single mutant allele introduced in a population fixed for the resident allele (Rousset and Billiard 2000; Rousset 2004; Lessard 2005). The effect on its probability of fixation of a single mutant allele expressing a small phenotypic deviation δ (weak selection) can be written as

$$\phi = \lim_{\mu \to 0} \frac{S}{1 - Q_{\mathbf{0},0}}, \tag{3}$$

where μ is the mutation rate from one allele to a new allele (i.e, infinite allele model), $Q_{\mathbf{0},0}$ is the stationary probability of identity between a pair of homologous genes sampled from two individuals chosen at random without replacement from the same deme, and S is Hamilton's inclusive fitness effect measuring the direction of selection on the mutant allele (e.g., Rousset and Billiard 2000; Rousset 2004, 2006). The inclusive fitness effect of the niche construction trait z will be evaluated by the direct fitness method (Taylor and Frank 1996; Frank 1998; Rousset and Billiard 2000), as

$$S = \frac{\partial w}{\partial z_\bullet} + \sum_{t=0}^{\infty} \sum_{\mathbf{k}} \frac{\partial w}{\partial z_{\mathbf{k},t}} Q_{\mathbf{k},t}, \tag{4}$$

which is derived in Lehmann (2006, eq. A.11). In this equation, $Q_{\mathbf{k},t}$ denotes the stationary probability that a gene sampled at the niche construction locus in a focal individual is identical with a homologous gene sampled in an individual chosen at random from deme $\mathbf{k}$ at t generations prior to the focal generation. Because S is computed only from first-order phenotypic effects on fitness (weak selection), the probabilities of identity (the $Q_{\mathbf{k},t}$'s) are evaluated under a neutral model only ($\delta = 0$). This is carried out in the Appendix (eqs. A27–A32) by following the approach to space-time probabilities of identity by descent pioneered by Epperson (1999, 2003).

There are two equally valid ways to interpret the partial derivatives of the fitness function in equation (4), which are all evaluated at the phenotypic value of the resident allele (i.e., monomorphic population: $z_\bullet = \ldots = z_{\mathbf{k},t} = \ldots = z$). First, under the "neighbor-modulated" interpretation (Frank 1998; Rousset 2004; Grafen 2006), $\partial w/\partial z_{\mathbf{k},t}$ measures the effect of the whole set of individuals living in deme $\mathbf{k}$ at t generations prior to the focal generation on the fitness of the focal individual bearing the mutant niche construction allele. Hence, the focal individual is envisioned as the recipient of the mutant allele expressed by other individuals in the population with a probability given by the extent to which they also carry the mutant niche construction allele (the $Q_{\mathbf{k},t}$'s). This "neighbor-modulated" interpretation follows directly and naturally from the definition of the fitness function w. Second, under the "inclusive fitness" interpretation (Frank 1998; Rousset 2004; Grafen 2006), $\partial w/\partial z_{\mathbf{k},t}$ measures the effect of the focal individual on the fitness of the whole set of individuals living in deme $\mathbf{k}$ at t generations posterior to the focal generation. The focal individual is now seen as the actor expressing the mutant niche construction allele and affecting the fitness of other individuals in the population, who carry the same allele as the focal individual according to the probabilities of identity (the $Q_{\mathbf{k},t}$'s). The two interpretations of the partial derivatives offer here two different perspectives of the intertemporal fitness effects resulting from the expression of niche construction ($\partial w/\partial z_{\mathbf{k},t}$ for $t > 0$). The "neighbor-modulated" approach emphasizes past history whereas the "inclusive fitness" approach emphasizes the future. Under neither of these approaches need the probabilities of identity be known to the individuals performing the actions because the niche construction trait is assumed to be expressed unconditionally with respect to the genotype of the recipients (i.e. no discrimination or nepotism), and the probabilities of identity are ultimately determined by the demographic regime of the population.

Even if the niche construction behavior seem at first glance to result in changes of gene frequencies occurring at different time epochs when seen from the "point of view" of a focal individual under the "neighbor-modulated" and the "inclusive fitness" approaches (past and present vs. present and future), the change of gene frequencies under these two perspectives is in fact strictly the same. Under both approaches, it is always the actor who expresses in terms of decreased reproduction the direct fitness costs of the niche construction behavior. This action results in a decrease in the frequency of the mutant allele in the generation of the actor, if everything else is held constant. Similarly, it is always the recipient who expresses in terms of increased reproduction the indirect benefits of the niche construction behavior, which is then likely to increase the frequency of the mutant allele in the generation of the recipient, if everything else is held constant. It is the total change in gene frequency that is taken into account by the gradient of selection S, which is obtained by integrating all the increments and decrements of gene frequencies resulting from the correlated expression of the mutant allele by individuals in the population in each generation, since its first appearance until its eventual fixation or loss from the population (Rousset 2003, 2004). Hence, a positive effect of the mutant allele on aggregate gene frequency change results in an increase of the probability of fixation of the mutant allele, that is, a positive inclusive fitness effect ($S > 0$). This means that, on average, an individual bearing the mutant allele has a higher individual fitness than an individual bearing the resident allele.

It follows that a candidate evolutionary stable strategy $z^\star$ (ESS) and its associated candidate ecological stable state $\mathcal{E}(z^\star)$ can be found by solving $S = 0$ for z. Further, evaluation of $dS/dz|_{z=z^\star}$ allows us to determine whether the trait is convergence stable (e.g., Eshel 1996; Geritz et al. 1998; Rousset 2004), that is, whether a population near to a candidate ESS will converge to it by selection. However, the present framework does not allow us to establish whether a niche construction trait is continuously stable, that is, whether it is an ESS or a branching point. Evaluating continuous stability in a spatial setting requires ascertaining the effect of selection on the probabilities of identity $Q_{\mathbf{k},t}$ (Ajar 2003).

FITNESS FUNCTION

The direct fitness w of a focal individual is obtained as the sum of its expected number of recruited offspring in the focal deme and on those reaching adulthood in other demes by dispersing. Call $b(z_\bullet, \mathbf{z})$ the average fecundity of the focal individual, which may depend in a complicated way on the vector of environment states $\mathbf{E}(\mathbf{z})$. A fraction $m_\mathbf{i}\, b(z_\bullet, \mathbf{z})$ of the focal individual's offspring enter in competition in deme $\mathbf{i}$ with a fraction $\sum_\mathbf{j} m_{\mathbf{i}-\mathbf{j}} b(z_{\mathbf{j},0}, \mathbf{z}_\mathbf{j})$ of the total number of offspring produced in the population, where $b(z_{\mathbf{j},0}, \mathbf{z}_\mathbf{j})$ is the average fecundity of individuals in deme $\mathbf{j}$. Taking into account all demes to which the focal individual's offspring eventually disperse, the direct fitness of the focal individual is given by

$$w(z_\bullet, \mathbf{E}(\mathbf{z})) = \sum_{\mathbf{i}} m_{\mathbf{i}} \frac{b(z_\bullet, \mathbf{z})}{\sum_{\mathbf{j}} m_{\mathbf{i}-\mathbf{j}} b(z_{\mathbf{j},0}, \mathbf{z}_{\mathbf{j}})}. \quad (5)$$

Under weak selection and additive gene action, effects of actors on fitness bear an additive structure (Rousset 2004; Grafen 2006), and so do effects of actors on the fertility b of the individuals in the population. With these assumptions, it is shown in the Appendix (see eqs. A1–A7), that without loss of generality, the direct fitness of the focal individual can be written as

$$w(y_\bullet, \mathbf{E}(\mathbf{y})) = \sum_{\mathbf{i}} m_{\mathbf{i}} \frac{1 + s y_\bullet + \sum_{t=0}^{\infty} \sum_{\mathbf{k}} s_{\mathbf{k},t} y_{\mathbf{k},t}}{\sum_{\mathbf{j}} m_{\mathbf{i}-\mathbf{j}} \left(1 + s y^R_{\mathbf{j},0} + \sum_{t=0}^{\infty} \sum_{\mathbf{k}} s_{\mathbf{k},t} y^R_{\mathbf{j}-\mathbf{k},t}\right)}, \quad (6)$$

where

$$s \equiv \frac{1}{b(z,z)} \frac{\partial b(z_\bullet, \mathbf{z})}{\partial z_\bullet}\bigg|_{z_\bullet = \ldots, z_{k,t}, \ldots = z} \quad (7)$$

is the effect of the focal individual on its fecundity relative to the average fecundity $b(z, z)$ of an individual carrying the resident allele in a population monomorphic for the resident, and $y_\bullet \equiv \delta$ is the phenotypic deviation of the focal individual relative to an individual carrying the resident allele. Furthermore,

$$s_{\mathbf{k},t} \equiv \frac{1}{b(z,z)} \frac{\partial b(z_\bullet, \mathbf{z})}{\partial z_{\mathbf{k},t}}\bigg|_{z_\bullet = \ldots, z_{k,t}, \ldots = z} \quad (8)$$

is the effect of the whole set of individuals living in deme $\mathbf{k}$ at t generations *prior* to the focal generation on the relative fecundity of the focal individual, $y^R{}_{\mathbf{k},t}$ is the average phenotype of an individual residing in that deme, relative to an individual carrying the resident allele, and

$$y^R_{\mathbf{k},t} = y_{\mathbf{k},t} \text{ except that } y^R_{\mathbf{0},0} = \frac{1}{N} y_\bullet + \left(\frac{N-1}{N}\right) y_{\mathbf{0},0}, \quad (9)$$

where $y_{\mathbf{k},t}$ is the average phenotype of an individual but excluding the focal individual himself from the average.

Equation 6 expresses the fitness of the focal individual in terms of the effects (positive or negative) of the various categories of actors (labeled by $\mathbf{k}$ and t) on its own relative fecundity, and on the number of offspring produced by all its competitors. This functional form is convenient for investigating the generic repercussions of the spatial structure of the population on an arbitrary niche construction trait's selective pressure. The effects on relative fecundity appearing in equation (6) are specific to each niche construction trait and can also be interpreted in the "inclusive-fitness" way. Under this interpretation, $s_{\mathbf{k},t}$ represents the effect of the focal individual on the relative fecundity of the whole set of individuals living in deme $\mathbf{k}$ at t generations *posterior* to the focal generation. This future-oriented interpretation appears to be more intuitive for understanding the forthcoming results, and I now endorse it for the rest of this article. Finally, I will refer to the set of effects $s_{\mathbf{k},0}$ as "contemporary" effects and to the set of effects $s_{\mathbf{k},t}$ for $t > 0$ as "intertemporal" or long-lasting effects.

Results

SELECTION ON NICHE CONSTRUCTING TRAITS

Substituting the fitness function (eq. 6) into the inclusive fitness effect (eq. 4), evaluating the partial derivatives at $y_\bullet = \ldots = y_{\mathbf{k},t} = \ldots 0$, using the stationary values for the probabilities of identity and simplifying (see eqs. A8–A21 in the Appendix) reveal that a mutant niche constructing allele is selected to increase in frequency in a population with a large number of demes (say $n_d \to \infty$) when $\phi > 0$, where

$$\phi = s + \frac{1}{N} \sum_{t=1}^{\infty} \sum_{\mathbf{k}} s_{\mathbf{k},t} P_{\mathbf{k},t}. \quad (10)$$

The relative fecundity effect $s_{\mathbf{k},t}$ in equation (10) is weighted by the factor $P_{\mathbf{k},t}/N$, where $P_{\mathbf{k},t}$ is the probability that an individual residing in deme $\mathbf{k}$ at t generations posterior to the focal generation has inherited a gene from an individual breeding in the focal deme, and $1/N$ is the probability that this ancestor is the focal individual himself. Hence, current fecundity costs to the actor are traded off against the effects of its behavior on the fecundity of all individuals living in the future of the population, each weighted by the probability ($P_{\mathbf{k},t}/N$) that the recipient is a descendant of the actor. The measure of genetic similarity $P_{\mathbf{k},t}/N$ reflects only the chance of direct descent of the genes of the recipient from those of the actor. All genetic similarity between actor and recipient caused by nondirect descent has cancelled out; a consequence of the increase of kin competition faced by recipients, and resulting from the niche construction trait affecting the fitness of all individuals within demes (see second term in eq. A12).

Equation (10) makes contact with a number of previous results from population genetics. In the absence of any spatial structure ($P_{\mathbf{k},t} \to 0$), selection favors the mutant only when it increases the fecundity of the actor (and/or the viability of its offspring), that is, its expected number of offspring produced before the regulation stage ($s > 0$), where s is sometimes referred to as the adaptive or survival value of a gene (Wright 1969; Gillespie 2004). If spatial structure is added to the system but there are no intertemporal fitness effects, selection favors again the mutant only when it increases the fecundity of the actor and/or the viability of its offspring (Maruyama 1972, 1974; Slatkin 1981). This result is true regardless of the type of contemporary phenotypic effects (positive or negative) actors exert on the fecundity of recipients living in the same or in different demes (Taylor 1992a, b; Rousset 2004). Finally, in the presence of an infinite island model of dispersal and intertemporal fitness effects, selection favors the mutant allele when the cost to the actor is lower than the sum of the fertility effects on individuals living in the focal deme in future

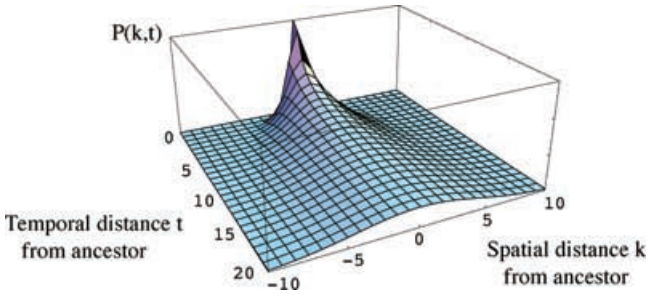

Figure 1. Normally distributed probability density $P_{k,t} \approx (e^{\frac{-k^2}{\sigma^2 t}})/(\sqrt{2\pi\sigma^2 t})$ that in a one-dimensional habitat a line of genes descending from an individual living in a focal deme will reside at distance k from the focal deme (spatial distance) at t generations posterior to the focal generation (temporal distance). The variance of the distance of dispersal is $\sigma^2 = 1$. When t is small, the line of genes descending from a focal individual is likely to be located near the focal deme, whereas for large t, the line of genes tend to be uniformly distributed on the lattice. The ancestor is located at the origin on the graph.

generation, weighted by the probability that they have inherited the niche constructing gene from the focal actor (Lehmann 2006). Equation (10) can be applied in the presence of both, extended phenotypic effects in space and time, and the corresponding selective pressure for finite populations is given by equation (A21) of the Appendix.

Because the movement of a random line of genes (i.e., a line of descent) can be interpreted as the movement of an immortal individual in space, it is conveniently described by a random-walk with step distribution given by the dispersal distribution (Skellam 1951; Sawyer 1975). The probability $P_{\mathbf{k},t}$ can therefore be approximated by the bivariate normal distribution with mean zero and variances $\sigma^2 t$ (Skellam 1951), where the parameter σ^2 is the variance of the axial distance of dispersal; a measure of the speed at which a line of genes moves away from the geographic position of its ancestor (Rousset 2004). The approach of $P_{\mathbf{k},t}$ to normality is very rapid if the dispersal distribution is roughly normal to begin with. Because the variance $\sigma^2 t$ increases as the number of generations goes by, the bell shape fades away, resulting ultimately in a uniform distribution of the focal individual's gene lineage in space (Fig. 1). Hence, pairs of individuals sampled at sufficiently large distances in time will all be unrelated, whatever the structure of the population.

EXPLICIT EXAMPLES

Selection with and without intertemporal fitness effects

To get an intuition for the intensity of selection on niche construction, the coefficients of selection $s_{\mathbf{k},t}$ will now be given an explicit functional form. Because the purpose here is only to compare the selective pressure on a trait with and without niche construction, I do not consider the environment explicitly and use a heuristic

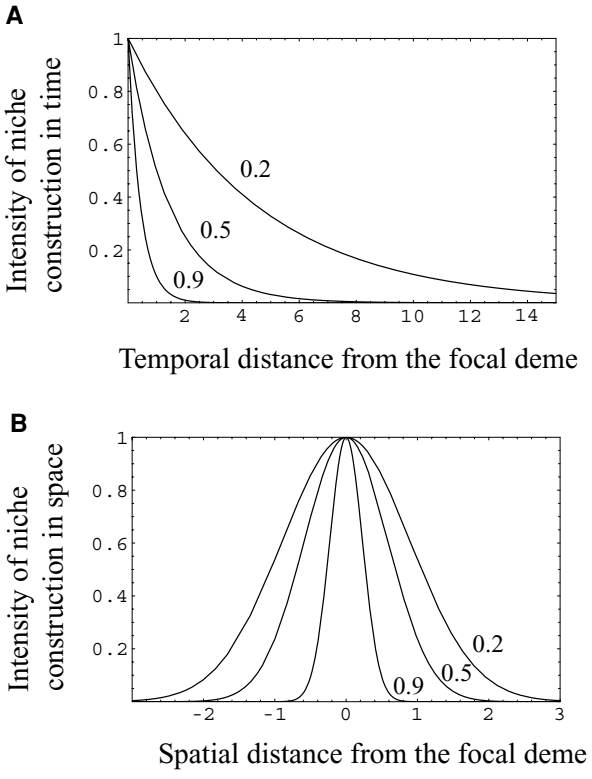

Figure 2. Niche construction effects in both space and time. (A) Extended phenotypic effect in time $B(1-\lambda_T)^t$ of equation (12) with $B = 1$ graphed as a function of time for increasing values of the temporal decay rate, namely $\lambda_T = 0.2$, $\lambda_T = 0.5$, and $\lambda_T = 0.9$. (B) Extended phenotypic effect in space $Be^{\frac{k^2}{\log \lambda_S}}$ of equation (12) with $B = 1$ graphed as a function of the distance from the focal deme for increasing values of the spatial decay rate, namely $\lambda_S = 0.2$, $\lambda_S = 0.5$, and $\lambda_S = 0.9$.

function for the $s_{\mathbf{k},t}$'s (a case with explicit environmental dynamics is considered in the next section). Suppose that the habitat is one dimensional and that the extended phenotype z results in a reduction of the relative fecundity of the actor by C, whereby

$$s = -C \tag{11}$$

and suppose that the phenotype increases the relative fecundity of individuals living in a deme at distance k from the focal deme at t generations posterior to the focal generation by

$$s_{k,t} \equiv B(1-\lambda_T)^t e^{\frac{k^2}{\log \lambda_S}}. \tag{12}$$

This function describes a situation in which an increment in fecundity of magnitude B dies out in space and time according to a spatial decay rate λ_S and a temporal decay rate λ_T (see Fig. 2), where the latter rate is a measure of the ecological inheritance of the niche construction trait.

Substituting the fecundity effects (s and $s_{k,t}$) into equation (10), assuming that $P_{\mathbf{k},t}$ follows a normal distribution and that

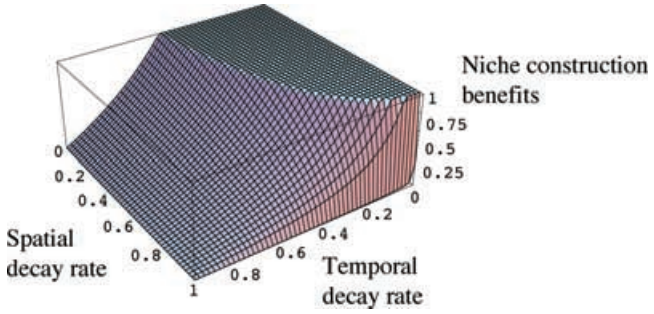

Figure 3. Benefits resulting from intertemporal fitness effects (right-hand side of eq. 13) graphed as a function of the temporal and spatial decay rates (λ_T and λ_S) for $B = 1$, $N = 1$, and $\sigma^2 = 0.25$. The benefits decrease with increasing temporal and spatial decay rates. In the absence of intertemporal fitness effects (i.e., no ecological inheritance or long-lasting fitness effects), the benefits are equal to zero. Consequently, if the niche construction trait is costly to the actor ($C > 0$ in eq. 13), it cannot evolve in the absence of ecological inheritance ($\lambda_T = 1$).

space is continuous, the condition for the invasion of the niche construction trait is approximated for large λ_S (fast spatial decay) by

$$\frac{C}{B} < \frac{\sqrt{1-\lambda_S}}{N\sqrt{2\sigma^2}}\left(\sum_{t=1}^{\infty}\frac{(1-\lambda_T)^t}{t^{\frac{1}{2}}}\right). \tag{13}$$

This equation shows that when all phenotypic effects on the environment are erased from one generation to the next ($\lambda_T = 1$), the trait cannot evolve if it results in a fecundity cost to the actor. This is a classical result (Maruyama 1974; Taylor 1992b; Rousset 2004). However, in the presence of ecological inheritance ($\lambda_T < 1$), the equality can be satisfied so that indirect future benefits drive the evolution of a costly trait (see Fig. 3). Future benefits can become substantial but decrease as the variance in dispersal σ^2 increases. We now turn to a more complicated example where the extended phenotypic effects $s_{k,t}$ are determined by the dynamics of the environment, itself a function of the evolving niche construction trait.

Selection for resource conservation

In this section, I derive the candidate evolutionary stable state of a trait involved in the consumption of an abiotic resource (e.g., nitrogen, phosphorous, water) and the associated environmental stable state of the resource. The consumption of the resource is assumed to result in a linear monotonic increase of the fitness of individuals using it (introducing diminishing return does not qualitatively change the following results but leads to a more complicated analysis). For simplicity, I again consider a one-dimensional habitat with no migration of the resource between locations. The organism is assumed to have a functional response of Type I for the resource (Holling 1959; Murdoch et al. 2003; Rueffler et al. 2006).

With the assumptions just spelt out, the fecundity of a focal individual can be written as

$$b(z_\bullet, \mathbf{z}) = 1 + \sum_{j=0}^{\infty}\mathcal{E}(\mathbf{z}_j)\beta_j a(1 - z_\bullet), \tag{14}$$

where $\mathcal{E}(\mathbf{z}_j)$ is the amount of the resource in a deme at distance j relative from the focal deme and β_j is the proportion of time spent by the focal individual searching for the resource in that deme ($\sum_j \beta_j = 1$). Search time is assumed to follow a normal distribution with mean zero and variance ω^2, which can be interpreted as a measure of the home range of the organism. The parameter a is the consumer search efficiency weighted by the attack rate (area or volume cleared of resource per individual, Murdoch et al. 2003), which is weighted by $(1 - z_\bullet)$ in equation (14). Hence, the mutant can be seen as decreasing the parameter a by magnitude δ, and one can now ask whether such a mutant allele will be selected for when restraint in resource use will increase resource abundance in later generations. The dynamics of the resources (the $\mathcal{E}(\mathbf{z}_j)$'s) are detailed in the Appendix (eq. A46) and are affected by the number and phenotypes of individuals consuming them, a natural rate r of replenishment and a natural rate μ of depletion. I further assume that resource consumption by the focal species decreases the replenishment rate of the resource by a factor α, which can be thought as an interference between consumption and replenishment.

In this consumer–resource setting, the effect of the focal individual on its relative fecundity evaluated at $z_\bullet = \ldots = z_{k,t} = \ldots = 0$ is given by

$$s = -\frac{a\mathcal{E}}{1 + a\mathcal{E}}, \tag{15}$$

(eq. A37 of the Appendix) where

$$\mathcal{E} = \frac{r\,(1 - \alpha a N)}{\mu + aN} \tag{16}$$

is the resource level determined by the resident allele (eq. A56 in the Appendix). The numerator of the last equation represents the net replenishment rate of the resource, which is affected by the consumption of the focal species through the interference parameter α. The denominator represents the net depletion rate of the resource (natural depletion plus depletion due to the focal species). The coefficient of selection s is a net fitness cost because, by refraining from consuming resources, the focal individual looses fecundity shares, which decreases its fitness.

The effect of the focal individual on the relative fecundity of the N individuals living in a deme at distance k from the focal

deme and at generation t posterior to the focal generation is given by

$$s_{k,t} = \frac{a(r\alpha + \mathcal{E})}{1 + a\mathcal{E}} \sum_{j=0}^{\infty} \beta_j \beta_{j-k} aN(1 - \mu - aN)^{t-1} \quad (17)$$

(eq. A38 of the Appendix). This is a net benefit because by refraining from consuming a marginal amount of $a\mathcal{E}$ units of resources in a deme at distance j from the focal deme in the focal generation (e.g., eq. 15), the focal individual increases the availability of the resource by a marginal amount of $a(\alpha + \mathcal{E})$ units in that deme. The fraction of the additional resources that are actually available for individuals living t generation in the future is given by $(1 - \mu - Na)^{t-1}$, where $\mu + Na$ represents the total depletion rate (or discount of the resource) due to both natural depletion and consumption of the resource by the individuals in the population in intervening times. The greater the depletion rate, the lower the impact of resource restraint on future generations.

Substituting the fecundity effects into the selective pressure (eq. 10), assuming that dispersal follows a normal distribution, that the home range of individuals is larger than the dispersal range ($\omega^2 > \sigma^2$) and that the habitat can be considered as continuous (see derivation eq. A45 in the Appendix), we find that the mutant allele spreads when

$$\frac{\mathcal{E}}{r\alpha + \mathcal{E}} < \frac{a}{2\sqrt{\pi}(\mu + aN)\omega} \left(1 - \frac{\sigma^2}{4(\mu + aN)\omega^2}\right). \quad (18)$$

The left-hand side can be interpreted as the ratio of the marginal cost to the marginal benefit of the act decreasing the attack rate. When $\alpha = 0$, marginal cost and benefit of the act are equal and we do not expect restraint in resource consumption to evolve. Restraining resource consumption is only worthwhile when the act creates a surplus. A necessary condition for that trait to evolve is thus that $\alpha > 0$ and it can indeed be checked that inequality 18 cannot be satisfied when $\alpha = 0$. For $\alpha > 0$, the condition of invasion (eq. 18) can be satisfied but it becomes more stringent when the variance in dispersal σ^2 increases, so that individuals living in the local environment of an actor in later generations are less likely to be its descendants, and when the home range ω^2 of individuals increases, so that the actor is less likely to consume local resources that could be used by its relatives living in future generations. Finally, the condition of invasion of restraint in resource use becomes more stringent when the natural rate μ of depletion increases so that the niche construction effects of previous generation have a lower impact on subsequent generations.

By successive allele replacement the population may eventually reach the point where both the left- and the right-hand side of inequality 18 are the same. For a given set of parameter values, the value of the attack rate $a^\star$ that equates both sides of inequality 18 is the candidate evolutionary stable attack rate, which, once substituted into equation (16) yields the corresponding evolutionary stable environmental state. These two quantities are plotted in Figure 4 as a function of α and for different values of the dispersal distribution σ^2 and the home range ω^2.

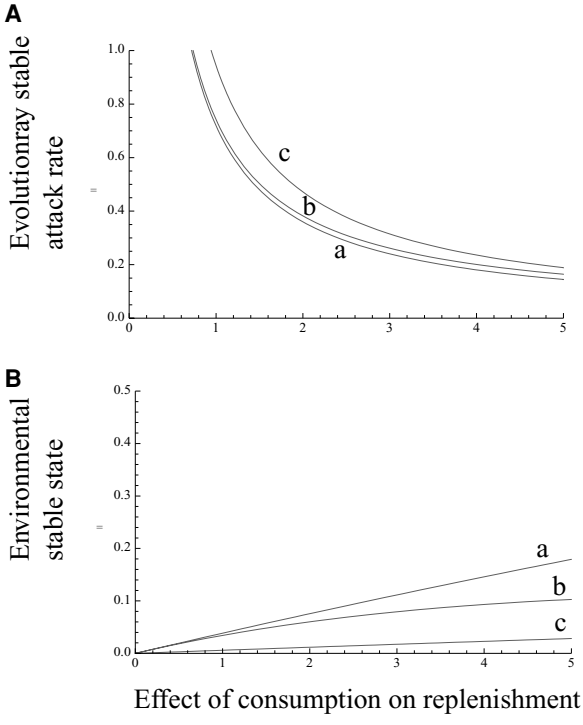

Figure 4. Candidate evolutionary stable attack rate $a^\star$ and associated stable environmental state $\mathcal{E}(a^\star)$. (A) Evolutionary stable attack rate $a^\star$ obtained by equating both sides of equation (18) and solving for a, and then graphed as a function of the parameter α (interference between consumption and replenishment) for different combination of parameter values. The lower line (labeled a) corresponds to $N = 1$, $\mu = 0.01$, $r = 0.1$, $\sigma^2 = 0.01$, and $\omega^2 = 1$. The second line (labeled b) corresponds to the same parameters values as the first line, except that the variance in dispersal is increased to 0.5. The third line (labeled c) corresponds to the same parameter values as the first line except that the home range is increased to 5. The attack rate decreases with increasing values of α because the marginal benefit of refraining from consuming the resource increases relative to the marginal cost. By contrast, an increase in the variance in dispersal or the home range results in an increase of the attack rate. (B) Environmental stable state $\mathcal{E}(a^\star)$ (see eq. 16) resulting from the attack rates graphed in panel A. The labels of the lines correspond to the ones given in panel A. The equilibrium level of the resource decreases with an increase in both the variance in dispersal and the home range. Note that in the absence of resource consumption ($a = 0$) the equilibrium value of the resource is given by $\mathcal{E} = 10$ (i.e., $\mathcal{E} = r/\mu$). If $\alpha = 0$, selection drives the resource to extinction.

Discussion

Niche construction, by which organisms modify the environment in which they live is likely to feed back on the selective pressure

of a variety of traits (e.g., Odling-Smee et al. 1996, 2003; Laland et al. 2001; Ihara and Feldman 2004; Borenstein et al. 2006). However, the selective pressure on niche constructing traits themselves has received little attention so far. Niche constructing traits or extended phenotypes in time (Dawkins 1982) can potentially affect the environment from local to global scales, and thus change the pattern of natural selection to which the phenotypes are exposed in subsequent generations. This article presents an inclusive fitness analysis of selection on such traits when evolution occurs in subdivided populations of finite and constant size.

SELECTION ON NICHE CONSTRUCTING PHENOTYPES

The main result of this article (see eq. 10) is that the selective pressure on a niche construction behavior in spatially subdivided populations depends on at least two classes of fitness effects. First, it depends on the effect of the behavior on the actor's own fecundity (where fecundity is defined as the total number of offspring produced that reach the regulation stage). Second, it depends on the effect of the behavior of the actor on the fecundity of all individuals living in subsequent generations, each weighted by the probability that it has inherited the genes underlying the niche construction behavior from the actor ($P_{\mathbf{k},t}/N$ in eq. 10). In other words, present direct benefits are traded off by future indirect costs (or vice versa). Equation 10 illustrates that phenotypic effects of genes extending far beyond the life span of an actor may be shaped by natural selection, whenever they impact the fitness of individuals that are likely to have inherited its genes. This suggests that the evolution of niche construction traits can be interpreted in terms of kin selection, but with individuals affecting the fitness of relatives posthumously.

The strength of selection on the trait that arise from intertemporal fitness costs and/or benefits depends on the variance in dispersal distance (σ^2), which characterizes entirely the population genetic structure in both space and time when the dispersal distribution is approximately normal and the number of demes is very large. Assuming that the fitness effects (the $s_{\mathbf{k},t}$'s) will eventually completely decay with increasing spatial and temporal distances, the intensity of selection on a niche construction trait decreases with an increase of the variance of dispersal (see eq. 13). This is so because the variance in dispersal measures the speed at which a line of genes moves away from the geographic position of its ancestor. In other words, it determines the extent to which individuals living in the local environment of an actor in later generations are likely to be its descendants. With a high variance in dispersal, the relatedness between individuals living in different generations at the same spatial location decreases markedly. Consequently, the likelihood that an individual living in later generations than the actor will benefit (or suffer) from the locally modified environment by its ancestor decreases, and ecological inheritance can no longer translate into inclusive fitness benefits.

SELECTION WITH AND WITHOUT INTERTEMPORAL FITNESS EFFECTS

Figure 3 compares the benefits (or costs) of a niche construction allele with and without intertemporal fitness effects for a given value of the dispersal distribution. When all phenotypic effects on the environment are erased from one generation to the next (i.e, no ecological inheritance, $\lambda_T = 1$ in eq. 12), a behavior costly to the actor cannot be selected under the assumptions of the present model because the contemporary benefits cancel out through the increase in kin competition (Taylor 1992b; Rousset 2004). By contrast, when effects on the environment are not completely erased from one generation to the next, which is likely to occur for inceptive perturbations (e.g., nests, burrow, Odling-Smee et al. 2003, table 2.1), a behavior costly to the actor can evolve if present costs are offset by future benefits. Ecological inheritance may thus be a potent factor accounting for the evolution and/or maintenance of altruistic traits in species constructing long-lasting artifacts such as the social insects. Undeniably, social and eusocial insects, not only provide direct care to kin, but also construct, maintain, and regulate hives and mounds (Wilson 1975). These are all costly, altruistic acts, but from which distant descendants may reap the benefits.

The selective gradient (eq. 10) is a surprisingly intuitive and simple result given the complexity of the model we began with (eq. 6). One might thus wonder whether its predictions are likely to hold in the presence of other reproductive schemes and/or demographic processes. Indeed, the life cycle assumes that all adult individuals die per unit of time (semelparous organism) and is thus specific. But importantly, this assumption is well documented to provide the most difficult situation for the evolution of social behaviors through inclusive fitness benefits in spatially subdivided populations (Taylor 1992a; Taylor and Irwin 2000; Rousset 2004; Lehmann et al. 2007). By contrast, assuming overlapping generations, increases the kin selection pressure on traits affecting interactions between individuals in subdivided populations. Indeed, in the presence of overlapping generations the contemporary fitness effects ($s_{\mathbf{k},0}$) no longer cancel out from the inclusive fitness equation (Taylor and Irwin 2000; Irwin and Taylor 2001). This occurs because a parent is now likely to interact directly with one of its offspring, thereby markedly increasing the intrageneration relatedness coefficients between local neighbors (the $Q_{\mathbf{k},0}$'s). The increase in the selective pressure is the strongest under the assumptions of "evolutionary graph theory" where exactly one individual dies per unit of time (e.g., Ohtsuki et al. 2006; Ohtsuki and Nowak 2006; Lehmann et al. 2007). Because overlapping-generations increase the kin selective pressure on any trait even in the absence of niche construction, we expect that overlapping generations will increase selection on a niche construction trait in general, because all space-time relatedness coefficients between local neighbors will be increased. An increase of the intensity of selection on niche

construction is further expected if any demographic assumption increasing the probabilities of identity is introduced (e.g., specific mating schemes, dispersal modes, life-history strategies). These comments suggest that different life cycle assumptions will quantitatively affect the present results, but in general, one expects stronger selection on intertemporal fitness effects than the present analysis suggests.

When the population is of finite size, the selective pressure on niche construction becomes more complicated and involves an additional term (compare eq. 10 and eq. A21 of the Appendix). The intertemporal fitness effects are then weighted by $(P_{\mathbf{k},t} - 1/n_d)/N$ instead of being weighted by $P_{\mathbf{k},t}/N$, where $1/n_d$ represents the probability that, under panmixia, any recipient living at any generation posterior to a focal actor has inherited the genes from an individual living in the focal deme in the focal generation. Hence, $1/n_d$ is a reference point for evaluating the genetic similarity between actor and recipient. In a panmictic population (i.e., $P_{\mathbf{k},t} = 1/n_d$) all intertemporal fitness effects vanish, because a recipient is then not more (or less) likely to bear genes identical by descent with the actor than is any individual taken at random from the population at any generation (i.e., relatedness vanishes). If recipients residing in a particular location of the habitat (say deme $\mathbf{k}$ at generation t in the future) have a probability of inheriting the genes of the actor that is greater than the one resulting from random dispersal (i.e., $P_{\mathbf{k},t} > 1/n_d$), those recipients are then likely to be positively related to him. Equation (A21) suggests that as long this is true, any positive intertemporal fitness effect ($s_{\mathbf{k},t} > 0$) on these recipients can only augment the inclusive fitness of the actor. By contrast, if a particular class of recipients have a probability of inheriting the genes of the actor that is lower than the one resulting from random dispersal (i.e., $P_{\mathbf{k},t} < 1/n_d$), these recipients are likely to be negatively related to him. Equation (A21) then suggests that any negative intertemporal fitness effect ($s_{\mathbf{k},t} < 0$) on these recipients may augment the inclusive fitness of the actor. Hence, natural selection can favor the expression of behaviors that are costly to the actor and spiteful posthumously.

Posthumous spiteful behaviors may occur in bacterial lineages. Indeed, by producing extracellular polymers, bacteria shape biofilms that are likely to extend over the life span of a single individual. Such polymer production is costly for the actor, beneficial for individuals bearing its gene lineage and deleterious for neighboring gene lineages (Xavier and Foster 2007). More generally, bacteria release into their environment intraspecific antagonist compounds such as bacteriocins and bacteriophages, a weaponry allowing them to suppress the growth of competing strains (Riley and Gordon 1999; Gardner et al. 2004). If the half-life of these compounds exceeds the half-life of the bacteria, ecological inheritance will be operating, which might eventually lead to posthumous harming being selected for. Similarly, intraspecific allelopathy occur in plants (Groner 1974), where allelochemicals are released into the environment by a focal plant, which subsequently inhibits the growth and development of neighboring plants of the same and different generations. This suggests that long-lasting phenotypic effects on fitness can not only promote selection on a focal gene lineage by augmenting its reproduction (i.e., helping) but also by decreasing competition (i.e., harming).

SELECTION FOR CONSERVING RESOURCES

The adaptive dynamics of the attack rate of a resource was derived here as an explicit example where the intertemporal fitness effects of niche construction can affect the evolution of a trait. The ecological context for the evolution of this attack rate can be thought of as a simple consumer–resource system (e.g., Roughgarden 1976; Abrams 1999; Murdoch et al. 2003; Vincent and Brown 2005; Abrams 2006), with the population of predators being of constant size but subdivided in space, and with an adaptive change occurring in the amount of prey consumed. In the absence of population structure, the attack rate of the resource would evolve toward the point where selection-driven extinction of the resource would occur. Here, I asked under what condition can restraint in resource consumption evolve, when the act is costly in the present but results in an increase of the abundance of the resource posthumously.

The primary condition for natural selection decreasing resource consumptions in a population in which all individuals face the same needs is that restraining resource consumption today creates a surplus of resources tomorrow. In other words, the benefit created by the act must exceed the cost. This is possible if resource consumption interferes with resource replenishment ($\alpha > 0$). When restraint in resource consumption can be selected for, the selective pressure of the trait decreases when: the variance (σ^2) in dispersal increases, the home range (ω^2) of individuals increases and the natural rate (μ) of depletion increases (see Fig. 4). The rate of depletion can be interpreted here as a measure of the "ecological inheritance" in the system. When $\mu = 1$, resources produced in past generations are naturally erased so that the level of the resource in any generation depends only on current production. In this case, restraint in resource consumption can never evolve. This absence of ecological inheritance may correspond to situations in which the generation time of the resource is much shorter that the generation time of the focal species so that the value of the environment reaches its steady state during a single generation of the focal species (e.g., Rueffler et al. 2006). By contrast, when $\mu < 1$, the resource level in a focal generation is likely to depend on the phenotypes of individuals living in adjacent generations and restraint in resource use can evolve, provided the indirect benefits resulting from the posthumous increase in resource abundance offset the current costs to the actor.

Even though the present model lacks any explicit population dynamics in the focal species, it sheds light on the selective pressure on behaviors affecting consumer–resource systems, which are the basic units of ecological communities (e.g., Roughgarden 1976; Abrams 1999, 2006; Murdoch et al. 2003; Vincent and Brown 2005). The model illustrates that the way organisms have been selected to consume their resources depends on intertwined life-history, ecological and demographic factors; where the values of the dispersal distribution, the home range of the focal species (here the predator), and the natural depletion rate dictate whether conservation or selection-driven extinction of the resource will occur. For instance, pastoralism, which has been described as causing environmental degradation (Fratkin 1997), includes low group density and high mobility, a situation not favorable for selection on resource conservation. The migration distribution and the home range are important for the evolution of resource consumption because they determines the structure of the populations, which itself determines how resource consumption will feed back on the inclusive fitness of the actor expressing the trait. The present formalization could also be used to investigate issues of optimal foraging such as specialist–generalist trades off (e.g., Abrams 2006; Rueffler et al. 2006), in the absence of explicit predator dynamics but with an explicit spatial structure of the population of predators. But more generally, both prey and predators may fluctuate in size, and evolution under this intertwined demographic processes can be analyzed with the framework of Rousset and Ronce (2004), which incorporates selection on niche construction traits as a special case by taking long-lasting fitness effects into account through the use of the concept of reproductive value.

The present analysis is based on the simplifying assumptions that selection is weak and that the absolute positions of individuals in both space and time have no specific effect on fitness (spatiotemporal homogeneity). But it suggests that the selective pressure on traits resulting in a physical modification of the environment can in general markedly feedback on the evolution of the trait through indirect kin selection pressure. In our own species, the transition from hunting and gathering to agriculture resulted in the permanent or semipermanent settlements that are associated with the domestication of plant and animals (Bellwood 2004). This ecological transition led to many inceptive perturbations such as the constructions of houses, fortifications, agricultural fields, and technological innovations, which potentially last far beyond the life span of the constructor. Permanent settlements reduce the movement of individuals in their habitat and thus decrease the variance in the distance of dispersal, creating more favorable conditions for selection on niche construction. This raises the intriguing question of the extent to which humans have been shaped by natural selection to behave in accordance to their impact on future generations, be it at a local or at a more global scale.

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LITERATURE CITED

Abrams, P. A. 1999. The adaptive dynamics of consumer choice. Am. Nat. 153:83–97.

———. 2006. Adaptive change in the resource-exploitation traits of a generalist consumer: the evolution of and coexistence of generalists and specialists. Evolution 60:427–439.

Ajar, E. 2003. Analysis of disruptive selection in subdivided populations. BMC Evol. Biol. 3:1–12.

André, J. B., C. Peeters, and C. Doums. 2001. Serial polygyny and colony genetic structure in the monogynous queenless ant *Diacamma cyaneiventre*. Behav. Ecol. Sociobiol. 50:72–80.

Aoki, K. 1982. A condition for group selection to prevail over counteracting individual selection. Evolution 36:832–842.

Begon, M., J. L. Harper, and C. R. Townsend. 1996. Ecology: individuals, populations and communities. 6th ed. Blackwell Science, Oxford.

Bellwood, P. 2004. First farmers: the origins of agricultural societies. Blackwell Publishers, Oxford.

Borenstein, E., J. Kendal, and M. W. Feldman. 2006. Cultural niche construction in a metapopulation. Theor. Pop. Biol. 70:92–104.

Darwin, C. 1883. The formation of vegetable mold through the action of worms, with observations on their habits. John Murray, London.

Dawkins, R. 1982. The extended phenotype. Oxford Univ. Press, Oxford.

Epperson, B. K. 1999. Gene genealogies in geographically structured populations. Genetics 152:797–806.

———. 2003. Geographical genetics. Princeton Univ. Press, Princeton, NJ.

Eshel, I. 1972. On the neighbor effect and the evolution of altruistic traits. Theor. Pop. Biol. 11:258–277.

———. 1996. On the changing concept of evolutionary population stability as a reflection of a changing point of view in the quantitative theory of evolution. J. Math. Biol. 34:485–510.

Evans, J. D. 1996. Queen longevity, queen adoption, and posthumous indirect fitness in the facultatively polygynous ant *Myrmica tahoensis*. Behav. Ecol. Sociobiol. 39:275–284.

Frank, S. A. 1998. Foundations of social evolution. Princeton Univ. Press, Princeton, NJ.

Fratkin, E. 1997. Pastoralism: governance and development issues. Annu. Rev. Anthropol. 26:235–261.

Gadagkar, R., K. Chandrasehekara, S. Chandran, and S. Bhagavan. 1993. Serial polygyny in the primitively eusocial wasp ropalidia marginata: implications for the evolution of sociality Pp. 1–15 *in* K. Laurent, ed. Queen number and sociality in insects. Oxford Univ. Press, Oxford.

Gandon, S., and F. Rousset. 1999. Evolution of stepping-stone dispersal rates. Proc. R. Soc. Lond. B 221:2507–2513.

Gardner, A., S. A. West, and A. Buckling. 2004. Bacteriocins, spite and virulence. Proc. R. Soc. Lond. B 271:1529–1535.

Geritz, S. A. H., J. A. J. Metz, E. Kisdi, and G. Meszéna. 1997. Dynamics of adaptation and evolutionary branching. Physic. Rev. Lett. 78:2024–2027.

Geritz, S. A. H., E. Kisdi, G. Meszéna, and J. A. J. Metz. 1998. Evolutionarily singular strategies and the adaptive growth and branching of the evolutionary tree. Evol. Ecol. 12:35–57.

Gillespie, J. H. 2004. Population genetics: a concise guide. Johns Hopkins, Baltimore & London.

Gotwald, W. 1995. Army ants: the biology of social predation. Cornell Univ. Press, Ithaca.

Grafen, A. 2006. Optimization of inclusive fitness. J. Theor. Biol. 238:541–563.

Grimmet, G., and D. Stirzaker. 2001. Probability and Random Processes. Oxford Univ. Press, Oxford.

Groner, M. G. 1974. Intraspecific allelopathy in kalanchoe daigremontiana. Bot. Gazett. 135:73–79.

Gyllenberg, M., and K. Parvinen. 2001. Necessary and sufficient conditions for evolutionary suicide. Bull. Math. Biol. 63:981–993.

Hamilton, W. D. 1964. The genetical evolution of social behaviour, I. J. Theor. Biol. 7:1–16.

———. 1975. Innate social aptitudes in man, an approach from evolutionary genetics. Pp. 133–157 *in* R. Fox, ed. Biosocial anthropology. Malaby Press, London.

Hansell, M. 1984. Animal architecture and building behavior. Longmans, New York.

Holling, C. S. 1959. Some characteristics of simple types of predation and parasitism. Canad. Entomol. 91:385–398.

Hoogland, J. 1995. Taxonomy and natural history. Pp. 7–25 *in* J. Hoogland, ed. The black-tailed prairie dog: social life of a burrowing mammal. Univ. of Chicago Press, Chicago.

Ihara, Y., and M. W. Feldman. 2004. Cultural niche construction and the evolution of small family size. Theor. Pop. Biol. 65:105–111.

Irwin, A. J., and P. D. Taylor. 2001. Evolution of altruism in stepping-stone populations with overlapping generations. Theor. Pop. Biol. 60:315–325.

Laland, K. L., F. J. Odling-Smee, and M. W. Feldman. 1999. Evolutionary consequences of niche construction and their implications for ecology. Proc. Natl. Acad. Sci. 96:10242–10247.

———. 2001. Cultural niche construction and human evolution. J. Evol. Biol. 14:22–33.

Le Galliard, J., R. Ferrière, and U. Dieckmann. 2003. The adaptive dynamics of altruism in spatially heterogeneous populations. Evolution 57:1–17.

Lehmann, L. 2006. The evolution of trans-generational altruism: kin selection meets niche construction. J. Evol. Biol. 20:181–189.

Lehmann, L., L. Keller, and D. Sumpter. 2007. The evolution of helping and harming on graphs: the return of the inclusive fitness effect. J. Evol. Biol. 20:2284–2295.

Lessard, S. 2005. Long-term stability from fixation probabilities in finite populations: new perspectives for ESS theory. Theor. Pop. Biol. 68:19–27.

Lewontin, R. C. 2000. The triple helix. genes, organism, and environment. Harvard Univ. Press, Cambridge, MA.

Malécot, G. 1975. Heterozygosity and relationship in regularly subdivided populations. Theor. Pop. Biol. 8:212–241.

Maruyama, T. 1970. Effective number of alleles in a subdivided population. Theor. Pop. Biol. 1:273–306.

———. 1972. Some invariant properties of a geographically structured finite population: distribution of heterozygotes under irreversible mutation. Gen. Res. Camb. 20:141–149.

———. 1974. A simple proof that certain quantities are independent of the geographical structure of population. Theor. Pop. Biol. 5:148–154.

Matsuda, H., and P. A. Abrams. 1994. Runaway evolution to self-extinction under asymmetrical competition. Evolution 48:1764–1772.

Murdoch, W. W., J. B. Cheryl, and R. M. Nisbet. 2003. Consumer-resource dynamics. Princeton Univ. Press, Princeton, NJ.

Nagylaki, T. 1983. The robustness of neutral models of geographical variation. Theor. Pop. Biol. 24:268–294.

Odling-Smee, F. J., K. L. Laland, and M. W. Feldman. 1996. Niche construction. Am. Nat. 147:641–648.

———. 2003. Niche construction. Princeton Univ. Press, Princeton, NJ.

Ohtsuki, H., and M. A. Nowak. 2006. Evolutionary games on cycles. Proc. R. Soc. Lond. B 273:2249–2256.

Ohtsuki, H., C. Hauert, E. Lieberman, and M. A. Nowak. 2006. A simple rule for the evolution of cooperation on graphs and social networks. Nature 441:502–505.

Orzack, S. H., and E. Sober (eds.) 2001. Adaptationism and optimality. Cambridge Univ. Press, Cambridge.

Parvinen, K. 2005. Evolutionary suicide. Acta Biotheor. 53:241–264.

Peeters, C. 1993. Monogyny and polygyny in ponerine ants with or without queens Pp. 234–261 *in* K. Laurent, ed. Queen number and sociality in insects. Oxford Univ. Press, Oxford.

Perrin, N., and L. Lehmann. 2001. Is sociality driven by the costs of dispersal or the benefits of philopatry? A role for kin discrimination mechanisms. Am. Nat. 158:471–483.

Reeve, H. K., and P. W. Sherman. 1993. Adaptation and the goal of evolutionary research. Quart. Rev. Biol. 68:1–32.

Riley, M. A., and D. M. Gordon. 1999. The ecological role of bacteriocins in bacterial competition. Trend. Microbiol. 7:129–133.

Roughgarden, J. 1976. Resource partitioning among competing species—a coevolutionary approach. Theor. Pop. Biol. 9:388–424.

Rousset, F. 1997. Genetic differentiation and estimation of gene flow from F-statistics under isolation by distance. Genetics 145:1219–1228.

———. 2003. A minimal derivation of convergence stability measures. J. Theor. Biol. 221:665–668.

———. 2004. Genetic structure and selection in subdivided populations. Princeton Univ. Press, Princeton, NJ.

———. 2006. Separation of time scales, fixation probabilities and convergence to evolutionarily stable states under isolation by distance. Theor. Pop. Biol. 69:165–179.

Rousset, F., and S. Billiard. 2000. A theoretical basis for measures of kin selection in subdivided populations: finite populations and localized dispersal. J. Evol. Biol. 13:814–825.

Rousset, F., and O. Ronce. 2004. Inclusive fitness for traits affecting metapopulation demography. Theor. Pop. Biol. 65:127–141.

Rueffler, C., T. J. M. Van Dooren, and J. A. J. Metz. 2006. The evolution of resource specialization through frequency-dependent and frequency-independent mechanisms. Am. Nat. 167:81–93.

Sawyer, S. 1975. Results for the stepping stone model for migration in population genetics. Ann. Prob. 4:699–728.

Silver, M., and E. Di Paolo. 2006. Spatial effects favour the evolution of niche construction. Theor. Pop. Biol. 70:387–400.

Skellam, J. G. 1951. Random dispersal in theoretical populations. Biometrika 38:196–218.

Skutch, A. F. 1987. Helpers at bird's nest. Univ. of Iowa Press, Iowa City.

Slatkin, M. 1981. Fixation probabilities and fixation times in a subdivided population. Evolution 35:477–488.

Taylor, P. D. 1992a. Altruism in viscous populations—an inclusive fitness model. Evol. Ecol. 6:352–356.

———. 1992b. Inclusive fitness in a homogeneous environment. Proc. R. Soc. Lond. B 240:299–302.

Taylor, P. D., and S. A. Frank. 1996. How to make a kin selection model. J. Theor. Biol. 180:27–37.

Taylor, P. D., and A. J. Irwin. 2000. Overlapping generations can promote altruistic behavior. Evolution 54:1135–1141.

van Baalen, M., and A. Rand. 1998. The unit of selection in viscous populations and the evolution of altruism. J. Theor. Biol. 193:631–648.

Vincent, T. L., and J. S. Brown. 2005. Evolutionary game theory, natural selection, and Darwinian dynamics. Cambridge Univ. Press, Cambridge.

Wakano, J. Y. 2007. Evolution of cooperation in spatial public goods games with common resource dynamics. J. Theor. Biol. 247:616–622.

Wilson, E. O. 1975. Sociobiology: the new synthesis. Harvard Press, Cambridge, MA.
Wolfram, S. 2003. Mathematica. 5th ed. Cambridge Univ. Press, Cambridge.
Wright, S. 1969. Evolution and the genetics of populations. II. The theory of gene frequencies. Univ. of Chicago Press, Chicago.
Xavier, J. B., and K. R. Foster. 2007. Cooperation and conflict in microbial biofilms. Proc. Natl. Acad. Sci. 104:876–881.

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APPENDIX

EFFECTS OF ACTORS ON RELATIVE FECUNDITY

Here, I show how the direct fitness function w (eq. 5) can be expressed in terms of effects of actors on the relative fecundity of recipients. Equation (5) can equivalently be written as

$$w(z_\bullet, \mathbf{E}(\mathbf{z})) = \sum_{\mathbf{i}} m_{\mathbf{i}} \frac{f(z_\bullet, \mathbf{z})}{\sum_{\mathbf{j}} m_{\mathbf{i}-\mathbf{j}} f(z_{\mathbf{j},0}, \mathbf{z}_{\mathbf{j}})}, \tag{A1}$$

where

$$f(z_\bullet, \mathbf{z}) \equiv \frac{b(z_\bullet, \mathbf{z})}{b(z, z)} \tag{A2}$$

is the average fecundity of the focal individual relative to the average fecundity $b(z, z)$ of an individual in a population monomorphic for the resident allele and

$$f(z_{\mathbf{j},0}, \mathbf{z}_{\mathbf{j}}) \equiv \frac{b(z_{\mathbf{j},0}, \mathbf{z}_{\mathbf{j}})}{b(z, z)} \tag{A3}$$

is the relative fecundity of individuals living in a deme at distance $\mathbf{j}$ from the focal deme.

Taylor expanding equation (A2) around the phenotypic value of the resident allele ($z_\bullet = \ldots, z_{k,t}, \ldots = z$), one has

$$f(z_\bullet, \mathbf{z}) = 1 + \frac{1}{b(z, z)} \left(\frac{\partial b(z_\bullet, \mathbf{z})}{\partial z_\bullet} (z_\bullet - z) + \sum_{t=0}^{\infty} \sum_{\mathbf{k}} \frac{\partial b(z_\bullet, \mathbf{z})}{\partial z_{\mathbf{k},t}} (z_{\mathbf{k},t} - z) \right) + O(\delta^2), \tag{A4}$$

where $O(\delta^2)$ is a remainder of order δ^2. This remainder can be neglected under the assumption of weak selection, under which one considers only effects of first order (i.e., effects of intensity δ). Similarly, by Taylor expanding equation (A3) around the phenotypic value of the resident allele, one has

$$f(z_{\mathbf{j},0}, \mathbf{z}_{\mathbf{j}}) = 1 + \frac{1}{b(z, z)} \left(\frac{\partial b(z_{\mathbf{j},0}, \mathbf{z}_{\mathbf{j}})}{\partial z_{\mathbf{j},0}} (z_{\mathbf{j},0} - z) + \sum_{t=0}^{\infty} \sum_{\mathbf{k}} \frac{\partial b(z_{\mathbf{j},0}, \mathbf{z}_{\mathbf{j}})}{\partial z_{\mathbf{j}-\mathbf{k},t}} (z_{\mathbf{j}-\mathbf{k},t} - z) \right) + O(\delta^2). \tag{A5}$$

I will now define several variables: $y_\bullet \equiv (z_\bullet - z) = \delta$ and $y_{\mathbf{k},t} \equiv (z_{\mathbf{k},t} - z)$, where $y_{\mathbf{k},t}$ represents the average phenotypic deviation (but excluding the focal individual from the average) of an individual living in deme $\mathbf{k}$ at t generations prior to the focal generation, relative to an individual carrying the mutant allele;

$$s \equiv \frac{1}{b(z, z)} \frac{\partial b(z_\bullet, \mathbf{z})}{\partial z_\bullet} \bigg|_{z_\bullet = \ldots, z_{k,t}, \ldots = z} \tag{A6}$$

and

$$s_{\mathbf{k},t} \equiv \frac{1}{b(z, z)} \frac{\partial b(z_\bullet, \mathbf{z})}{\partial z_{\mathbf{k},t}} \bigg|_{z_\bullet = \ldots, z_{k,t}, \ldots = z} \tag{A7}$$

which are effects of actors on the relative fecundity of the focal individual. With theses variables in hand and noting that $\partial b(z_\bullet, \mathbf{z})/\partial z_\bullet = \partial b(z_{\mathbf{j},0}, \mathbf{z}_{\mathbf{j}})/\partial z_{\mathbf{j},0}$ and $\partial b(z_\bullet, \mathbf{z})/\partial z_{\mathbf{k},t} = \partial b(z_{\mathbf{j},0}, \mathbf{z}_{\mathbf{j}})/\partial z_{\mathbf{j}-\mathbf{k},t}$ at $z_\bullet = \ldots z_{k,t}, \ldots = z$, shows that equations (A4) and (A5) correspond, respectively, to the numerator and denominator of equation (6).

INCLUSIVE FITNESS EFFECT

In this appendix, I evaluate the inclusive fitness effect (eq. 4) explicitly when the fitness function w is given by equation (6). To this aim, I use classical results on Fourier analysis. All the results used in this appendix are spelt out in Rousset (2004, chapter 3) or in Grimmet and Stirzaker (2001).

Substituting equation (6) into equation (4), evaluating the derivatives at $y_\bullet = \ldots = y^R_{\mathbf{k},t} = \ldots = 0$) and rearranging reveals S can be written as

$$S = s \left(1 - \sum_{\mathbf{i}} \sum_{\mathbf{j}} m_{\mathbf{i}} m_{\mathbf{i}-\mathbf{j}} Q^R_{\mathbf{j},0} \right) + \sum_{t=0}^{\infty} \sum_{\mathbf{k}} s_{\mathbf{k},t} \left(Q_{\mathbf{k},t} - \sum_{\mathbf{i}} \sum_{\mathbf{j}} m_{\mathbf{i}} m_{\mathbf{i}-\mathbf{j}} Q^R_{\mathbf{j}-\mathbf{k},t} \right), \tag{A8}$$

where

$$Q^R_{\mathbf{k},t} = Q_{\mathbf{k},t} \text{ except that } Q^R_{\mathbf{0},0} = \frac{1}{N} + \left(\frac{N-1}{N} \right) Q_{\mathbf{0},0}. \tag{A9}$$

Because dispersal and fitness effects are assumed to be spatially homogeneous, we have $m_{\mathbf{k}} = m_{-\mathbf{k}}$, $s_{\mathbf{k},t} = s_{-\mathbf{k},t}$, and $Q_{\mathbf{k},t} = Q_{-\mathbf{k},t}$.

Decomposing S into contemporary and intertemporal fitness effects yields

$$S = S_0 + S_T, \tag{A10}$$

where

$$S_0 \equiv s \left(1 - \sum_{\mathbf{i}} \sum_{\mathbf{j}} m_{\mathbf{i}} m_{\mathbf{i}-\mathbf{j}} Q^R_{\mathbf{j},0} \right) + \sum_{\mathbf{k}} s_{\mathbf{k},0} \left(Q_{\mathbf{k},0} - \sum_{\mathbf{i}} \sum_{\mathbf{j}} m_{\mathbf{i}} m_{\mathbf{i}-\mathbf{j}} Q^R_{\mathbf{j}-\mathbf{k},0} \right) \tag{A11}$$

gathers all contemporary fitness effects and is similar to equation (7.19) of Rousset (2004), and

$$S_T \equiv \sum_{t=1}^{\infty} \sum_{\mathbf{k}} s_{\mathbf{k},t} \left(Q_{\mathbf{k},t} - \sum_{\mathbf{i}} \sum_{\mathbf{j}} m_{\mathbf{i}} m_{\mathbf{i}-\mathbf{j}} Q_{\mathbf{j}-\mathbf{k},t} \right) \tag{A12}$$

which gathers all intertemporal fitness effects.

Substituting the equilibrium values of the spatial probabilities of identity (eq. A24) into equation (A11), the contemporary inclusive fitness effect simplifies to

$$S_0 = s(1 - Q_{\mathbf{0},0}/\gamma) + \sum_{\mathbf{k}} s_{\mathbf{k},0}(Q_{\mathbf{k},0} - Q_{-\mathbf{k},0}/\gamma), \tag{A13}$$

where $\gamma \equiv (1-\mu)^2$ denotes the probability that two genes have not mutated during reproduction. This equation can equivalently be written as

$$S_0 = (1 - Q_{\mathbf{0},0}) \left[s \left(\frac{1}{\gamma} - \frac{1-\gamma}{\gamma(1 - Q_{\mathbf{0},0})} \right) - \left(\frac{1-\gamma}{\gamma(1 - Q_{\mathbf{0},0})} \right) \sum_{\mathbf{k}} s_{\mathbf{k},0} Q_{\mathbf{k},0} \right]. \tag{A14}$$

Substituting the equilibrium values of the space-time probabilities of identity (eq. A32) into equation (A12), the intertemporal inclusive fitness effect can be expressed as

$$S_T = (1 - Q_{\mathbf{0},0}) \sum_{t=1}^{\infty} \sum_{\mathbf{k}} \sum_{\mathbf{h}} s_{\mathbf{k},t} \frac{\sqrt{\gamma}^t \psi_{\mathbf{h}}^t}{n_{\mathrm{d}} N \left(1 - \gamma \psi_{\mathbf{h}}^2\right)} \times \left(e^{-\imath \mathbf{k} \cdot \theta(\mathbf{h})} - \sum_{\mathbf{i}} \sum_{\mathbf{j}} m_{\mathbf{i}} m_{\mathbf{i}-\mathbf{j}} e^{-\imath(\mathbf{k}-\mathbf{j}) \cdot \theta(\mathbf{h})} \right), \tag{A15}$$

where $\psi_{\mathbf{h}} \equiv \sum_{\mathbf{i}} m_{\mathbf{i}} e^{\imath \mathbf{i} \cdot \theta(\mathbf{h})}$ is the characteristic function of the dispersal distribution with $\imath \equiv \sqrt{-1}$ and $\theta(\mathbf{h}) \equiv 2\pi(h_x/n_x,\ h_y/n_y)$. The vector representations of $\theta(\mathbf{h})$ and of the spatial coordinates $\mathbf{i}$ were introduced to carry out the analysis in a 2D model. But all the following calculations apply directly to a 1D model as well, in which case the characteristic function of the dispersal distribution becomes $\psi_h \equiv \sum_i m_i e^{\imath i \theta(h)}$ with $\theta(h) \equiv 2\pi h/n_{\mathrm{d}}$, and all the sums in equation (A15) are then taken over the lattice points of a one-dimensional lattice.

The second term in the parentheses of equation (A15) can be expressed as

$$\begin{aligned} &\sum_{\mathbf{i}} \sum_{\mathbf{j}} m_{\mathbf{i}} m_{\mathbf{i}-\mathbf{j}} e^{-\imath(\mathbf{k}-\mathbf{j}) \cdot \theta(\mathbf{h})} \\ &= \sum_{\mathbf{i}} \sum_{\mathbf{j}} m_{\mathbf{i}} m_{\mathbf{i}-\mathbf{j}} e^{-\imath(\mathbf{k}-\mathbf{j}-\mathbf{i}+\mathbf{i}) \cdot \theta(\mathbf{h})} \\ &= \sum_{\mathbf{i}} \sum_{\mathbf{j}} m_{\mathbf{i}} m_{\mathbf{i}-\mathbf{j}} e^{\imath \mathbf{i} \cdot \theta(\mathbf{h})} e^{-\imath(\mathbf{i}-\mathbf{j}) \cdot \theta(\mathbf{h})} e^{-\imath \mathbf{k} \cdot \theta(\mathbf{h})} \\ &= \psi_{\mathbf{h}}^2 e^{-\imath \mathbf{k} \cdot \theta(\mathbf{h})}. \end{aligned} \tag{A16}$$

With this equality, equation (A15) becomes

$$S_T = (1 - Q_{\mathbf{0},0}) \left(\sum_{t=1}^{\infty} \sum_{\mathbf{k}} \sum_{\mathbf{h}} s_{\mathbf{k},t} \frac{\sqrt{\gamma}^t \psi_{\mathbf{h}}^t \left(1 - \psi_{\mathbf{h}}^2\right)}{n_{\mathrm{d}} N \left(1 - \gamma \psi_{\mathbf{h}}^2\right)} e^{-\imath \mathbf{k} \cdot \theta(\mathbf{h})} \right). \tag{A17}$$

Adding up equations (A14) and (A17), substituting the result into equation (3), using equation (A26) and recalling that when $\mu \to 0$ one has $\gamma \to 1$ and $Q_{-\mathbf{k},0} \to 1$, the effect of the mutant on its probability of fixation is then given by

$$\phi = s - \frac{1}{N n_{\mathrm{d}}} \left(s + \sum_{\mathbf{k}} s_{\mathbf{k},0} \right) + \sum_{t=1}^{\infty} \sum_{\mathbf{k}} \frac{s_{\mathbf{k},t}}{N} \sum_{\mathbf{h}} \lim_{\mu \to 0} \left(\frac{\sqrt{\gamma}^t \psi_{\mathbf{h}}^t \left(1 - \psi_{\mathbf{h}}^2\right)}{n_{\mathrm{d}} \left(1 - \gamma \psi_{\mathbf{h}}^2\right)} \right) e^{-\imath \mathbf{k} \cdot \theta(\mathbf{h})}. \tag{A18}$$

The limit in the last sum can be taken by noting first that $\psi_{\mathbf{0}} = \sum_{\mathbf{i}} m_{\mathbf{i}} e^0 = \sum_{\mathbf{i}} m_{\mathbf{i}} = 1$. Thereby

$$\begin{aligned} &\sum_{\mathbf{h}} \lim_{\mu \to 0} \left(\frac{\sqrt{\gamma}^t \psi_{\mathbf{h}}^t \left(1 - \psi_{\mathbf{h}}^2\right)}{n_{\mathrm{d}} \left(1 - \gamma \psi_{\mathbf{h}}^2\right)} \right) e^{-\imath \mathbf{k} \cdot \theta(\mathbf{h})} \\ &= \frac{1}{n_{\mathrm{d}}} \sum_{\mathbf{h} \neq \mathbf{0}} \psi_{\mathbf{h}}^t e^{-\imath \mathbf{k} \cdot \theta(\mathbf{h})} \\ &= \frac{1}{n_{\mathrm{d}}} \sum_{\mathbf{h} \neq \mathbf{0}} \psi_{\mathbf{h}}^t e^{-\imath \mathbf{k} \cdot \theta(\mathbf{h})} + \frac{\psi_{\mathbf{0}}^t}{n_{\mathrm{d}}} - \frac{1}{n_{\mathrm{d}}} = P_{\mathbf{k},t} - \frac{1}{n_{\mathrm{d}}}, \end{aligned} \tag{A19}$$

where

$$P_{\mathbf{k},t} = \frac{1}{n_{\mathrm{d}}} \sum_{\mathbf{h}} \psi_{\mathbf{h}}^t e^{-\imath \mathbf{k} \cdot \theta(\mathbf{h})} \tag{A20}$$

is the inverse Fourier transform of the t's fold convolution of the characteristic function $\psi_{\mathbf{h}}$ of the dispersal distribution. This is the probability that a random line of genes descending from an individual residing in the focal individual will be in deme $\mathbf{k}$ at t generations posterior to the focal generation ($\sum_{\mathbf{k}} P_{\mathbf{k},t} = 1$). With this result, the effect of the mutant on its probability of fixation finally becomes

$$\phi = s + \frac{1}{N} \sum_{t=1}^{\infty} \sum_{\mathbf{k}} s_{\mathbf{k},t} \left(P_{\mathbf{k},t} - \frac{1}{n_{\mathrm{d}}} \right) - \frac{1}{N n_{\mathrm{d}}} \left(s + \sum_{\mathbf{k}} s_{\mathbf{k},0} \right). \tag{A21}$$

This equation shows that if the population is panmictic, that is, if the dispersal distribution is uniform ($m_{\mathbf{k}} = 1/n_{\mathrm{d}}$) so that $P_{\mathbf{k},t} = 1/n_{\mathrm{d}}$ for all deme $\mathbf{k}$ and time t, all intertemporal effects on fitness vanish. Consequently, in this case, equation (A21) boils down to

$$\phi = s - \frac{1}{N n_{\mathrm{d}}} \left(s + \sum_{\mathbf{k}} s_{\mathbf{k},0} \right), \tag{A22}$$

which is equivalent to equation (7.21) of Rousset (2004).

Alternatively, if we assume that the number of demes becomes very large (say $n_d \to \infty$), the last term in equation (A21) will vanish. In that case, classical results show that the normal distribution function is a good approximation to equation (A20) for large t and for any dispersal distribution (Grimmet and Stirzaker 2001). In one dimension, this is

$$P_{k,t} \approx \frac{e^{\frac{-k^2}{2\sigma^2 t}}}{\sqrt{2\pi\sigma^2 t}} \tag{A23}$$

where $\sigma^2 = \mathrm{E}[k^2]$ is the variance of the axial distance of dispersal, a measure of the speed at which a line of descent moves away from its ancestor. The closer the dispersal distribution is to a normal distribution, the better the approximation.

PROBABILITIES OF IDENTITY

In this appendix, I describe how to evaluate probabilities of identity by descent between pairs of homologous genes sampled in individuals residing at different spatial and temporal locations. In so doing, I follow the lead of Epperson (1999, 2003).

Spatial probabilities of identity

The probability of identity between pairs of homologous genes sampled in two different adult individuals living at $\mathbf{k}$ steps apart on the lattice in the same generation satisfies at steady state the recursion

$$Q_{\mathbf{k},0} = \gamma \sum_{\mathbf{i}} \sum_{\mathbf{j}} m_{\mathbf{i}} m_{\mathbf{i}-\mathbf{j}} Q^{\mathrm{R}}_{\mathbf{k}-\mathbf{j},0}, \tag{A24}$$

where $Q^{\mathrm{R}}_{\mathbf{k}-\mathbf{j},0} = Q_{\mathbf{k}-\mathbf{j},0}$ except that $Q^{\mathrm{R}}_{\mathbf{0},0} = (1 + (N-1)Q_{\mathbf{0},0})/N$ (e.g., eq. A9). Each term of the sum is obtained as the probability that two individuals residing at $\mathbf{k} - \mathbf{j}$ steps from each other before dispersal will be residing at $\mathbf{k}$ steps from each other after dispersal (e.g., Malécot 1975, eq. 2; Epperson 2003, eq. 5.5; Rousset 2004, eq. 3.47). Standard formulae (e.g., Malécot 1975; Rousset 2004) show that the characteristic function of the probabilities of identity $\mathcal{Q}(\mathbf{h}) \equiv \sum_{\mathbf{i}} Q_{\mathbf{i},0} e^{I\mathbf{i}\cdot\theta(\mathbf{h})}$, where $\theta(\mathbf{h}) \equiv 2\pi(h_x/n_x, h_y/n_y)$, can be expressed as a function of the characteristic function $\psi_{\mathbf{h}} \equiv \sum_{\mathbf{i}} m_{\mathbf{i}} e^{I\mathbf{i}\cdot\theta(\mathbf{h})}$ of the dispersal distribution, as

$$\mathcal{Q}(\mathbf{h}) = \frac{(1 - Q_{\mathbf{0},0})}{N} \frac{\gamma\psi_{\mathbf{h}}^2}{\left(1 - \gamma\psi_{\mathbf{h}}^2\right)}. \tag{A25}$$

Finally, I mention that from the recursions for the probabilities of identity (eq. A24) one can establish a useful formula, which will be helpful in several calculations, and that relates the diversity in a deme $(1 - Q_{\mathbf{0},0})$ to total population size Nn_d by the equality

$$\lim_{\mu \to 0} \frac{1-\gamma}{1 - Q_{\mathbf{0},0}} = \frac{1}{n_d N} \tag{A26}$$

(Rousset, 2004, eq. 3.68).

Space-time probabilities of identity

The probability $Q_{\mathbf{k},t}$ that a gene sampled in a focal individual residing in a focal deme in a focal generation is identical by descent with a homologous gene sampled from an individual chosen at random in deme $\mathbf{k}$ at t generations prior to the focal generation satisfies for $t \geq 2$ the recursion

$$Q_{\mathbf{k},t} = \sqrt{\gamma} \sum_{\mathbf{i}} m_{\mathbf{i}} Q_{\mathbf{k}-\mathbf{i},t-1}. \tag{A27}$$

This equation is obtained by noting that with probability $m_{\mathbf{i}}$ the focal individual has migrated $\mathbf{i}$ steps from its natal deme, in which case a gene sampled in its parent (probability of identity of 1) has a probability of identity $Q_{\mathbf{k}-\mathbf{i},t-1}$ with a homologous gene sampled in an individual chosen at random from position $\mathbf{k} - \mathbf{i}$ relative to the parental deme at $t-1$ generations prior to the parental generation. The boundary condition of the system of equations (A27) is obtained be evaluating the probability of identity $Q_{\mathbf{k},1}$ between a gene sampled in a focal individual and a homologous gene sampled from an individual of the parental generation chosen at random from deme $\mathbf{k}$. This is

$$\begin{aligned} Q_{\mathbf{k},1} &= \sqrt{\gamma} \sum_{\mathbf{i}} m_{\mathbf{i}} Q^{\mathrm{R}}_{\mathbf{k}-\mathbf{i},0} \\ &= \sqrt{\gamma} \left(\sum_{\mathbf{i}} m_{\mathbf{i}} Q_{\mathbf{k}-\mathbf{i},0} + m_{\mathbf{k}} \frac{(1 - Q_{\mathbf{0},0})}{N} \right), \end{aligned} \tag{A28}$$

because with probability $m_{\mathbf{k}}$ the focal individual originates from the same deme as the individual sampled from the parental generation, in which case the individual of the parental generation is the parent of the focal individual with probability $1/N$.

Call $\mathcal{Q}(\mathbf{h})_t \equiv \sum_{\mathbf{k}} Q_{\mathbf{k},t} e^{I\mathbf{k}\cdot\theta(\mathbf{h})}$ the characteristic function of the probabilities of identity between pairs of genes sampled at t generation of interval (for $t > 0$). Then, by Fourier transforming equation (A27), one has

$$\begin{aligned} \mathcal{Q}(\mathbf{h})_t &= \sum_{\mathbf{k}} \sqrt{\gamma} \sum_{\mathbf{i}} m_{\mathbf{i}} Q_{\mathbf{k}-\mathbf{i},t-1} e^{I\mathbf{k}\cdot\theta(\mathbf{h})} \\ &= \sqrt{\gamma} \sum_{\mathbf{i}} \sum_{\mathbf{k}} m_{\mathbf{i}} e^{I\mathbf{i}\cdot\theta(\mathbf{h})} Q_{\mathbf{k}-\mathbf{i},t-1} e^{I(\mathbf{k}-\mathbf{i})\cdot\theta(\mathbf{h})} \\ &= \sqrt{\gamma}\psi_{\mathbf{h}} \mathcal{Q}(\mathbf{h})_{t-1}, \end{aligned} \tag{A29}$$

which gives a recursion on $\mathcal{Q}(\mathbf{h})_t$. The solution is $\mathcal{Q}(\mathbf{h})_t = \sqrt{\gamma}^t \psi_{\mathbf{h}}^t \mathcal{Q}(\mathbf{h})_1$, where the boundary condition $\mathcal{Q}(\mathbf{h})_1$ is obtained by Fourier transforming equation (A28), which gives

$$\mathcal{Q}(\mathbf{h})_1 = \sqrt{\gamma}\psi_{\mathbf{h}} \left(\mathcal{Q}(\mathbf{h}) + \frac{(1 - Q_{\mathbf{0},0})}{N} \right). \tag{A30}$$

Hence, for $t \geq 1$

$$\mathcal{Q}(\mathbf{h})_t = \sqrt{\gamma}^t \psi_{\mathbf{h}}^t \left(\mathcal{Q}(\mathbf{h}) + \frac{(1 - Q_{\mathbf{0},0})}{N} \right), \tag{A31}$$

from which the stationary probability of identity $Q_{\mathbf{k},t}$ for $t \geq 1$ can be obtained as

$$Q_{\mathbf{k},t} = \frac{(1 - Q_{\mathbf{0},0})}{N}\mathcal{L}_{\mathbf{k}}(\mathcal{F}), \tag{A32}$$

where

$$\mathcal{L}_{\mathbf{k}}(\mathcal{F}) = \frac{1}{n_{\mathrm{d}}}\sum_{\mathbf{h}}\mathcal{F}(\mathbf{h})e^{-I\mathbf{k}\cdot\theta(\mathbf{h})} \tag{A33}$$

is the inverse Fourier transform at distance $\mathbf{k}$ of the function

$$\mathcal{F}(\mathbf{h}) = \frac{\sqrt{\gamma}^{t}\psi_{\mathbf{h}}^{t}}{1 - \gamma\psi_{\mathbf{h}}^{2}}. \tag{A34}$$

APPLICATION: RESOURCE CONSERVATION

In this appendix, I derive the explicit selective pressure on a niche constructing trait involved in the consumption of an abiotic resource.

Inclusive fitness effect of the attack rate

From equation (14) in the main text, the fecundity of a focal individual engaged in the consumer–resource system is given by

$$b(z_{\bullet}, \mathbf{z}) = 1 + \sum_{j=0}^{\infty}\mathcal{E}(\mathbf{z}_j)\beta_j a(1 - z_{\bullet}), \tag{A35}$$

where the resource values are obtained from equations (A50) and (A54), namely

$$\mathcal{E}(\mathbf{z}_j) = \sum_{x=1}^{\infty}\prod_{t=1}^{x-1} r\left(1 - \mu - \sum_{k=0}^{\infty}\beta_{j-k}a(1 - z_{k,t})N\right). \tag{A36}$$

From these two expressions, and using equations (A55) and (A56), yields

$$s \equiv \frac{1}{b(z,\mathbf{z})}\frac{\partial b(z_{\bullet},\mathbf{z})}{\partial z_{\bullet}}\bigg|_{z_{\bullet}=\ldots=z_{k,t}=\ldots=0} = -\frac{a\mathcal{E}}{1 + a\mathcal{E}}. \tag{A37}$$

and

$$\begin{aligned} s_{k,t} &\equiv \frac{1}{b(z,\mathbf{z})}\frac{\partial b(z_{\bullet},\mathbf{z})}{\partial z_{k,t}}\bigg|_{z_{\bullet}=\ldots=z_{k,t}=\ldots=0} \\ &= \frac{a}{1 + a\mathcal{E}}\sum_{j=0}^{\infty}\beta_j\frac{\partial\mathcal{E}(\mathbf{z}_j)}{\partial z_{k,t}} \\ &= \frac{a(r\alpha + \mathcal{E})}{1 + a\mathcal{E}}\sum_{j=0}^{\infty}(1 - \mu - aN)^{t-1}\beta_j\beta_{j-k}aN. \end{aligned} \tag{A38}$$

Substituting the fecundity effects into equation (10), one has

$$\begin{aligned} S = s + \frac{1}{N}\sum_{t=1}^{\infty}\sum_{k}s_{k,t}P_{k,t} = \frac{a}{1 + a\mathcal{E}}\Bigg(&-\mathcal{E} + a\,(r\alpha + \mathcal{E}) \\ &\times \sum_{t=1}^{\infty}(1 - \mu - aN)^{t-1}\sum_{k=0}^{\infty}\sum_{j=0}^{\infty}\beta_j\beta_{j-k}P_{k,t}\Bigg). \end{aligned} \tag{A39}$$

I will now approximate S by passing on to continuous space, and use equation (A23) for the spatiotemporal gene lineage distribution and

$$\beta_k = \frac{e^{\frac{-k^2}{2\omega^2}}}{\sqrt{2\pi\omega^2}} \tag{A40}$$

as interaction kernel. With these two functions, the inclusive fitness can be approximated as

$$\begin{aligned} S \approx \frac{a}{1 + a\mathcal{E}}\Bigg(&-\mathcal{E} + a(r\alpha + \mathcal{E})\sum_{t=1}^{\infty}(1 - \mu - aN)^{t-1} \\ &\times \int_{-\infty}^{-\infty}\int_{-\infty}^{-\infty}\frac{e^{-\frac{1}{2}\left(\frac{k^2}{\sigma^2 t} + \frac{j^2 + (j-k)^2}{\omega^2}\right)}}{2\pi\omega^2\sqrt{2\pi\sigma^2 t}}\,dj\,dk\Bigg). \end{aligned} \tag{A41}$$

Evaluating the double integral in the parentheses of equation (A41) with Mathematica (Wolfram 2003) gives $1/(\omega\sqrt{2\pi}\sqrt{2 + \sigma^2 t/\omega^2})$, and the remaining sum in the inclusive fitness can be written as

$$\frac{a}{\omega\sqrt{2\pi}}\sum_{t=1}^{\infty}\frac{(1 - \mu - aN)^{t-1}}{\sqrt{2 + t\frac{\sigma^2}{\omega^2}}} = \frac{a}{\sigma\sqrt{2\pi}}\sum_{t=0}^{\infty}\frac{(1 - \mu - aN)^{t}}{\sqrt{1 + t + 2\frac{\omega^2}{\sigma^2}}}. \tag{A42}$$

Taylor expanding the summand of the right-hand side around $x = \omega/\sigma$, when x becomes large, that is when the home range (ω^2) is larger than the speed at which genes move away from the focal deme (σ^2) yields

$$\begin{aligned} \frac{(1 - \mu - aN)^{t}}{\sqrt{1 + t + 2x^2}} = &\frac{(1 - \mu - aN)^{t}}{\sqrt{2}\,x} \\ &-\frac{(1 + t)\,(1 - \mu - aN)^{t}}{4\sqrt{2}\,x^3} + O\left(\frac{1}{x^4}\right). \end{aligned} \tag{A43}$$

With these approximations, the inclusive fitness becomes

$$\begin{aligned} S \approx \frac{a}{1 + a\mathcal{E}}\Bigg[&-\mathcal{E} + \frac{a\,(r\alpha + \mathcal{E})}{2\sqrt{\pi}(\mu + aN)\omega}\left(1 - \frac{\sigma^2}{4(\mu + aN)\omega^2}\right)\Bigg] \\ &+ O\left(\tfrac{1}{x^4}\right). \end{aligned} \tag{A44}$$

Setting $S > 0$ to find the condition under which the mutant invades and neglecting the residue $O(1/x^4)$, the mutant spreads when

$$\frac{\mathcal{E}}{r\alpha + \mathcal{E}} < \frac{a}{2\sqrt{\pi}(\mu + aN)\omega}\left(1 - \frac{\sigma^2}{4(\mu + aN)\omega^2}\right). \tag{A45}$$

Resource dynamics

To obtain the explicit values of $\mathcal{E}(\mathbf{z}_j)$, going into equations (14) and (A35), I derive in this section the recurrence equations for the dynamics of the environment. The functional form for the dynamics of the resource is very similar to those presented by Laland et al. (1999) and Odling-Smee et al. (2003).

The amount of an abiotic resource $\mathcal{E}_{h+1}$ in a focal deme at time $h+1$ is expressed as a function of the resources present in that deme at time h according to the equation

$$\mathcal{E}_{h+1} = \mathcal{E}_h - \mathcal{E}_h\mu - \mathcal{E}_h\left(\sum_{k=0}^{n_d-1}\beta_k g(z_{k,h})\right) + r\left(\sum_{k=0}^{n_d-1}\beta_k f(z_{k,h})\right). \tag{A46}$$

The righthand side consists of four terms: the number of resources available at time h, the amount of resources depleted naturally at rate μ, the amount of resources depleted due to all individuals consuming it, where $g(z_{k,h})$ represents the rate at which individuals with phenotype $z_{k,h}$ living in a deme at distance k from the focal deme consume the resource, and, finally, the replenishment of the resource occurring at a rate r, which is affected by the phenotype of the individuals in the population through the function f.

Solving the recurrence equation (A46) by standard methods gives

$$\mathcal{E}_h = \prod_{x=0}^{h-1}\left(1-\mu-\sum_{k=0}^{n_d-1}\beta_k g(z_{k,x})\right)\mathcal{E}_0 + \sum_{x=0}^{h-1} r\left(\sum_{k=0}^{n_d-1}\beta_k f(z_{k,x})\right)\prod_{t=x+1}^{h-1}\left(1-\mu-\sum_{k=0}^{n_d-1}\beta_k g(z_{k,t})\right), \tag{A47}$$

where $z_{k,i}$ is the average phenotype of an individual living at $h-i$ generations prior to h: if $i=0$, the individual lives in the period 0, whereas if $i=h-1$, the individual lives one generation before h. To evaluate the inclusive fitness effect, we need an expression for $\mathcal{E}_h$ where the phenotype $z_{k,i}$ can be interpreted as being the average phenotype of an individual living at i generations prior to h, namely if $i=1$, the individual lives one generation before h, whereas if $i=h$, the individual lives in the period 0. To that aim, equation (A47) will be expressed, by a change of variable, as

$$\mathcal{E}_h = \prod_{x=1}^{h}\left(1-\mu-\sum_{k=0}^{n_d-1}\beta_k g(z_{k,x})\right)\mathcal{E}_0 + \sum_{x=1}^{h} r\left(\sum_{k=0}^{n_d-1}\beta_k f(z_{k,x})\right)\prod_{t=1}^{x-1}\left(1-\mu-\sum_{k=0}^{n_d-1}\beta_k g(z_{k,t})\right), \tag{A48}$$

where $z_{k,i}$ now reads as the average phenotype of individuals living at i generations prior to h. Asymptotically ($h\to\infty$), the first line of this equation vanishes, and the whole expression becomes

$$\mathcal{E}(\mathbf{z}) = \sum_{x=1}^{\infty} r\left(\sum_{k=0}^{n_d-1}\beta_k f(z_{k,x})\right)\prod_{t=1}^{x-1}\left(1-\mu-\sum_{k=0}^{n_d-1}\beta_k g(z_{k,t})\right), \tag{A49}$$

which is the amount of resources in the focal deme expressed in terms of the average phenotypes of all those individuals living in the population in previous generations [$\mathbf{z}\equiv(\mathbf{z}_0,\mathbf{z}_1,\ldots,\mathbf{z}_t,\ldots)$ where $\mathbf{z}_t\equiv(z_{0,t},z_{1,t},\ldots,z_{k,t},\ldots)$]. From this equation we finally obtain the environment in a deme at distance j from the focal deme as

$$\mathcal{E}(\mathbf{z}_j) = \sum_{x=1}^{\infty} r\left(\sum_{k=0}^{n_d-1}\beta_k f(z_{j-k,x})\right)\prod_{t=1}^{x-1}\left(1-\mu-\sum_{k=0}^{n_d-1}\beta_k g(z_{j-k,t})\right). \tag{A50}$$

Using the chain rule and the product rule for finding the derivative of a function, the derivative of $\mathcal{E}(\mathbf{z}_j)$ with respect to $z_{k,t}$ and evaluated at $z_{0,0}=\ldots=z_{k,t}=\ldots=z$ is found to be

$$\left.\frac{\partial\mathcal{E}(\mathbf{z}_j)}{\partial z_{k,t}}\right|_{z_{k,t}=z} = r\beta_{j-k}(1-\mu-g(z))^{t-1} \times\left(\frac{\partial f(z_{k,t})}{\partial z_{k,t}} - \frac{f(z)}{\mu+g(z)}\frac{\partial g(z_{k,t})}{\partial z_{k,t}}\right)\Bigg|_{z_{k,t}=z} \tag{A51}$$

and the amount of the resource in a monomorphic population is given by

$$\mathcal{E}(z) = \frac{rf(z)}{\mu+g(z)}. \tag{A52}$$

With a functional response of Type I for the resource and from the assumptions of the main text, we have

$$g(z_{k,t}) = a(1-z_{k,t})N, \tag{A53}$$

and

$$f(z_{k,t}) = 1-\alpha a(1-z_{k,t})N, \tag{A54}$$

which, once substituted into equations (A51) and (A52), gives

$$\left.\frac{\partial\mathcal{E}(\mathbf{z}_j)}{\partial z_{k,t}}\right|_{z_{k,t}=0} = \beta_{j-k}\,(1-\mu-aN)^{t-1}\,aN(r\alpha+\mathcal{E}) \tag{A55}$$

where

$$\mathcal{E} = \frac{r\,(1-\alpha aN)}{\mu+aN}. \tag{A56}$$