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Implications of Habitat Choice for Protected Polymorphisms

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Contents

Abstract

In this paper we reexamine how heterogeneous environments can enable protected polymorphisms. Building on the classical models by Levene and Dempster of dispersal and selection in two habitats, we systematically investigate how the maintenance of polymorphisms is affected by (i) local versus global density regulation and (ii) constant versus variable output from habitats to the next generation. We show that, for populations capable of habitat choice, a third independent and fundamental class of models needs to be considered. It is characterized by local density regulation (like Levene's model) and variable habitat output (like Dempster's model). Our results indicate that the conditions determining whether a system allows for protected polymorphisms qualitatively differ in the presence and absence of matching habitat choice (which occurs when individuals prefer the habitat to which they are best adapted). Without such habitat choice, the salient distinction is not between local or global density regulation but rather between constant or variable habitat output. With matching habitat choice this situation is reversed. Analysis of the third class of models introduced here suggests that the joint evolution of matching habitat choice and local-adaptation polymorphism is easier than was previously understood.

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Implications of Habitat Choice for Protected Polymorphisms

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Introduction

A central question in evolutionary biology concerns the evolution of protected polymorphisms, i.e., the circumstances under which each of the alleles involved in a polymorphism can increase in frequency when starting out at low frequency. Clearly, the maintenance of such polymorphisms requires frequency-dependent selection at some level. Some traits are subject to frequency-dependent selection at a local level: their impact on viability or fecundity always depends on allelic frequencies. For instance, the scale-eating fish *Perissodus microlepis* in Africa's lake Tanganyika exhibits a protected genetic dimorphism. The 'left-handed' morph, whose mouth is twisted to the left, eats scales on its victims' right flank. By contrast, the right-handed morph, whose mouth is twisted to the right, eats scales on its victims' left flank. When one morph is more abundant than the other, the victims become more vigilant to attacks on the corresponding side, thereby rendering the other morph selectively advantageous (Hori 1993). Handedness in this species is thus under local frequency-dependent selection. Similarly, traits affecting competitive ability experience local density- and frequency-dependent selection: their demographic effects will depend not only on the number of competitors but also on these competitors' competitive ability. Explaining protected polymorphisms in traits under local frequency-dependent selection is straightforward: polymorphisms are protected whenever local selection creates an advantage of rarity, so that overly rare morphs can recover to their equilibrium frequency, and absent morphs can invade. However, not all traits experience local frequency-dependent selection. How can polymorphisms in these traits then be maintained? This is the key question addressed in this paper. One way to bring about frequency-dependent selection for such traits is to introduce a second level of selection. This is exactly what Levene (1953) did in his multiple-niche model, which we now describe.

Levene (1953) realized that environmental heterogeneity could be a major factor for the maintenance of protected polymorphisms. Dempster (1955) soon challenged this view. Both authors focused on a single population (with random mating in the diploid version of the models) whose environment is composed of several habitats. Within habitats, the trait under selection has a frequency-independent and habitat-dependent effect on viability (or fecundity). The two models assumed different life cycles (as we

will explain below). Polymorphism could be maintained in Levene's model, while it was much harder in Dempster's model. Since then, these two models have widely been recognized as prototypical cases of two different types of selection regime, which traditionally are referred to as soft and hard selection, respectively. It has repeatedly been shown that the conditions for the maintenance of polymorphism are far more stringent under hard selection than under soft selection (Christiansen 1975; Hedrick 1990b; Karlin and Campbell 1981; de Meeûs et al. 1993; van Tienderen 1997). Taking Levene's and Dempster's models as reference points, there has been considerable debate about what kind of life-cycle characteristics promote the emergence of frequency-dependent selection, and thus the potential for coexistence, in a single panmictic population. After decades of study, problems remain, however, when applying basic insights about hard and soft selection to previously unexplored ecological settings, since this requires a clear understanding of what formally distinguishes, in various specific cases, hard and soft selection. In this paper we take up this challenge: how can environmental heterogeneity promote coexistence of traits that cannot coexist without it?

It has been shown early on that matching habitat choice (occurring when individuals tend to prefer the habitat to which they are best adapted) could considerably broaden the conditions for the maintenance of stable polymorphisms (Maynard Smith 1966). This naturally led to examining the joint evolution of habitat choice and local adaptation (de Meeûs et al. 1993; Rausher 1994; Johnson et al. 1996; Kisdi 2002). Theoretical results in this area have recently been summarized as the "soft-selection/hard-selection dilemma" (de Meeûs 2000). While it has been shown that maintaining polymorphisms of local adaptations is only possible under soft selection (see Jaenike 1990 and Mayhew 1997 for reviews), the conditions for such polymorphism to occur under soft selection are quite stringent, except when matching habitat choice is already developed to an extent that it allows individuals to primarily inhabit the habitat they are best adapted to (de Meeûs et al. 1993), or if individuals are philopatric (Maynard Smith 1966; Diehl and Bush 1989; Meszéna et al. 1997; Geritz and Kisdi 2000; Kisdi 2002). Both of these cases, however, violate the assumption of initially considering a single panmictic population, and thus raise the question whether sufficient matching habitat choice or philopatry can ever evolve from scratch. It turns out that such evolution is only possible under two conditions: either a polymorphism for local adaptation already exists, or selection is hard (de Meeûs et al. 1993). Hence matching habitat choice favors the evolution of protected polymorphisms under soft selection, whereas such habitat choice can only develop under hard selection. These results thus suggest that the concomitant evolution of local-adaptation polymorphisms and matching habitat choice is not easy.

In this paper, we revisit Levene's and Dempster's classical work and show that when the process of habitat choice is integrated into the ecological setting, various widespread definitions of hard and soft selection lead to contradictory and misleading conclusions. In their currently used form, these two classes of models essentially differ in the nature of density regulation (local in Levene's model versus global in Dempster's model) and in the contribution of habitats to the next generation (constant in Levene's model versus variable in Dempster's model). In the present paper, we first point out that

Dempster's original work did not restrict attention to global regulation. The class of models now bearing his name has therefore been interpreted too narrowly. This observation naturally leads us to analyze a third possible class of models, which combine local density regulation (like in Levene's model) with variable habitat output (like in Dempster's model). We then show that the model properties that correlate with frequency dependence qualitatively differ in the presence and absence of matching habitat choice. Our study (1) helps clarifying what life-cycle characteristics promote the emergence of local-adaptation polymorphisms through frequency dependence and (2) offers a solution to de Meeûs's soft-selection/hard-selection dilemma, thereby opening up novel perspectives for the concurrent evolution of local adaptation and habitat choice. We finally discuss the implications of our findings for the evolution of specialization.

Classical dispersal-selection models

As a basis for our subsequent analysis, let us recall Levene's and Dempster's models and their main implications. The model of Levene (1953) is illustrated in Figure 1a and is characterized by a periodic sequence of steps as follows:

1. Dispersal from a large pool of zygotes into two different habitats (in the original model of Levene, there are two or more habitats, or "niches").
2. Local genotype-dependent and density-independent viability selection within habitats.
3. Local density-dependent and genotype-independent density regulation within habitats (such that each habitat contributes a constant number of individuals to the next step).
4. Formation of a common pool and genotype-independent reproduction (with random mating in the diploid version of the model).

Thus, in Levene's model density regulation is local and, within each habitat, occurs after selection. This implies that, with respect to the trait considered, population sizes just before pooling are independent of the genetic composition of populations in the two habitats. This will be called 'constant output' in the following. This selection scheme has been named 'soft selection' by numerous authors (e.g., Christiansen 1974, 1975; Karlin and Campbell 1981; Karlin 1982; Walsh 1984; Via and Lande 1985; Rausher 1994; Maynard Smith 1998; Whitlock 2002). Because realized fitness decreases with habitat density and because habitat density just before regulation depends on the genetic composition in the habitat, selection is frequency-dependent. The fitness of an individual with a given genotype therefore depends on the composition of the (habitat) group it belongs to. This can be seen as a form of group selection according to the definition of Wade (1985; Damuth and Heisler 1988; Goodnight and Wade 2000; but see the alternative view of Nunney 1985).

An alternative class of two-habitat dispersal-selection models was introduced verbally by Dempster (1955). This class of models is illustrated in Figure 1b and is characterized by a periodic sequence of steps as follows:

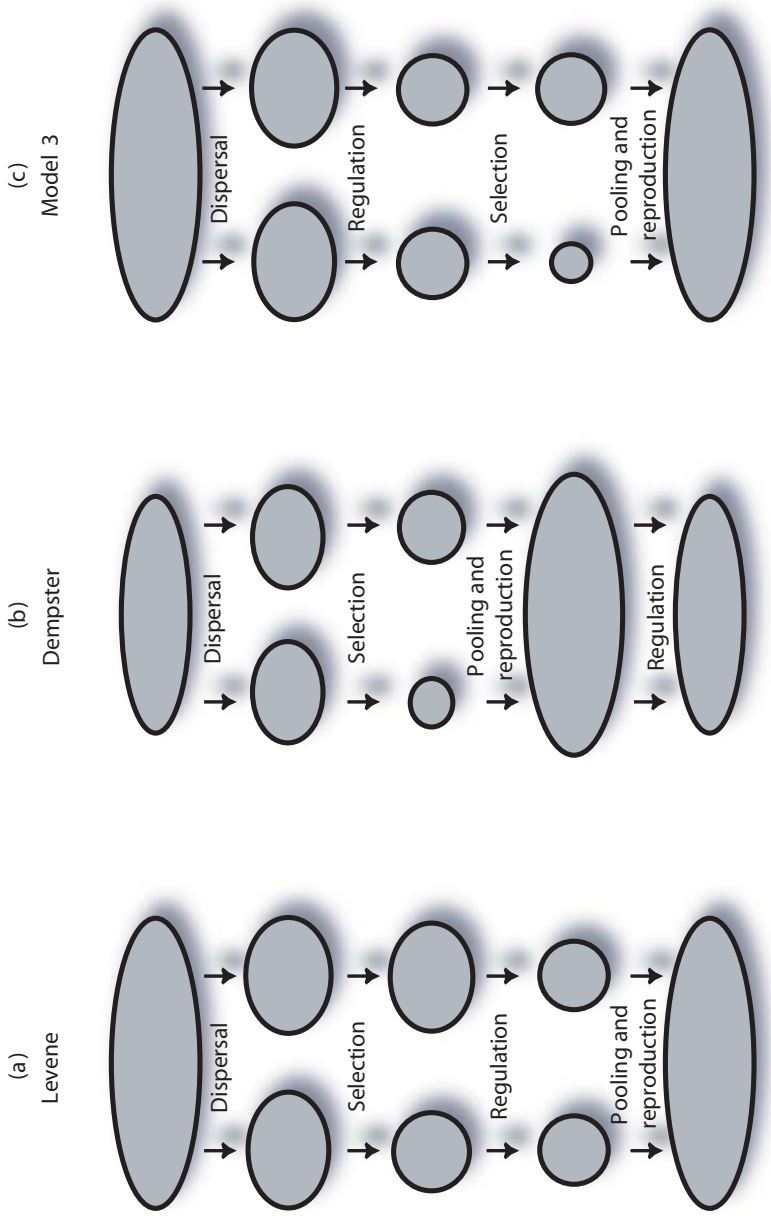

Figure 1. Life cycles in the three fundamental classes of dispersal-selection models. The models differ in the sequence of dispersal, selection, regulation, and pooling. This entails differences in whether density regulation is local or global, and whether habitat output is variable or constant. These distinctions, in turn, determine whether selection is frequency-dependent or not. At all stages, the sizes of grey areas schematically illustrate how the actual number of individuals in each habitat might change in the course of one model cycle.

1. Dispersal from a large pool of zygotes into two different habitats.
2. Local genotype-dependent and density-independent viability selection within habitats (such that each habitat contributes a variable number of individuals to the next step).
3. Formation of a common pool and genotype-independent reproduction (with random mating in the diploid version of the model).
4. Global density-dependent and genotype-independent density regulation within the pool.

This scheme implies that density regulation is global and that each habitat contributes individuals to the pool in proportion to their fitness. This is what is generally called 'hard selection' (e.g., Christiansen 1974, 1975; Karlin and Campbell 1981; Karlin 1982; Walsh 1984; Via and Lande 1985; Rausher 1994; Maynard Smith 1998; Whitlock 2002). Under such a selection regime, density regulation does not change genotype frequencies in the population. An individual's fitness therefore only depends on its own genotype and selection is thus based on absolute, rather than relative, fitness. Hard selection implies that fitness is frequency-independent. As the group of individuals in each habitat contributes to the next generation depending on the genotypic composition of individuals in that habitat, selection in this model has also been seen as a form of group selection according to the definition of Damuth (1985; for a review see Damuth and Heisler 1988).

Hard and soft selection

The terms hard and soft selection were imported by Wallace from the vocabulary of international monetary exchange (Wallace 1968, 1975). A country's currency is "soft" when, although being almost worthless as compared to other countries' currencies, it perfectly enables any kind of commercial or financial transaction within the country itself. The effective value of the currency thus depends on the context. Similarly, when the fitness of a genotype depends on the presence of other genotypes, selection is defined as soft. By contrast, hard selection occurs when the fitness of a genotype does not depend on the presence of other genotypes. More precisely, Wallace defined soft selection as frequency- and density-dependent and hard selection as frequency- and density-independent.

Applying Wallace's definition to dispersal-selection models, authors soon recognized Levene's and Dempster's models as characteristic examples of soft and hard selection regimes, respectively (Christiansen 1975; Karlin 1982). Subsequently, it seems, Levene's and Dempster's models have assumed the roles of prototypes, essentially defining which characteristics of an organism's life cycle determine whether the selection is soft (i.e., frequency-dependent) or hard.

Since Dempster's model has been widely interpreted as being globally regulated (e.g., Christiansen 1974; Karlin and Campbell 1981; Walsh 1984; de Meeûs et al. 1993), some authors have suggested that the distinction between hard and soft selection is synonymous to one between global and local density regulation, respectively (e.g., Christiansen 1974, 1985; de Meeûs et al. 1993; Rausher 1994; Kelly 1997; van

Tienderen 1997). Others, among them Dempster himself, pointed out that the critical difference between the models of Dempster and Levene amounted to assuming either a constant number of zygotes, or a constant number of fertile adults (Dempster 1955; Maynard Smith 1966; Christiansen 1975; Karlin and Campbell 1981; Maynard Smith 1998). Closer inspection shows, however, that hard selection does not really require a constant number of zygotes, but, instead, a variable number of adults contributing to the next generation. This is why the distinction between hard and soft selection has sometimes been rephrased as one between "variable output" versus "constant output," respectively (e.g., Via and Lande 1985; Whitlock 2002). Another way to put this is to stress that, under hard selection, selection affects population density just before pooling, whereas it does not do so under soft selection (Kisdi 2001). If the carrying capacities of habitats are constant through time, this further implies that, under hard selection, local population sizes fluctuate with allele frequencies, whereas they remain constant under soft selection (Karlin 1982).

Other authors have focused on the mechanistic origin of variable versus constant output in terms of the relative timing of population regulation and selection. Prout (1980) and later Sasaki and de Jong (1999) suggested that, with local density regulation, the temporal sequence of selection and regulation was key. In Sasaki and de Jong's model, selection was soft (i.e., frequency-dependent) when, between the consecutive rounds of pooling and dispersal, regulation occurred after selection, and hard otherwise. Evidently, the output from a habitat to the population-wide pool is constant if density regulation occurs just before pooling, and variable otherwise (Prout 1980). It has also been suggested that soft selection applies whenever selection and density regulation are mechanistically coupled and thus occur at the same time, whereas hard selection applies in the absence of such a coupling (Arnold and Anderson 1983; Ingvarsson 1999; de Meeûs and Goudet 2000). Similarly, since selection occurs just before pooling in Dempster's model and just after it in Levene's model, soft selection has sometimes been interpreted as juvenile selection, while hard selection supposedly operates on adults (Christiansen 1975; Karlin and Campbell 1981; Karlin 1982; Holsinger and Pacala 1990).

Confronted with this proliferation of definitions and interpretations, it must be realized that, as far as Levene's and Dempster's models are concerned, all the various distinctions discussed above can be reduced to only two salient dimensions. Density regulation is either global or local, and the output from habitats is either variable or constant (Fig. 1).

A third fundamental class of dispersal-selection models

The last section has clarified that, at least in the restricted context of simple two-habitat dispersal-selection models, there are two essential distinctions that appear to determine whether environmental heterogeneity renders selection frequency-dependent or not in specific models. These two salient dichotomies describe, respectively, whether density regulation is local or global and whether habitat output is constant or variable.

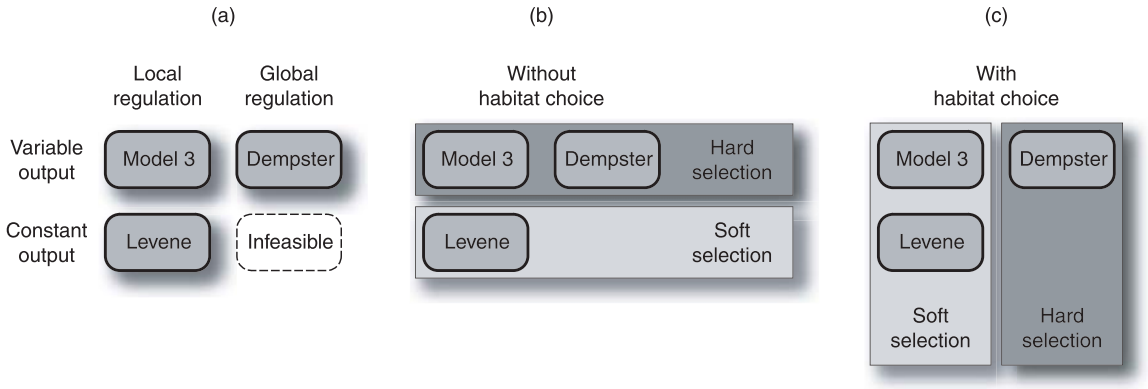

Figure 2. (a) The three fundamental classes of dispersal-selection models sorted according to whether density regulation is local or global (columns) and whether habitat output is variable or constant (rows). How these distinctions translate into whether selection is frequency-dependent or not critically depends on the absence or presence of matching habitat choice. (b) Without matching habitat choice, selection is frequency-independent (frequency-dependent) when habitat output is variable (constant). (c) By contrast, with matching habitat choice, selection is frequency-independent (frequency-dependent) when density regulation is global (local).

Figure 2a shows the $2\times 2=4$ combinatorial options arising from the two distinctions. Of these, one is infeasible: constant habitat output requires local density regulation and therefore cannot be assumed under global density regulation. Two other combinations directly correspond to Levene's model (local regulation, constant output) and to Dempster's model (global regulation, variable output). The fourth option is realized by a third class of two-habitat dispersal-selection models that has seldom been examined (but see Prout 1980; Sasaki and de Jong 1999). This class is illustrated in Figure 1c and is characterized by a periodic sequence of steps as follows:

1. Dispersal from a large pool of zygotes into two different habitats.
2. Local density-dependent and genotype-independent density regulation within habitats.
3. Local genotype-dependent and density-independent viability selection within habitats (such that each habitat contributes a variable number of individuals to the next step).
4. Formation of a common pool and genotype-independent reproduction (with random mating in the diploid version of the model).

This scheme is called "Model 3" in Figures 1 and 2 and is characterized by a combination of local density regulation and variable habitat output.

It is interesting to realize that Figure 1 indeed summarizes all possible two-habitat dispersal-selection models that can be meaningfully constructed from permuting the four processes of dispersal, selection, regulation, and pooling (Prout 1980). This can be seen as follows. Because all sequences are periodic, their first step can by fixed by convention; we have found it convenient to use the dispersal step for this purpose throughout Figure 1. Of the remaining $(4-1)!=6$ permutations, three are not feasible since selection in all considered models, being concerned with traits conferring local adaptation to alternative habitats, is local and thus cannot feasibly be preceded by pooling.

Is selection in Model 3 frequency-dependent or is it not? Below we show that the answer to this question depends on whether or not dispersal involves habitat choice.

Conditions for protected polymorphisms

To better understand the similarities and differences between the three fundamental dispersal-selection models highlighted above, we now focus on their implications for the coexistence of locally adapted populations.

Throughout, we consider a haploid species, with one diallelic locus (with alleles A and a) that pleiotropically determines both habitat choice and local viabilities. For simplicity, we only consider a case with two habitats, 1 and 2, that occur at frequencies c_1 and $c_2 = 1 - c_1$, respectively. The local viability of A individuals (resp. a individuals) is w_1 (resp. v_1) in habitat 1 and w_2 (resp. v_2) in habitat 2, with $w_1 > v_1$ and $w_2 < v_2$. In other words A individuals are fitter than a in habitat 1 and a individuals are fitter than A in habitat 2.

Let $h_{x,i}$ be the proportion of individuals of genotype $x = A, a$ that is in habitat $i = 1, 2$ after the dispersal/habitat choice step (therefore $h_{x,1} + h_{x,2} = 1$, assuming no genotype-dependent cost to choice). In the absence of any habitat choice, zygotes are distributed at random according to the frequencies at which the habitats occur,

$$\begin{aligned} h_{A,1} &= h_{a,1} = c_1 \\ h_{A,2} &= h_{a,2} = c_2 \end{aligned} . \tag{1a}$$

If, by contrast, habitat choice is strict, A individuals refuse to go to habitat 2. Thus they always find themselves in habitat 1 once the dispersal phase is over, whereas a individuals never go to habitat 1,

$$\begin{aligned} h_{A,1} &= h_{a,2} = 1 \\ h_{A,2} &= h_{a,1} = 0 \end{aligned} . \tag{1b}$$

It is useful to notice that $h_{x,i}$ is not a simple trait individuals express, like preference or acceptance. Instead, it must be envisaged as a complex phenotype resulting from all the processes occurring during the dispersal step, including mortality during dispersal. We use this so-called habitat-choice function because it allows a very general formulation that encompasses all possible habitat-choice processes. For instance, individuals can first land at random in habitats and then choose to stay or leave (acceptance). Alternatively, they can recognize their preferred habitat through visual or chemical cues and head for this habitat only. Individuals can be fertilized females choosing a habitat where to lay eggs or, alternatively, juveniles choosing a habitat where to develop. Finally, in the case of egg laying, dispersing females can either lay eggs in several habitats or lay all their eggs in the same clutch. In consequence, the habitat-choice functions depend on both the frequencies of habitats (c_i) and the underlying traits coding for habitat choice. With this approach, saying that $h_{x,i}$ evolves to a value h_0 means that the traits determining habitat choice are selected in such a way that the resulting probability for an individual with genotype x to reside in habitat i after dispersal and choice is h_0.

The frequencies of alleles A and a in the mixing pool are denoted by p and $q = 1 - p$, respectively. We are interested in the conditions for the robust maintenance of an allelic polymorphism and thus in the conditions for both alleles to be protected, which occurs when each allele can invade a population harboring only the other allele (Prout 1968).

Model 1: Levene's model with habitat choice (Dispersal – Selection – Regulation – Pooling)

As shown in Appendix I for Levene's model with habitat choice, allele A is protected if

$$c_1(h_{A,1}w_1 - h_{a,1}v_1)h_{a,2}v_2 + c_2(h_{A,2}w_2 - h_{a,2}v_2)h_{a,1}v_1 > 0 \ , \tag{2a}$$

and allele a is protected if

$$c_1(h_{a,1}v_1 - h_{A,1}w_1)h_{A,2}w_2 + c_2(h_{a,2}v_2 - h_{A,2}w_2)h_{A,1}w_1 > 0 \ . \tag{2b}$$

Levene's original model ignored habitat choice. The conditions above, when considered in the absence of habitat choice (see Eq. 1a) thus collapse to those found by Gliddon and Strobeck (1975) for the haploid version of Levene's model:

$$c_1 \frac{w_1}{v_1} + c_2 \frac{w_2}{v_2} > 1 \tag{3a}$$

and

$$c_1 \frac{v_1}{w_1} + c_2 \frac{v_2}{w_2} > 1 \ . \tag{3b}$$

By contrast, when assuming that habitat choice is strict (see Eq. 1b), it is clear that Conditions (2) are always fulfilled, because the same fraction of each genotype contributes to the pool in each generation. This confirms previously established results according to which, under a Levene-type life cycle, matching habitat choice broadens the conditions for which protected polymorphisms are expected (Maynard Smith 1966; Taylor 1976; Garcia-Dorado 1986, 1987; Hedrick 1990a, 1990b; de Meeûs et al. 1993; Johnson et al. 1996; see Hedrick 1986 for review).

Model 2: Dempster's model with habitat choice (Dispersal – Selection – Pooling – Regulation)

While Dempster's original model ignored habitat choice, one can expect that even when incorporating habitat choice into his model, the basic conclusion remains unaffected: local-adaptation polymorphisms are never protected (de Meeûs et al. 1993). Independently of whether there is habitat choice or not, the frequency of the allele specialized on the less productive habitat decreases in each generation.

Our calculations confirm this understanding (Appendix II). For Dempster's model with strict habitat choice the change in the frequency p of allele A in the mixing pool from one dispersal-selection cycle to the next is given by

$$\Delta p = pq\frac{(h_{A,1}w_1 + h_{A,2}w_2) - (h_{a,1}v_1 + h_{a,2}v_2)}{p(h_{A,1}w_1 + h_{A,2}w_2) + q(h_{a,1}v_1 + h_{a,2}v_2)} \ . \tag{4}$$

Since the sign of this change is independent of gene frequencies, selection leads to the fixation of either *A* or *a*, with the outcome only depending on the sign of the numerator above; no polymorphism can thus be maintained.

Model 3 (Dispersal – Regulation – Selection – Pooling)

For Model 3, we demonstrate in Appendix III that allele *A* is protected if

$$c_1 w_1 \frac{h_{A,1}}{h_{a,1}} + c_2 w_2 \frac{h_{A,2}}{h_{a,2}} > c_1 v_1 + c_2 v_2 \ , \tag{5a}$$

and, similarly, that allele *a* is protected if

$$c_1 v_1 \frac{h_{a,1}}{h_{A,1}} + c_2 v_2 \frac{h_{a,2}}{h_{A,2}} > c_1 w_1 + c_2 w_2 \ . \tag{5b}$$

We can now compare the conditions for protected polymorphisms to occur in the three classes of models considered in the absence and presence of habitat choice. Since, independently of habitat choice, no polymorphism can be maintained in Model 2, we focus on the comparison between Models 1 and 3.

The implications of habitat choice for the maintenance of polymorphisms

Matching habitat choice absent

Without matching habitat choice, individuals are distributed among habitats independently of local-adaptation genotypes (Eq. 1a). As shown above, in Model 1 protected polymorphisms can be maintained for a certain range of parameters.

In Model 3 without habitat choice, the sign of Δp for the change in allele frequency from one dispersal-selection cycle to the next is determined by the quantity $c_1(w_1 - v_1) + c_2(w_2 - v_2)$, which is independent of allele frequencies and similar to the expression obtained for Dempster's model in the absence of habitat choice. Thus no polymorphism can be maintained, and allele *A* (resp. *a*) will be fixed when this quantity is positive (resp. negative). Notice that if some constant habitat choice occurs that is independent of local adaptation, no polymorphism can be maintained either. We can thus conclude that, in the absence of matching habitat choice, whether habitat output is variable or constant determines the feasibility of protected polymorphisms (Fig. 2b).

Matching habitat choice present

In the presence of matching habitat choice, a necessary condition for protected polymorphisms in Models 1 and 3 is that $h_{A,1}w_1 - h_{a,1}v_1$ and $h_{A,2}w_2 - h_{a,2}v_2$ are of

opposite sign, see Equations (2) and (5). Since allele *A* confers local adaptation to habitat 1 and allele *a* to habitat 2 ($w_1 > v_1$ and $w_2 < v_2$), and provided that individuals prefer the habitat they are best adapted to ($h_{A,1} > h_{A,2}$ and $h_{a,1} < h_{a,2}$, which implies $h_{A,1} > h_{a,1}$ and $h_{A,2} < h_{a,2}$), we have $h_{A,1}w_1 > h_{a,1}v_1$ and $h_{A,2}w_2 < h_{a,2}v_2$, so that the two quantities above are then always of opposite sign. The conditions for protected polymorphism are then

$$\frac{h_{a,1}}{h_{a,2}}\frac{v_1}{v_2} < \frac{c_1}{c_2}\frac{h_{A,1}w_1 - h_{a,1}v_1}{h_{a,2}v_2 - h_{A,2}w_2} < \frac{h_{A,1}}{h_{A,2}}\frac{w_1}{w_2} \tag{6a}$$

for Model 1 and

$$\frac{h_{a,1}}{h_{a,2}} < \frac{c_1}{c_2}\frac{h_{A,1}w_1 - h_{a,1}v_1}{h_{a,2}v_2 - h_{A,2}w_2} < \frac{h_{A,1}}{h_{A,2}} \tag{6b}$$

for Model 3, with the left inequalities implying protection of allele *A* and the right ones protection of allele *a*.

Since $v_2 > v_1$, the left inequality is less restrictive for Model 1 than it is for Model 3. Consequently, if allele *A* is protected in Model 3, it is also protected in Model 1. Since $w_1 > w_2$, we can also conclude that if allele *a* is protected in Model 3, it is necessarily protected in Model 1. The conditions for polymorphism are thus more restrictive in Model 3 than in Model 1, so that the conditions for the maintenance of polymorphism of local adaptations in Model 3 are intermediate between those for Levene's and Dempster's models with matching habitat choice. We can thus conclude that, in the presence of matching habitat choice, the distinction whether density regulation is local or global is critical for determining the existence of protected polymorphisms (Fig. 2c).

The trait dependence of selection regimes

It is interesting to realize that the question as to which of the three model classes analyzed above most adequately matches the life cycle of a given organism can have different answers depending on which trait is considered.

To illustrate this point, let us consider a particular biological organism exposed to a seasonal environment: the lettuce root aphid *Pemphigus bursarius* (L.) can utilize two different habitats, soil and poplar trees, during winter, and feeds on lettuce and chicory leaves and roots during summer. Individuals have the option of spending the winter as so-called hiemalis in the soil; these are cold-tolerant and survive prolonged periods of starvation (Phillips et al. 2000). Alternatively, aphids can produce winged alatae in autumn. These then migrate to the aphid's primary host, poplar trees (Phillips et al. 1999).

As shown by Phillips et al. (2000), it is likely that the hiemalis individuals in the soil have evolved starvation resistance. Let us imagine that such resistance is selected against on poplar trees, possibly because of trade-offs with other traits selected there. We can then ask whether, under these circumstances (Selection A in Fig. 3), a population polymorphism for starvation resistance can be maintained, thus allowing for local adaptation to both soil and poplar. Since population densities are much reduced,

density-dependent regulation is unlikely to occur over the winter. Thus, with regard to poplar and soil habitats, regulation is not local with respect to these habitats, as it occurs outside of them. Therefore winter starvation resistance is experiencing frequency-independent selection as in Model 2, and thus no polymorphism can be expected.

In spring, individuals migrate to their secondary hosts, mainly lettuce and chicory (Pooling 2 and Dispersal 2 in Fig. 3). Although it is quite likely that hiemalis stay in the soil and directly re-infect the roots of germinating lettuce or chicory after winter, let us imagine that they first have to infect lettuce and chicory leaves. Assume that defense mechanisms against herbivory differ on lettuce and chicory. We can again assume that some trade-offs, here between the tolerance to secondary compounds produced by either lettuce or chicory, prevent the simultaneous optimization of aphid performance on both secondary hosts. A trait characterizing specialization to a summer host is likely to be expressed and selected for before density regulation on these hosts occurs (Selection B in Fig. 3). This means that, for this toxicity tolerance trait, the salient sequence of steps involves local density regulation and constant habitat output. The conditions for frequency-dependent selection as in Model 1 are thus met, independent of whether habitat choice is random or occurs according to local adaptation.

On their summer hosts, the aphids then reproduce clonally, and local density-dependent regulation occurs on each host (Regulation in Fig. 3). During the summer, the survivors colonize roots and start to feed on these. We thus consider a third quantitative trait that affects performance during this stage of the aphid's life cycle. This could be, for example, the ability to pierce tubes of the phloem tissues, or the efficiency of nutrient assimilation on lettuce or chicory roots. For any such utilization efficiency trait, the number of surviving aphids per plant, or the number of winged individuals produced per plant is larger for more efficient aphids (Selection C in Fig. 3). Under which conditions can a polymorphism be maintained for this third trait? In fact, such an efficiency trait experiences selection as described by Model 3. If spring colonization is independent of host type (no habitat choice), selection on the efficiency trait is frequency-independent, and thus no polymorphism can be maintained. By contrast, if there is habitat choice such that aphids tend to settle on the secondary host to which their efficiency trait is adapted, such a polymorphism could evolve. (The matching habitat choice required here is actually likely to evolve given that a polymorphism for toxicity tolerance can be maintained at any rate.)

Finally, the aphid's annual life cycle is concluded, either by re-colonization of their primary host or by the production of hiemalis that remain in the soil (Pooling 1 and Dispersal 1 in Fig. 3).

This example highlights that, depending on whether we focus on quantitative traits determining starvation resistance, toxicity tolerance, or utilization efficiency, the salient part of the aphid's life cycle is best described, respectively, by Models 2, 1, and 3.

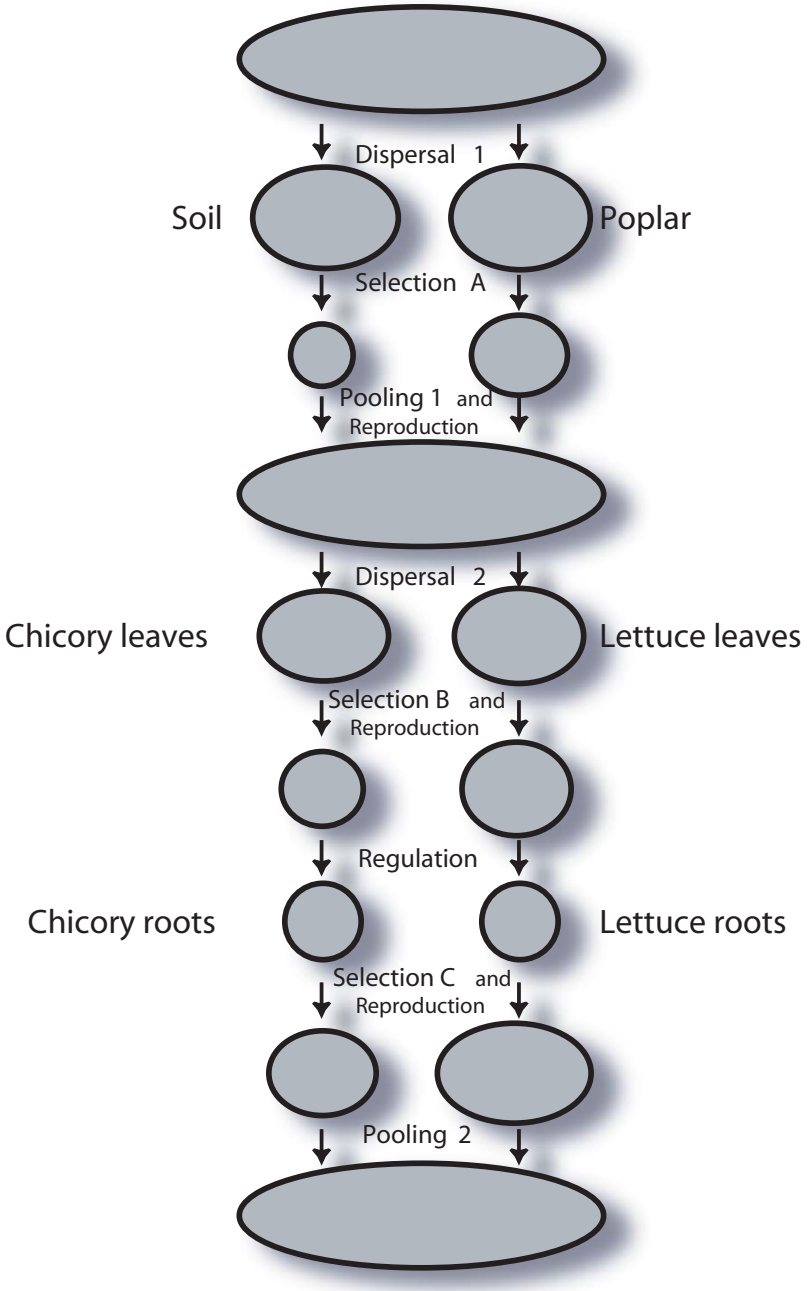

Figure 3. Life cycle in a more complex dispersal-selection model. Applied to the partly realistic example of the lettuce root aphid described in the text, the steps here correspond to the dispersal of sexual aphids to soil and poplar tree (Dispersal 1), selection of starvation resistance in the soil and against it on poplar trees (Selection A), dispersal to lettuce and chicory leaves (Pooling 2 and Dispersal 2), selection on resistance against lettuce or chicory toxicity (Selection B), asexual reproduction and local density regulation (Regulation), migration to the host's root system and selection on utilization efficiency there (Selection C), and production of hiemalis that remain in the soil or of winged individuals that disperse to polar trees (Pooling 1). At all stages, the sizes of grey areas schematically illustrate how the actual number of individuals in each habitat might change in the course of one model cycle. As detailed in the text, which one of the three fundamental classes of dispersal-selection models applies to such a life cycle, and, thus, whether selection is frequency-dependent or not, critically depends on which particular adaptive trait is considered to evolve.

Discussion

In this paper, we have investigated the effect of matching habitat choice on the maintenance of within-population variation for local adaptation in the three possible fundamental classes of dispersal-selection models. We have shown that Model 3, characterized by a combination of local density-regulation and variable habitat output, is closely related to Dempster's model in the absence of matching habitat choice, whereas it is akin to Levene's model when habitat choice occurs in accordance with local adaptation. More generally, we have shown that selection is frequency-independent if matching habitat choice is absent and habitat output is proportional to local fitness, or if matching habitat choice is present and density regulation is global. By contrast, selection is frequency-dependent if matching habitat choice is absent and habitat output is constant, or if matching habitat choice is present and density regulation is local. Model 3 can thus lead either to frequency-independent selection (without matching habitat choice) or to frequency-dependent selection (with matching habitat choice). In the presence of matching habitat choice, conditions for the maintenance of protected polymorphisms in Model 3 are intermediate between those resulting from Dempster's and Levene's models.

Selection is frequency-independent when density regulation does not change overall gene frequencies. This can happen when density regulation is global (as in Dempster's model) or when density regulation acts on habitats that feature the same local gene frequencies (Model 3 without matching habitat choice). By contrast, selection is frequency-dependent when density regulation changes global gene frequencies. This only happens when density regulation is local and acts on habitats differing in local gene frequencies. Local gene frequencies can differ either as a result of local selection (as in Levene's model) or because of differential habitat choice (Model 3 with matching habitat choice).

In the analysis presented here, mathematics, and thus the population dynamics that occur within habitats, were kept as simple as possible. Although the considered local dynamics, life cycles, and ecological settings are likely to be a good approximation of what really happens for some traits in some species (see the aphid example above), there will also be traits for which this simplified framework does not fit so well. It is therefore important that a careful analysis of the literature indicates that none of our assumptions seems to be critical for the qualitative results we found. Although a mathematical demonstration of this claim is clearly beyond the scope of this paper, it seems that the insights and criteria we have focused on here are generalizing well. The following paragraphs summarize the key considerations in support of a wider relevance of our findings.

First, the models presented here aim at investigating the evolution of stable polymorphisms in heterogeneous environments when it is the most problematic, i.e., in a single population in which individuals are distributed and pooled at each generation. Recently, Christiansen (2001) studied the case of several populations connected by migration (as in the well-known island model). He argued that the difference between selection under variable habitat output (hard selection) and under constant habitat output (soft selection) vanishes as populations approach complete isolation. Indeed, in the

model by Christiansen (2000, see also Christiansen 1975), relative population sizes are assumed to be either independent of or dependent on genotypic frequencies. This is similar to assuming that regulation is local and occurs either before or after selection. Christiansen assumed only partial mixing, i.e., some degree of philopatry, which is closely related to habitat choice. On that basis, Christiansen then finds, as we do in Model 3, that increasing philopatry allows the maintenance of polymorphism even in models with variable habitat output. Thus Christiansen's claim is congruent with our own results.

Second, in this paper we have shown that, in the absence of matching habitat choice, the key factor for determining whether or not polymorphisms can be maintained is not the coincidence of selection and regulation as it has sometimes been suggested (e.g., Ingvarsson 1999; de Meeûs and Goudet 2000), but, instead, the relative order of selection and regulation between the periodic dispersal events. Model 3 has seldom been examined, but Prout (1980) reported that an attempt to analyze Model 3 with random dispersal did not reveal anything new as compared to Dempster's work. Similar conclusions were reached by Sasaki and de Jong (1999), who studied the evolution of phenotypic plasticity in the context of more complex life cycles. Based on simulations of phenotypic evolution, Sasaki and de Jong found that reaction-norm polymorphisms could not evolve when regulation occurred before selection, whereas such polymorphisms could be maintained when regulation occurred after selection. What we have shown in this paper, however, is that this conclusion is restricted to models dealing with random habitat choice (which occurs, e.g., under passive dispersal), and no longer applies once matching habitat choice is taken into account.

Finally, one can ask how critical is the assumption, made in the tradition of Levene and Dempster, that by considering a single regulation step that simply eliminates excess density one can assess the implications of density-dependent demographic rates? In particular, we did not take density-dependent selection into account (which would occur when selection and regulation are simultaneous). More realistic population dynamics and/or density-dependent selection regimes have been incorporated and analyzed in models of soft and hard selection (Arnold and Anderson 1983; Christiansen 1985; Holsinger and Pacala 1990, Meszena et al. 1997; Kisdi 2000). Close inspection of all these results (analysis not shown) demonstrates that our conclusion of habitat selection being able to transform frequency-independent selection into frequency-dependent selection appears to be quite robust under variation of the population dynamics considered. Despite this general consistency, however, it must be realized that no general results of the kind discussed in this paper are currently available when population dynamics within habitats are arbitrary.

Among the few attempts to take into account more realistic ecological settings are models that analyze some mixture of the life cycles captured by Dempster's and Levene's model (De Meeûs and Goudet 2000; Lenormand 2002; Whitlock 2002). The common idea underlying these different models is that in natural structured populations, not all sub-populations are likely to be density-regulated. Because of environmental variability, and in particular due to local extinctions, some populations will be exponentially growing while others will have reached carrying capacity: these models

relax the hypothesis that all habitats are under the same selection-regulation regime. In Whitlock's (2002) approach, a local population's contribution to the next generation is a linear combination of constant and variable habitat output. Whitlock keeps the coefficients of such combination constant; in particular, they are independent of gene frequencies. Also de Meeûs and Goudet (2000) assume that a fixed proportion of the population is globally regulated, while the remaining proportion is locally regulated. In the same vein, Lenormand (2002) presents a two-patch model in which one patch can be considered as a source and is locally regulated, while the other is a sink and thus not regulated. Conditions for polymorphism protection have only been analyzed by de Meeûs and Goudet (2000). They showed that in their model transitory polymorphisms can be selected for under a wider range of conditions than is the case in Levene's model; such polymorphisms, however, are always lost in the long run, i.e., they are not evolutionarily stable. In all three models the proportion of non-regulated (or variable-output) habitats is constant. In reality, however, one would expect this proportion to depend on population dynamics, and thus on ecological factors such as carrying capacities, habitat frequencies, genotypes conferring local adaptation and their frequencies, extinction rates and recolonisation patterns, and thus also on habitat-choice genotypes and their frequencies.

The results presented here have important consequences for the understanding of adaptive processes underlying the evolution of specialization. It is well known that specialization is a two-fold process that can involve the evolution of habitat choice and the evolution of local adaptation. As emphasized earlier, studying the joint evolution of local adaptation and habitat choice has led to the formulation of the "soft-selection/hard-selection dilemma" (de Meeûs 2000). It stipulates that when selection is hard (i.e., frequency-independent), as in Dempster's model, habitat choice may evolve but local adaptations cannot coexist, whereas in Levene's model the matching habitat choice that broadens the conditions for the coexistence of local adaptations cannot evolve without such coexistence (de Meeûs et al. 1993).

This "chicken and egg" problem is overcome in Model 3. To see this, let us consider Model 3 with two diallelic haploid loci, one coding for local adaptation and the other for habitat choice. Just as before we assume that allele *A* at the local-adaptation locus confers a greater viability in habitat 1 than in habitat 2, while allele *a* confers a greater viability in habitat 2 than in habitat 1. With regard to the habitat-choice locus, *B* individuals choose the habitat they are best adapted to and *b* individuals distribute randomly. Matching habitat choice as exhibited by *B* individuals (implying a correlation between habitat choice and local adaptation) is known to occur, for instance, in alfalfa and clover aphids (Caillaud and Via 2000). We start the evolutionary process without a polymorphism in local adaptation (e.g., by assuming that allele *A* is fixed) and without any preferences in habitat choice (i.e., by assuming that allele *b* is fixed). Considering the life cycle of Model 3, selection is frequency-independent initially because habitat choice is absent and regulation occurs before selection such that habitat output is variable. When allele *a* is introduced in the population at low frequency (through mutation or immigration), it is either not protected and disappears, or it is protected and its frequency increases until *A* is eliminated (which of these outcomes applies depends

on the habitat frequencies and on the local viabilities of the two competing alleles). In the absence of *B*, therefore, no polymorphism at the local-adaptation locus can emerge. However, if *B* is introduced, because selection is frequency-independent, allele *B* is selected for at the habitat-choice locus, even in the absence of any polymorphism for local adaptation (de Meeûs et al. 1993; Ravigné et al. unpubl.). Notice that this outcome is specific to Models 2 and 3; it would not occur in a monomorphic population if selection occurred before regulation, as in Model 1. With allele *B* being fixed at the habitat-choice locus, all individuals have genotypes *AB* and all choose habitat 1: habitat 2 thus becomes an empty niche. Now, when allele *a* is (again) introduced, genotypes *aB* all disperse to habitat 2. When, as in Model 3, density regulation is local, these variant *aB* individuals enjoy a higher fitness than the resident *AB* individuals. It is clear that this outcome is prevented if regulation is global, as in Model 2. By contrast, in Model 3 selection has switched from frequency-independent to frequency-dependent and a polymorphism can now evolve at the local-adaptation locus, leading to the coexistence of two habitat specialists with differential habitat choice.

In the scenario just described we have assumed that the choice of habitat is based on preferences that are pleiotropically determined by the local-adaptation locus: an individual has either no preference or it chooses the habitat according to its genotype at the *A* locus. Although pleiotropy might be a plausible assumption (e.g., see Caillaud and Via 2000), it would also be interesting to investigate the evolution of specialization when local adaptation and habitat preferences can evolve fully independently. This is even more relevant because we have demonstrated in this paper that, in the presence of matching habitat choice, conditions for protected polymorphism to arise in Model 3 strongly depend on the actual habitat preferences. At least in principle it could thus be that, when local adaptations and habitat preferences evolve freely, the conditions for protected polymorphism are never met. Up to now, few studies have addressed the joint evolution in these two groups of traits (de Meeûs et al. 1993; Rausher 1994; Johnson et al. 1996; Kisdi 2002). We suggest such investigations as exciting opportunities for further research (see Kirkpatrick and Ravigné 2002 for a review on this topic in the context of sympatric speciation).

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Appendix I: Levene's model with matching habitat choice

In Model 1 (Fig. 1a), local regulation follows selection. Thus, just before pooling, the frequencies p_1' and q_1' of alleles A and a, respectively, are

$$p_1' = \frac{ph_{A,1}w_1}{ph_{A,1}w_1 + qh_{a,1}v_1} \ ,$$

$$q_1' = 1 - p_1'$$

in habitat 1, while those in habitat 2 are

$$p_2' = \frac{ph_{A,2}w_2}{ph_{A,2}w_2 + qh_{a,2}v_2} \ ,$$

$$q_2' = 1 - p_2' \ .$$

Since each habitat contributes in proportion to its frequency, allele frequencies after pooling are $p' = c_1 p_1' + c_2 p_2'$ and $q' = 1 - p'$, which gives

$$p' = c_1 \frac{ph_{A,1}w_1}{ph_{A,1}w_1 + qh_{a,1}v_1} + c_2 \frac{ph_{A,2}w_2}{ph_{A,2}w_2 + qh_{a,2}v_2} \ ,$$

$$q' = 1 - p' \ .$$

The change $\Delta p = p' - p$ in the frequency of allele A between consecutive rounds of pooling thus is

$$\Delta p = p \frac{c_1 h_{A,1}w_1(ph_{A,2}w_2 + qh_{a,2}v_2) + c_2 h_{A,2}v_2(ph_{A,1}w_1 + qh_{a,1}v_1) - (ph_{A,1}w_1 + qh_{a,1}v_1)(ph_{A,2}w_2 + qh_{a,2}v_2)}{(ph_{A,1}w_1 + qh_{a,1}v_1)(ph_{A,2}w_2 + qh_{a,2}v_2)} \ ,$$

which can be rewritten as

$$\Delta p = pq \frac{c_1(h_{A,1}w_1 - h_{a,1}v_1)(ph_{A,2}w_2 + qh_{a,2}v_2) + c_2(h_{A,2}w_2 - h_{a,2}v_2)(ph_{A,1}w_1 + qh_{a,1}v_1)}{(ph_{A,1}w_1 + qh_{a,1}v_1)(ph_{A,2}w_2 + qh_{a,2}v_2)} \ .$$

Thus Δp has the same sign as the quantity

$$c_1(h_{A,1}w_1 - h_{a,1}v_1)(ph_{A,2}w_2 + qh_{a,2}v_2) + c_2(h_{A,2}w_2 - h_{a,2}v_2)(ph_{A,1}w_1 + qh_{a,1}v_1) \ ,$$

and, by considering the invasion cases $p \approx 0$ and $p \approx 1$, we find the conditions for the protection of allele A,

$$c_1(h_{A,1}w_1 - h_{a,1}v_1)h_{a,2}v_2 + c_2(h_{A,2}w_2 - h_{a,2}v_2)h_{a,1}v_1 > 0 \ ,$$

and for the protection of allele a,

$$c_1(h_{a,1}v_1 - h_{A,1}w_1)h_{A,2}w_2 + c_2(h_{a,2}v_2 - h_{A,2}w_2)h_{A,1}w_1 > 0 \ .$$

Appendix II: Dempster's model with matching habitat choice

In Model 2 (Fig. 1b), pooling occurs just after selection, so that each habitat contributes to the pool according to its average fitness. After pooling, we therefore have

$$p' = p\frac{h_{A,1}w_1 + h_{A,2}w_2}{p(h_{A,1}w_1 + h_{A,2}w_2) + q(h_{a,1}v_1 + h_{a,2}v_2)} ,$$

$$q' = 1 - p'$$

and thus

$$\Delta p = pq\frac{h_{A,1}w_1 + h_{A,2}w_2 - h_{a,1}v_1 - h_{a,2}v_2}{p(h_{A,1}w_1 + h_{A,2}w_2) + q(h_{a,1}v_1 + h_{a,2}v_2)} .$$

Appendix III: Model 3 with matching habitat choice

In Model 3 (Fig. 1c), the allele frequencies after local regulation are

$$p_1 = \frac{ph_{A,1}}{ph_{A,1} + qh_{a,1}} ,$$

$$q_1 = 1 - p_1$$

in habitat 1 and

$$p_2 = \frac{ph_{A,2}}{ph_{A,2} + ph_{a,2}} ,$$

$$q_2 = 1 - p_2$$

in habitat 2. After selection, the allele frequencies are

$$p_1' = \frac{ph_{A,1}w_1}{ph_{A,1} + qh_{a,1}} ,$$

$$q_1' = \frac{qh_{a,1}v_1}{ph_{A,1} + ph_{a,1}}$$

in habitat 1 and

$$p_2' = \frac{ph_{A,2}w_2}{ph_{A,2} + qh_{a,2}}$$

$$q_2' = \frac{qh_{a,2}v_2}{ph_{A,2} + ph_{a,2}}$$

in habitat 2.

After pooling, weighing the contributions from each habitat by the habitat frequencies, the frequencies of alleles A and a are

$$p' = \frac{c_1 \dfrac{h_{A,1} w_1}{p h_{A,1} + q h_{a,1}} + c_2 \dfrac{h_{A,2} w_2}{p h_{A,2} + q h_{a,2}}}{c_1 \dfrac{p h_{A,1} w_1 + q h_{a,1} v_1}{p h_{A,1} + q h_{a,1}} + c_2 \dfrac{p h_{A,2} w_2 + q h_{a,2} v_2}{p h_{A,2} + q h_{a,2}}} p \ ,$$

$$q' = 1 - p' \ .$$

The first expression can be rewritten as

$$p' = \frac{c_1 h_{A,1} w_1 (p h_{A,2} + q h_{a,2}) + c_2 h_{A,2} w_2 (p h_{A,1} + q h_{a,1})}{c_1 (p h_{A,1} w_1 + q h_{a,1} v_1)(p h_{A,2} + q h_{a,2}) + c_2 (p h_{A,2} w_2 + q h_{a,2} v_2)(p h_{A,1} + q h_{a,1})} p \ ,$$

from which we obtain the change in the frequency of allele A between consecutive rounds of pooling,

$$\Delta p = pq \frac{c_1 (h_{A,1} w_1 - h_{a,1} v_1)(p h_{A,2} + q h_{a,2}) + c_2 (h_{A,2} w_2 - h_{a,2} v_2)(p h_{A,1} + q h_{a,1})}{c_1 (p h_{A,1} w_1 + q h_{a,1} v_1)(p h_{A,2} + q h_{a,2}) + c_2 (p h_{A,2} w_2 + q h_{a,2} v_2)(p h_{A,1} + q h_{a,1})} \ .$$

We can thus conclude that allele A is protected if

$$c_1 (h_{A,1} w_1 - h_{a,1} v_1) h_{a,2} + c_2 (h_{A,2} w_2 - h_{a,2} v_2) h_{a,1} > 0$$

and that allele a is protected if

$$c_1 (h_{a,1} v_1 - h_{A,1} w_1) h_{A,2} + c_2 (h_{a,2} v_2 - h_{A,2} w_2) h_{A,1} > 0 \ .$$