THE ROYAL SOCIETY

doi 10.1098/rspb.2000.1144

Adaptation to marginal habitats: contrasting influence of the dispersal rate on the fate of alleles with small and large effects

Tadeusz J. Kawecki

Zoology Institute, University of Basel, Rheinsprung 9, CH-4051 Basel, Switzerland (t.kawecki@unibas.ch)

The focus of this paper is the relationship between the dispersal rate and the conditions for invasion of a rare allele that improves performance in a marginal sink habitat at the expense of reducing fitness in the main source habitat. Classic multiple-niche population–genetic models predict that the conditions for the invasion of such an allele always become more favourable as the dispersal rate decreases. Precisely the opposite prediction was reached in demographic fitness-sensitivity studies. This study reconciles those contradictory predictions and identifies the assumptions responsible for the discrepancy. I show that whether a lower dispersal rate makes the conditions for the invasion of the allele more or less stringent depends on the magnitude of the effects of the allele. If the effect is large relative to the degree of maladaptedness of the original genotype to the marginal habitat, the conditions become less stringent with decreasing dispersal rate. The opposite is the case for mutations with very small effects. For a broad range of mutations with intermediate effects the conditions are most stringent under an intermediate dispersal rate.

Keywords: local adaptation; source–sink dynamics; novel environment; population genetics; niche expansion; gene flow

1. INTRODUCTION

The ecological niche and geographic range of a species are affected over evolutionary time by its ability to adapt to new, initially marginal habitats. Adaptation to new habitats posits a problem for evolution. On the one hand, marginal populations tend to be demographic sinks (Pulliam 1988; Dias 1996). Since the population is initially not adapted to the marginal habitat, reproductive success there is low and immigration may be necessary in order to maintain the local population. On the other hand, the immigrants bring along genes that, in their evolutionary history, were mostly exposed to natural selection in the core habitat. This gene flow tends to swamp local adaptation. Under what circumstances is adaptation to a marginal sink habitat more likely? In particular, which dispersal rates between the habitats are most and least favourable?

One way of approaching this problem is to study the fate of a rare allele that improves fitness in the marginal (sink) habitat at the cost of reducing fitness in the core (source) habitat. Two kinds of theoretical studies have addressed the effect of the dispersal rate on the conditions for the invasion of a rare allele showing this kind of antagonistic pleiotropy. On the one hand, classic population–genetic multiple-niche models assume that dispersal between habitats affects the genetic composition of local populations, but not their size. Those models invariably predict that the conditions for such an allele increasing when rare become less stringent as the dispersal rate decreases, with complete isolation being the most favourable (Deakin 1966; Maynard Smith 1970; Christiansen 1974; Karlin & Campbell 1981; reviewed in Felsenstein 1976; Hedrick *et al.* 1976). This neglects the possibility that the local population may become extinct when cut off from immigration and, in general, that dispersal may strongly affect local population sizes. On the other hand, several more recent models have included explicit source–sink population dynamics, but in turn sacrificed genetics for a fitness-sensitivity approach (Holt & Gaines 1992; Kawecki 1995; Holt 1996*a*,*b*). This approach compares the sensitivity of the overall fitness, averaged over habitats in an appropriate way, to performance (e.g. lifetime reproductive success) in different habitats (for general background on sensitivity analysis see Caswell (1989)). Since that approach is based on taking derivatives, it is equivalent to considering alleles with infinitesimal effects. Those models predict that a lower dispersal rate always makes the conditions for the invasion of a rare allele beneficial in the sink and harmful in the source more stringent. The predictions of the two kinds of models are thus exactly opposite.

This paper attempts to resolve this paradox. In a two-patch model with passive dispersal and explicit source–sink population dynamics, I consider the conditions for the increase of a rare allele with finite (i.e. not infinitesimal), antagonistic effects on fitness in the two habitat patches. I show that whether lower dispersal rate makes the conditions for increase of the allele more or less stringent depends on the magnitude of the effects of the allele. If the effect is large relative to the degree of maladaptedness of the original genotype to the marginal habitat, the conditions become less stringent with decreasing dispersal rate. The opposite is the case for mutations with very small effects; for a broad range of mutations with intermediate effects the conditions are most stringent under an intermediate dispersal rate.

2. THE MODEL

The model analysed below is derived from Holt (1996*a*). I consider a population that lives in two habitat

Received 9 December 1999 *Accepted* 27 March 2000
© 2000 The Royal Society

patches connected by passive dispersal. The population is assumed to be large enough for stochastic processes (demographic stochasticity and genetic drift) to be neglected. Initially the population is genetically monomorphic. The genotype fixed in the population is well adapted to habitat 1, but its intrinsic rate of increase in habitat 2 is negative. Therefore, habitat 2 is an absolute sink: the local population is maintained by dispersal from the source habitat (habitat 1). For reasons of mathematical tractability, density dependence is only assumed to operate in habitat 1; this assumption is relaxed in the next section. The dynamics of the initial, monomorphic population are described by

$$\frac{dN_1}{dt} = (r_1 - bN_1 - m)N_1 + mN_2 \quad (1)$$

and

$$\frac{dN_2}{dt} = mN_1 + (r_2 - m)N_2, \quad (2)$$

where N_i is the population size and r_i is the intrinsic rate of increase in habitat i ($r_1 > 0$ and $r_2 < 0$), b measures the strength of the density dependence and m is the per capita dispersal rate between the habitats (assumed to be habitat independent). These dynamics lead to a stable equilibrium with population sizes $N_1^* = [r_1 + mr_2/(m - r_2)]/b$ and $N_2^* = N_1^* m/(m - r_2)$. The equilibrium fraction of individuals inhabiting habitat 2 is thus independent of r_1; this is a consequence of no density dependence in habitat 2. Note however that $r_1 > -mr_2/(m - r_2)$ is required for the two coupled populations to persist (i.e. for N_1^*, $N_2^* > 0$).

Consider now a mutant allele that, in the heterozygous state, reduces the intrinsic rate of increase in habitat 1 by s_1 and improves it in habitat 2 by s_2 ($-s_1$ and $+s_2$ are the selection coefficients on the heterozygote in habitats 1 and 2, respectively). When will this allele invade (i.e. increase in frequency) when rare? As usual for a non-recessive allele, whether it invades is determined by the fitness (in this case the logarithmic rate of increase) of the heterozygote (e.g. Crow & Kimura 1970; Liberman 1988; Charlesworth 1994). The dynamics of the number of heterozygotic individuals in the two habitats M_1 and M_2, introduced at a low frequency ($M_1, M_2 << N_1^*, N_2^*$) into the equilibrium population of the resident genotype, is described by the set of equations

$$\frac{dM_1}{dt} = (r_1 - s_1 - bN_1^* - m)M_1 + mM_2 \quad (3)$$

and

$$\frac{dM_2}{dt} = mM_1 + (r_2 + s_2 - m)M_2. \quad (4)$$

These equations are linear; since the mutant allele is rare, mutant homozygotes and the contribution of the heterozygotes to density dependence are neglected. (These equations could be reformulated in terms of the mutant allele frequencies by dividing them by $2N_1^*$ and $2N_2^*$, respectively.) After converging to a stable distribution between the habitats, the numbers of heterozygotes will grow at a logarithmic rate given by the dominant eigenvalue of the matrix of the coefficients corresponding to equations (3) and (4). This eigenvalue, which measures the fitness of the heterozygote, equals

$$\lambda = \frac{1}{2}\left[F_1 + F_2 - 2m + \sqrt{(F_1 - F_2)^2 + 4m^2}\right], \quad (5)$$

where $F_1 = r_1 - bN_1^* - s_1$ and $F_2 = r_2 + s_2$ (Holt 1996*a*). The analogous eigenvalue for the common genotype is zero, since the population is at equilibrium. Positive λ therefore implies an increase in both the number of heterozygous individuals and the mutant allele frequency: the allele will invade if $\lambda > 0$. One can expect that this condition will not be satisfied if the reduction in fitness in habitat 1 (s_1) is large and the improvement in fitness in habitat 2 (s_2) is slight. For a given s_1, how large must s_2 be in order for the mutant allele to invade? With a little algebra one can show that the condition $\lambda > 0$ can be expressed as $s_2 > s_2^*$, where

$$s_2^* = \frac{(m - r_2)^2 s_1}{m_2 + (m - r_2)s_1}. \quad (6)$$

Equation (6) thus gives the minimum improvement in fitness in habitat 2 needed to compensate for a given reduction in fitness in habitat 1. The larger s_2^*, the more stringent the condition for invasion of the mutant allele.

Several conclusions can be drawn from analysing equation (6). First, the condition depends on r_2 but not on r_1 and b (compare with Holt (1996*a*)). This reflects the fact that the relative sizes of the subpopulations in the two habitats (N_1^*/N_2^*) are independent of r_1. When r_2 is larger (less negative), s_2^* is smaller, i.e. the condition for invasion of the rare allele is more favourable when habitat 2 is a 'milder' sink. Second, as might be expected, s_2^* is an increasing function of s_1. More interestingly however, the ratio s_2^*/s_1 decreases with increasing s_1, that is, the factor by which the minimum fitness advantage in habitat 2 needed to compensate for the fitness reduction in habitat 1 is smaller for mutations with large effects. Third, the relationship between s_2^* and the dispersal rate is not straightforward.

The fitness-sensitivity approach used by Holt (1996*a*) is equivalent to considering mutations with infinitesimal effects ($s_1 \to 0$). As s_1 tends to zero, s_2^* tends to $s_1(m - r_2)^2/m^2$, which decreases monotonically with increasing m. The condition $s_2 > s_1(m - r_2)^2/m^2$ is equivalent to equation (5) in Holt (1996*a*). It confirms his conclusion that a higher dispersal rate leads to less stringent conditions for increase of the rare allele.

That conclusion does not generally hold for mutations of finite effects, i.e. when $s_1 > 0$ (figure 1). As m tends to zero (i.e. to complete isolation), s_2^* tends to $-r_2$: the plots for different s_1-values converge at $m = 0$ in figure 1. In other words, close to complete isolation, the intrinsic rate of increase of the mutant heterozygote in habitat 2, $r_2 + s_2$, must be positive for the allele to increase, even if the reduction in fitness in habitat 1 (s_1) is slight. This implies that evolution of improved performance in the sink habitat by accumulation of consecutive mutations with small effects is unlikely if dispersal is very limited. At the other end of the spectrum, as m tends to infinity (i.e. the environment becomes increasingly fine grained), s_2^* tends to s_1, that is, at very high dispersal rates, for the mutant allele to spread it suffices that its positive effect on fitness in habitat 2 is larger than the reduction in fitness in

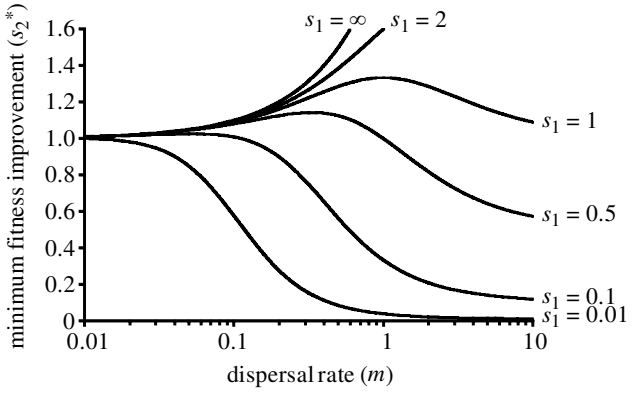

Figure 1. The condition for invasion of a rare allele which improves fitness in the marginal habitat (habitat 2) but reduces fitness in the core habitat (habitat 1) as a function of the dispersal rate m between the habitats. The condition is expressed as the minimum improvement in fitness in the marginal habitat s_2^* which permits an allele reducing fitness in the source habitat by s_1 to increase in frequency when rare; $s_1 = \infty$ implies that the allele is lethal in habitat 1. No density dependence in the sink was assumed. These results are for $r_2 = -1$; as long as $r_2 < 0$, the parameters of the model can be rescaled by choosing a time-scale on which $r_2 = -1$.

habitat 1. The condition for increase of the rare allele is therefore more stringent under very low ($m \to 0$) than very high ($m \to \infty$) dispersal if $s_1 < -r_2$; the reverse is true for mutant alleles with larger effects.

What happens between these extremes? In the vicinity of $m = 0$, s_2^* is always an increasing function of m. The derivative

$$\frac{ds_2^*}{dm} = \frac{(m - r_2)[(m - r_2)s_1 + 2mr_2]s_1}{[m^2 + (m - r_2)s_1]^2} \tag{7}$$

is always positive at $m = 0$ (the first term in the numerator is positive since $r_2 < 0$). One can show that $ds_2^*/dm < 0$ if

$$m > \frac{s_1 r_2}{s_1 + 2r_2} \tag{8}$$

and

$$s_1 + 2r_2 < 0. \tag{9}$$

In other words, for $s_1 < -2r_2$ there is an intermediate dispersal rate $m = s_1 r_2/(s_1 + 2r_2)$ at which the condition for the increase in the mutant allele is most demanding. The maximum is $s_2^* = -4r_2^2/(s_1 + 4r_2)$. The maximum is not evident in figure 1 for small s_1; if $s_1 << -r_2$ it occurs at a low dispersal rate ($m \approx s_1/2$) and exceeds the limit at $m \to 0$ only slightly. The proposition that higher dispersal always leads to more favourable conditions for an invasion of the rare allele thus holds to good approximation when $s_1 << -r_2$. However, as s_1 increases, the range of dispersal rates over which s_2^* is an increasing function of m becomes wider and the 'hump' in the curve becomes more pronounced (figure 1). For mutations with $s_1 > -2r_2$ (which implies a large mutation effect or a 'mild' sink), s_2^* is a monotonically increasing function of m, converging to $s_2^* = s_1$ from below as m tends to infinity. For mutations lethal in habitat 1 ($s_1 \to \infty$), $s_2^* = m - r_2$ (compare with Gomulkiewicz *et al.* (1999)). To summarize, even in this simple model, the relationship between the dispersal rate and the conditions for the invasion of a mutant allele is complex and differs qualitatively between alleles with small and large effects.

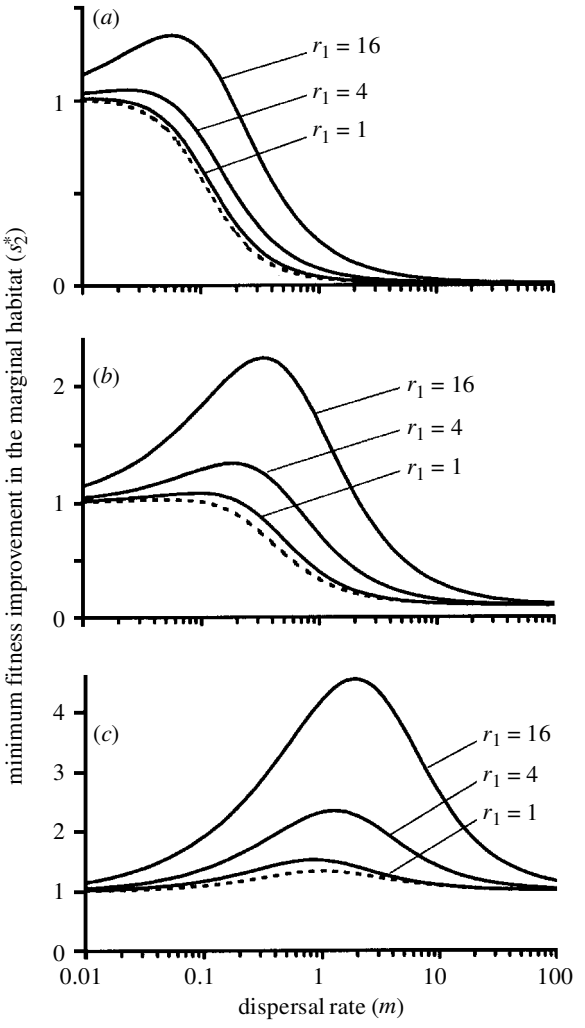

Figure 2. The minimum improvement in fitness in the marginal habitat needed for the rare allele to invade (s_2^*) as a function of the dispersal rate m under density dependence in the sink (solid lines), compared with the density-independent case (broken lines). (*a*) $s_1 = 0.01$, (*b*) $s_1 = 0.1$ and (*c*) $s_1 = 1$.

3. DENSITY DEPENDENCE IN THE SINK HABITAT

In this section I relax the assumption of no density dependence in the marginal habitat. Based on a fitness-sensitivity analysis, Holt (1996*a*) suggested that adding sink density dependence to the above model further weakens selection on performance in the sink, making conditions for adaptation to it more stringent. A similar conclusion was reached by Gomulkiewicz *et al.* (1999), who analysed the fate of rare alleles in a 'black hole' sink (no dispersal back from the sink to the source). In addition to confirming their results for alleles with finite effects under bidirectional dispersal, it is of interest to see how density dependence affects the relationship between the dispersal rates and the conditions for the invasion of a rare allele.

Adding density dependence in the sink also allows one to generalize the above results to cases when $r_2 > 0$. Even if the population in the marginal habitat could persist in the absence of immigration, coupling it with a high-quality core habitat will usually result in a source–sink

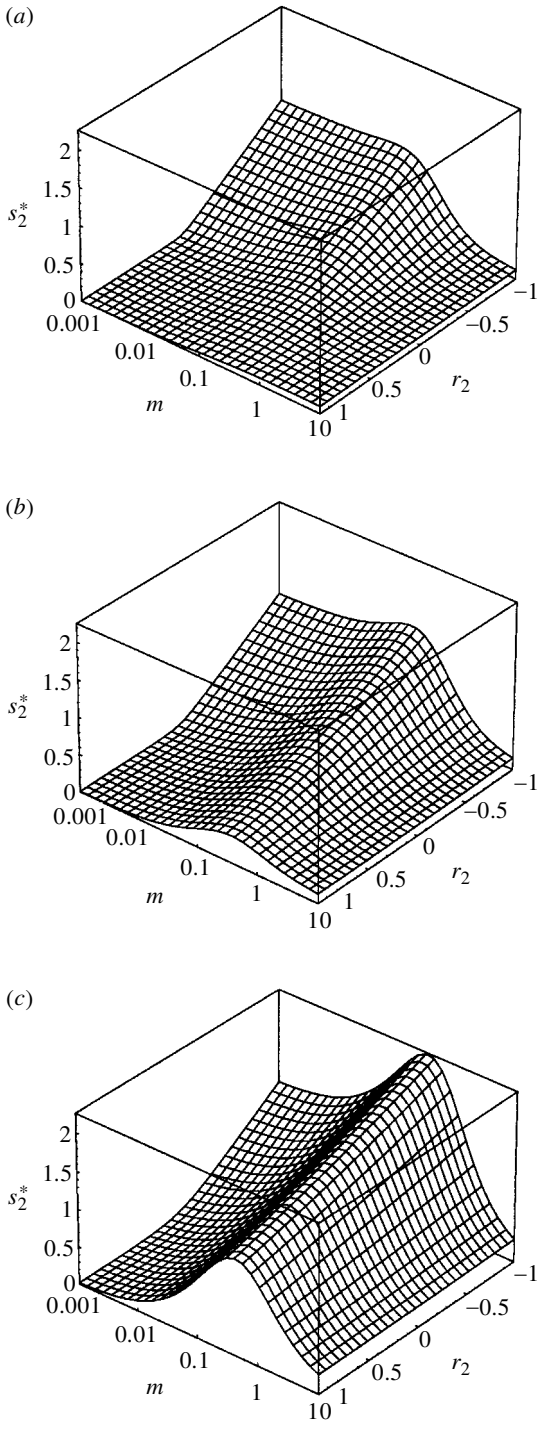

Figure 3. The minimum improvement in fitness in the marginal habitat needed for the rare allele to invade (s_2^*) as a function of the dispersal rate m and the intrinsic rate of increase in the marginal habitat r_2 when there is density dependence in the marginal habitat. (*a*) $r_1 = 1$, (*b*) $r_1 = 4$ and (*c*) $r_1 = 16$. $s_1 = 0.1$ in all panels.

population structure (at least when the population dynamics lead to a stable equilibrium). The net flow of migrants from the source to the sink habitat will maintain the population in the sink above the local carrying capacity, resulting in negative local population growth rate (deaths exceeding births) at the equilibrium; the reverse will hold in the source (Holt 1985; Pulliam 1988; Kawecki 1995). Does whether or not a sink habitat can maintain a population without immigration make a qualitative difference for adaptation in it? In particular, does it affect the conclusions about the non-monotonic effect of the dispersal rate? I address this question in the present model by comparing the results for $r_2 < 0$ and $r_2 > 0$.

I modify the model by adding a density-dependent term to equation (2) to obtain

$$\frac{\mathrm{d}N_2}{\mathrm{d}t} = mN_1 + (r_2 - bN_2 - m)N_2, \qquad (10)$$

while the term $-bN_2^*M_2$ is added to the right-hand side of equation (4). Equations (1) and (3) remain unchanged. The eigenvalue describing the asymptotic growth rate of the number of heterozygotes when the mutant allele is rare is given by equation (5) where $F_1 = r_1 - bN_1^* - s_1$ and $F_2 = r_2 - bN_2^* + s_2$. By setting $\lambda = 0$ and solving for s_2, one can express the minimum fitness improvement in habitat 2, which permits the rare allele to increase in terms of the equilibrium population densities of the common genotype, N_1^* and N_2^*:

$$s_2^* = \frac{m^2}{r_1 - s_1 - bN_1^* - m} - r_2 + bN_2^* + m. \qquad (11)$$

The equilibrium population densities can be found analytically, but the resulting unwieldy and uninformative expression is not presented here. Instead, qualitative results based on its behaviour at $m \to 0$ and $m \to \infty$ are discussed and illustrated with numerical examples.

The qualitative results can be summarized as follows.

(i) Both equilibrium population densities are inversely proportional to b; this parameter does not affect s_2^* even though it occurs in equation (11).
(ii) As the dispersal rate tends to infinity, s_2^* tends to s_1, as in the density-independent case.
(iii) If $r_2 \leqslant 0$, $s_2^* \to -r_2$ as $m \to 0$, but if $r_2 > 0$, $s_2^* \to 0$ as $m \to 0$.
(iv) For any r_2, s_2^* is an increasing function of m in the immediate vicinity of $m = 0$, again as in the density-independent case.

Figure 2*a*,*b* illustrates how density dependence in the sink affects s_2^* between these two extremes, when $r_2 < 0$. As expected, density dependence in the sink makes the condition for increase of the rare allele more stringent and this effect increases with increasing r_1 (recall that under no sink density dependence s_2^* is independent of r_1). The effect of density dependence on s_2^* is strongest at intermediate dispersal rates. The non-monotonic nature of the relationship between m and s_2^* is more pronounced under sink density dependence and, when r_1 is large, it is evident even for alleles with a relatively small effects (figure 2*a*).

From results (ii) and (iii) it follows that the condition for invasion of the rare allele is more favourable under complete isolation than under infinite dispersal if $s_1 > -r_2$. This is always satisfied for $r_2 > 0$. Nonetheless, the shape of the relationship between m and s_2^* does not change qualitatively as r_2 changes from negative to positive (figure 3). Numerical analysis of the model suggests that s_2^* has a maximum at an intermediate m whenever $s_1 < r_1 - r_2$. The maximum becomes more pronounced as the difference between the two habitats $(r_1 - r_2)$ increases

(figure 3). The conclusion, that the conditions for adaptation to a marginal habitat tend to be most stringent under intermediate dispersal rates, thus remains upheld when the marginal habitat can maintain a population without immigration. Only when the difference in quality of the two habitats is smaller than the effect of the rare allele in the core habitat does s_2^* increase monotonically with the dispersal rate (the left-front edge of figure 3*a*).

4. DISCUSSION

The focus of this paper is the relationship between the dispersal rate and the conditions for invasion of a rare allele that improves fitness in a marginal sink habitat but reduces fitness in the core source habitat. The main conclusion is that this relationship is not generally monotonic: its sign and shape depend on the magnitude of the effect of the rare allele. If the allele reduces fitness in the main habitat only slightly, the condition for its spread is most favourable at the maximum dispersal rate and becomes increasingly stringent as the dispersal rate decreases. Conversely, if the effect of the allele is large enough to reverse the source and sink roles between the habitats, the condition becomes increasingly favourable as the dispersal rate decreases. For a broad range of alleles with intermediate effects, the condition for increase when rare is most stringent at an intermediate dispersal rate (figure 1). This non-monotonic character of the relationship is most pronounced when there is density dependence in the sink and the difference in the intrinsic rate of increase between the habitats is large (figure 2). These qualitative results apply irrespective of whether or not the local population in the marginal habitat could persist without immigration (figure 3), as long as the absolute fitness in the marginal habitat is lower than the fitness in the main habitat, resulting in a source–sink population structure.

The above results reconcile the apparently contradictory predictions of the population–genetic multiple-niche models and the demographic fitness-sensitivity models discussed in § 1. The fitness-sensitivity models (Holt & Gaines 1992; Kawecki 1995; Holt 1996*a*) predict that the conditions favouring adaptation to a sink habitat become less stringent as the dispersal rate increases. This is what the above model predicts for an allele with an infinitesimally small effect on the fitness in the main habitat. On the other hand, the assumption made in the classic multiple-niche genetic models that dispersal has a negligible effect on population dynamics (Deakin 1966; Maynard Smith 1970; Christiansen 1974; Felsenstein 1976; Hedrick *et al*. 1976; Karlin & Campbell 1981) would hold if the two habitats were of identical quality and, therefore, exchanged the same numbers of migrants. In this case ($r_1 = r_2$), the above model predicts that the condition for increase in the rare allele is most favourable when there is complete isolation and becomes increasingly stringent as the dispersal rate increases. The same prediction was obtained in those multiple-niche models.

It remains to be seen how general the qualitative results of this model are. Examination of alternative assumptions about population dynamics and gene action go beyond the scope of this paper. However, a discrete-time model with a different form of density dependence produced qualitatively similar results (T. J. Kawecki, unpublished data). Also note that the deterministic condition for invasion of an allele is only one aspect of adaptive evolution in a new habitat. Interestingly, Gomulkiewicz *et al*. (1999) recently found that the establishment rate of new beneficial mutations was highest under an intermediate immigration rate for a black hole sink. The reason is that the supply rate of new mutations increases but the probability of fixation decreases with increasing dispersal rate. That study and the one presented here underscore the need for incorporating both the ecological and genetic effects of dispersal in studies on evolution in marginal habitats.

Most ecologically important characters are affected by many loci with small effects and adaptations to a new natural environment are also likely to be polygenic. On the other hand, the adaptation of natural populations to anthropogenic environmental changes, notably the evolution of resistance to pesticides and the ability of pests to attack initially resistant crop varieties, often involves one or a few major loci. The results of this paper suggest that the effect of dispersal and gene flow on adaptation to new environments may differ qualitatively depending on whether the adaptation involves one locus with a large effect or many loci with small effects. This would not only have consequences for understanding the evolutionary dynamics of species' ranges, but also for integrated pest management strategies aiming at slowing down the evolutionary responses of pest populations to control measures (Gould 1998). As this study demonstrates, both ecological and genetic aspects of dispersal are likely to affect adaptation to new environments and their joint effect need not be straightforward.

D. Ebert, T. Killingback, R. Holt and S. Stearns commented on a previous version of the manuscript. This work was supported by Swiss Nationalfonds grant 3100-053601.98. It was stimulated in part by a workshop at the National Center for Ecological Analysis and Synthesis, University of California at Santa Barbara, USA.

REFERENCES

Caswell, H. 1989 *Matrix population models*. Sunderland, MA: Sinauer Associates.

Charlesworth, B. 1994 *Evolution in age-structured populations*. Cambridge University Press.

Christiansen, F. B. 1974 Sufficient conditions for protected polymorphism in a subdivided population. *Am. Nat.* **108**, 157–166.

Crow, J. F. & Kimura, M. 1970 *An introduction to population genetics theory*. New York: Harper & Row.

Deakin, M. A. B. 1966 Sufficient conditions for genetic polymorphism. *Am. Nat.* **100**, 690–692.

Dias, P. C. 1996 Sources and sinks in population biology. *Trends Ecol. Evol.* **11**, 326–330.

Felsenstein, J. 1976 The theoretical population genetics of variable selection and migration. *A. Rev. Genet.* **10**, 253–280.

Gomulkiewicz, R., Holt, R. D. & Barfield, M. 1999 The effects of density-dependence and immigration on local adaptation in a 'black-hole' sink environment. *Theor. Popul. Biol.* **55**, 283–296.

Gould, F. 1998 Sustainability of transgenic insecticidal cultivars: integrating pest genetics and ecology. *A. Rev. Entomol.* **43**, 701–726.

Hedrick, P. W., Ginevan, M. E. & Ewing, E. P. 1976 Genetic polymorphism in heterogeneous environments. *A. Rev. Ecol. Syst.* **7**, 1–32.

Holt, R. D. 1985 Population dynamics in two-patch environments: some anomalous consequences of an optimal habitat distribution. *Theor. Popul. Biol.* **28**, 181–208.

Holt, R. D. 1996*a* Adaptive evolution in source–sink environments: direct and indirect effects of density-dependence on niche evolution. *Oikos* **75**, 182–192.

Holt, R. D. 1996*b* Demographic constraints in evolution: towards unifying the evolutionary theories of senescence and niche conservatism. *Evol. Ecol.* **10**, 1–11.

Holt, R. D. & Gaines, M. S. 1992 Analysis of adaptation in heterogeneous landscapes: implications for the evolution of fundamental niches. *Evol. Ecol.* **6**, 433–447.

Karlin, S. & Campbell, R. B. 1981 The existence of a protected polymorphism under conditions of soft as opposed to hard selection in a multideme population system. *Am. Nat.* **117**, 262–275.

Kawecki, T. J. 1995 Demography of source–sink populations and the evolution of ecological niches. *Evol. Ecol.* **9**, 38–44.

Liberman, U. 1988 External stability and ESS: criteria for initial increase of new mutant allele. *J. Math. Biol.* **26**, 477–485.

Maynard Smith, J. 1970 Genetic polymorphism in a varied environment. *Am. Nat.* **104**, 487–490.

Pulliam, H. R. 1988 Sources, sinks, and population regulation. *Am. Nat.* **132**, 652–661.