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How does pollen versus seed dispersal affect niche evolution?

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Code for numerical iteration and simulation of the model deposited at Dryad: doi:10.5061/dryad.hd412

Abstract

In heterogeneous landscapes, the genetic and demographic consequences of dispersal influence the evolution of niche width. Unless pollen is limiting, pollen dispersal does not contribute directly to population growth. However, by disrupting local adaptation, it indirectly affects population dynamics. We compare the effect of pollen versus seed dispersal on the evolution of niche width in heterogeneous habitats, explicitly considering the feedback between maladaptation and demography. We consider two scenarios: the secondary contact of two subpopulations, in distinct, formerly isolated habitats, and the colonization of an empty habitat with dispersal between the new and ancestral habitat. With an analytical model, we identify critical levels of genetic variance leading to niche contraction (secondary contact scenario), or expansion (new habitat scenario). We confront these predictions with simulations where the genetic variance freely evolves. Niche contraction occurs when habitats are very different. It is faster as total gene flow increases or as pollen predominates in overall gene flow. Niche expansion occurs when habitat heterogeneity is not too high. Seed dispersal accelerates it, whereas pollen dispersal tends to retard it. In both scenarios very high seed dispersal leads to extinction. Overall, our results predict a wider niche for species dispersing seeds more than pollen.

Keywords: Local adaptation, gene flow, source-sink dynamics, secondary contact, genetic variance, demography.

1 Introduction

A species' niche can be defined as the range of environmental conditions in which it persists without immigration (Hutchinson, 1957). Theory about niche evolution thus lies at the intersection of ecology and genetics, asking how demography constrains adaptation and, conversely, how local adaptation affects the distribution of population numbers across habitats (Holt, 2009). In a landscape that is heterogeneous with respect to selection, the genetic and demographic consequences of dispersal jointly influence the evolution of niche width (see reviews in e.g. Bridle and Vines, 2007; Kawecki, 2008; Sexton et al., 2009; Holt and Barfield, 2011). Models of dispersal and adaptation in heterogeneous environments often characterize dispersal with a single parameter. Many organisms however display a life cycle with several dispersal stages. In plants with seed and pollen dispersal, demographic dispersal and colonization are partly uncoupled from gene flow (Thrall et al., 1998; Kremer et al., 2012).

In this paper, we investigate how dispersal through pollen versus seeds affects niche width evolution in a heterogeneous environment. We consider two scenarios of niche evolution. In the "secondary contact scenario", two populations of the same species adapted to distinct habitats, previously isolated, come into contact: we examine the effect of pollen dispersal on the probability and dynamics of demographic collapse and niche contraction following contact. Study of this scenario may clarify the consequences of, for example, secondary contact between crops and their wild relatives (Ellstrand et al., 1999). In the "new habitat scenario", the species colonizes a new habitat initially outside its niche, with persistent gene flow from the ancestral habitat: we examine the effect of pollen dispersal on the probability and dynamics of niche expansion. This scenario may offer insights into, for example, plant adaptation to extreme habitats such as mine tailings (Antonovics, 2006). To fit the case of plant dispersal, we here focus on a species with sexual reproduction, dispersal before selection and passive dispersal of propagules between habitats. We further assume that growth or decline in the sink is initially little affected by the local density and we allow local adaptation to depend on genetic variation at many loci.

In the absence of pollen limitation, pollen dispersal does not contribute directly to population growth. However, by disrupting local adaptation, pollen dispersal indirectly affects population dynamics via detrimental effects on fitness. Similarly, in animals with separate sexes, male dispersal has been predicted to differ from female dispersal in the effect on the process of adaptation to marginal habitats when only females directly contribute to population growth (Kawecki, 2003). Pollen and seed dispersal are furthermore expected to exert different constraints on local adaptation (Nagylaki, 1997; Hu and Li, 2001, 2002; Lopez et al., 2008). First, the extent of pollen and seed dispersal, and therefore their relative contribution to gene flow, varies considerably among plant species (Ouborg et al., 1999; Petit et al., 2005). Second, for equal numbers of pollen grains and seeds dispersed, seeds carry twice as many nuclear genes as pollen (Hu and Ennos, 1999). Finally, for the same number of gene copies dispersed, Lopez et al. (2008) predicted that, when selection is strongly divergent between habitats, the genetic load from pollen dispersal should be higher than that from seed, because of the relative inefficiency of purging of maladaptive alleles in heterozygotes formed from immigrating pollen. Yet, the previous authors did not explore the demographic consequences of the genetic load nor its impact on niche evolution.

Adaptation to marginal habitats leading to niche expansion (and failure to adapt leading to niche conservatism) has been intensively studied in the context of source-sink models (e.g. Holt and Gomulkiewicz, 1997a; Gomulkiewicz et al., 1999; Tufto, 2001; Ronce

and Kirkpatrick, 2001; Holt et al., 2003). Dispersal of individuals between source and sink populations tends to homogenise their sizes and genetic compositions, with antagonistic consequences for adaptation to marginal habitats (see reviews in Kawecki, 2008; Holt and Barfield, 2011). Influx of genes that enhance adaptation in the source may compromise fitness locally, maintaining the sink population in a state of severe maladaptation and low local abundance. Nevertheless, by boosting the sink population size, immigration of individuals from the source reduces the asymmetry of gene flow between source and sink, weakening the constraints on adaptation to marginal conditions. Furthermore, immigration from large and genetically diverse populations can facilitate adaptation to marginal conditions by increasing the amount of genetic variation exposed to selection in the sink. Many models integrating these multifarious effects of dispersal predict that the net effect of increasing the rate of seed dispersal between habitats is to facilitate and accelerate niche expansion (Holt and Gaines, 1992; Kawecki, 1995; Holt, 1996a,b; Holt and Gomulkiewicz, 1997a; Barton, 2001; Ronce and Kirkpatrick, 2001; Holt et al., 2003; Polechová et al., 2009). Exact effects of dispersal on niche expansion depend on the timing of dispersal within the life cycle, the pattern of dispersal between habitats, density-dependence of population growth in the sink, mating system and genetic architecture of local adaptation (see review in Holt and Barfield, 2011). Pollen dispersal is expected to differ from seed dispersal in its effects on population demography, the evolution of genetic variance and genetic divergence in marginal habitats (Lopez et al., 2008), which raises questions about its specific consequences for niche expansion. Hu and He (2006) found that, by affecting the spread of deleterious and advantageous mutations, pollen dispersal could slow down (when immigrating genes are maladaptive to recipient populations) or accelerate (when they are adaptive) range expansion in homogeneous environments. Butlin et al. (2003) found that pollen dispersal distances affect the probability of range expansion along environmental gradients (see also a short investigation of that question in Antonovics et al., 2001).

Conversely, niche contraction following secondary contact was described by Ronce and Kirkpatrick (2001) as a process called migrational meltdown. In strongly heterogeneous habitats, the genetic load generated by gene flow between two initially locally adapted populations can be large enough to cause demographic collapse. Small stochastic asymmetries in population sizes can be exaggerated by the stronger maladaptive effect of gene flow in smaller populations, resulting in a spiral of lower fitness and population size and the ultimate loss of a viable population in one of the habitats. The likelihood of migrational meltdown and niche contraction with pollen dispersal has not previously been explored. The higher genetic load found by Lopez et al. (2008) with pollen contribution to gene flow suggests that pollen and seed dispersal could differently affect migrational meltdown.

Here, we investigate the effects of pollen versus seed dispersal on niche width evolution, building on previous theory on niche evolution, in particular Ronce and Kirkpatrick (2001) and Holt et al. (2003). We explicitly model the feedbacks between population dynamics and adaptation, i.e. that local adaptation is influenced by population size, and that, conversely, maladaptation results in reduction in population size. We consider a single quantitative trait under selection toward two different phenotypic optima in two distinct habitats. Genetic variance for this trait is critical to the process of niche expansion and contraction, because it both provides the potential for adaptation and depresses mean fitness. Following Gomulkiewicz and Houle (2009) who defined critical levels of genetic variance allowing an isolated population to adapt to an environmental change, we use an analytical model to identify critical levels of genetic variance leading to niche contraction (secondary contact scenario), niche expansion (new habitat scenario), or extinction (both scenarios). We confront these analytical predictions with individual-based simulations

where the genetic variance freely evolves, and we examine the joint dynamics of adaptation and change in abundance.

2 Models

2.1 Investigated scenarios

In the "secondary contact scenario", we consider the contact of two genetically differentiated populations of the same species, initially living in two isolated, distinct habitats. Both populations are initially adapted to their local environment and at carrying capacity; the niche spanned by the species can be considered "wide". We determine conditions under which both populations persist at carrying capacity, i.e. the niche of the species remains wide. When this does not happen, either both populations go extinct, or niche contraction occurs. In the latter case, one habitat is a source (its population remains at carrying capacity), while the other one is a sink: due to maladaptation, its population cannot persist without immigration from the source. The niche spanned by the species can then be considered "narrow".

In the "new habitat scenario", we consider the possible invasion of a newly available habitat by a population initially adapted to a different habitat. The original habitat is initially a source, while the newly available habitat is a sink. The niche of the species is thus initially narrow. When adaptation proceeds to the point of persistence in both habitats, then the species' niche has become wide, i.e. niche expansion has occurred.

2.2 General assumptions

We consider self-compatible, annual, hermaphroditic plants with no seed bank. The environment consists of two habitat types. In each habitat, selection for an optimal phenotype acts on a single quantitative trait. The habitats are identical in all respects except that their optimal phenotypes, θ_1 and θ_2, differ. Habitats are connected by pollen and seed dispersal: m_p and m_s are, respectively, the probability that a pollen grain or a seed changes habitat at the dispersal stage (see Table 1 for a summary of the notation). Dispersal probabilities are independent of the habitat, and there is no survival cost to dispersal.

We define habitat heterogeneity Δ as the difference between the optimal phenotypes in the two habitats: $\Delta = \theta_2 - \theta_1$. Without loss of generality, we assume that habitat heterogeneity is positive. The phenotype of an individual is the sum of its genotypic value and a Gaussian environmental effect with mean 0 and variance V_e. We measure maladaptation in each habitat, $\bar{G}_1$ and $\bar{G}_2$, as the distance between the mean genotypic value in each habitat, $\bar{g}_1$ and $\bar{g}_2$, and its optimal value: $\bar{G}_1 = \bar{g}_1 - \theta_1$ and $\bar{G}_2 = \theta_2 - \bar{g}_2$. Population size, measured relative to carrying capacity K, in habitat i is $N_i = n_i/K$ where n_i is the number of individuals in habitat i.

We assume discrete and non-overlapping generations with the following life cycle: (i) selection, (ii) density regulation, (iii) gametogenesis, pollen dispersal and syngamy, and (iv) seed dispersal. We found that reversing the order of density regulation and selection did not affect our qualitative conclusions. A juvenile with phenotype z in habitat i survives selection according to a Gaussian function with width ω

$$W_i(z) = \exp\left(-\frac{(z-\theta_i)^2}{2\omega^2}\right)$$

(ω^2 is inversely related to the strength of selection).

Variable	Definition
N_i	Population size relative to carrying capacity in habitat i
$\bar{G}_i$	Mean maladaptation in habitat i
Parameter	**Definition**
m_s	Seed dispersal probability
m_p	Pollen dispersal probability
Δ	Habitat heterogeneity, i.e. difference between optimal phenotypes in the two habitats: $\Delta = \theta_2 - \theta_1$
ω	Width of the fitness function
V_g	Genetic variance before selection
V_e	Environmental variance
f	Mean fecundity
K	Carrying capacity
L	Number of loci
U	Mutation rate per genome
σ^2	Variance of the mutations size
Other notation	**Definition**
θ_i	Optimal phenotype in habitat i
n_i	Number of individuals in habitat i: $n_i = N_i K$
$\bar{g}_1$	Mean genotypic value in habitat 1: $\bar{g}_1 = \bar{G}_1 + \theta_1$
$\bar{g}_2$	Mean genotypic value in habitat 2: $\bar{g}_2 = \theta_2 - \bar{G}_2$
m_t	Total gene flow: $m_\mathrm{t} = m_\mathrm{s} + \frac{1}{2} m_\mathrm{p}(1 - 2m_\mathrm{s})$
v	Average survival probability of individuals with an average genotypic value at the optimum: $v = \sqrt{\omega^2/(V_\mathrm{s} + V_\mathrm{g})}$
V_s	$V_\mathrm{s} = \omega^2 + V_\mathrm{e}$

Table 1: Notation. Parameter V_g is used in the analytical model only. Parameters K, L, U and σ^2 are used in the simulation model only.

We assume a "ceiling" form of density regulation: when the population size after selection in a given habitat exceeds its carrying capacity K, K individuals are randomly sampled, otherwise, all individuals survive density regulation. We also used a continuous density regulation function (Beverton-Holt, see Appendix E). Fecundity does not depend on the habitat: each plant produces on average f ovules. It is assumed that pollen is not limiting, i.e. all ovules are fertilized. We consider partial philopatry, i.e. we use values of m_p and m_s lower than 0.5.

2.3 Analytical model

In the analytical model, we assume a Gaussian distribution of genotypic values before selection with mean $\bar{g}_i$ in habitat i and fixed genetic variance V_g. The mean fitness in habitat i is then

$$\bar{W}_i = v \exp\left(-\frac{\bar{G}_i^2}{2(V_\mathrm{s} + V_\mathrm{g})}\right) \tag{1}$$

where $V_\mathrm{s} = \omega^2 + V_\mathrm{e}$ and $v = \sqrt{\omega^2/(V_\mathrm{s} + V_\mathrm{g})}$ is the average survival probability of individuals with a genotypic value at the optimum. We ignore demographic stochasticity. Changes in mean phenotype and population size along the life cycle are detailed in Appendix A.

We use the analytical model to determine critical levels of genetic variance leading to niche contraction (secondary contact scenario), niche expansion (new habitat scenario), or extinction (both scenarios).

2.4 Simulation model

We have also simulated the two scenarios without any assumption regarding the distribution of genotypes, allowing the genetic variance to evolve. Simulations are individual-based and take into account stochastic effects due to limited population size.

The simulation model employed here has been described in detail elsewhere (Ronce et al., 2009). The individual's genotypic trait value is the sum of allelic effects over L unlinked loci. During gametogenesis, mutations occur at a specified rate U per diploid genome. For a mutation occurring in a given allele, its effect is modeled as that of the original allele, plus a normal deviation with zero mean and variance σ^2. Thus, there is no constraint on the number of alleles that might be segregating at a particular locus, nor is there a constraint on the allelic effect sizes, other than that imposed by selection. Consequently, the genetic variance of each population is free to evolve. At the fertilization stage, the number of juveniles generated in habitat i is taken as a Poisson random variable, with mean equal to f times the number of individuals after density regulation in habitat i.

In the secondary contact scenario, populations in the two habitats are allowed to evolve separately for 1000 generations to achieve mutation-selection-drift balance (as in Holt et al., 2003). We checked that this equilibrium was reached in our simulations (not shown). After 1000 generations gene flow between populations begins. In the new habitat scenario, a single population is generated in habitat 1, with K individuals having their trait at the optimum θ_1, and allowed to evolve for 1000 generations. At this point, dispersal begins between this population and habitat 2, initially empty.

After dispersal begins, the number of generations is counted before a new demographic equilibrium is reached. In the secondary contact scenario, we tally the number of generations until one or the other population drops in size to the point that it is maintained well below the carrying capacity (threshold set to $0.05K$) while the other remains at carrying capacity. In the new habitat scenario, we count the number of generations until the initially empty habitat harbors a population at carrying capacity. Simulations were stopped after 10^5 generations if still at the initial demographic equilibrium. Extinction was recorded when all individuals died. For a given scenario and set of parameter values, a minimum of 100 replicate simulations were run.

3 Results

3.1 Secondary contact scenario

3.1.1 Expected equilibrium at fixed genetic variance

In the secondary contact scenario, the niche of the species is initially wide. The populations in both habitats are isolated and are locally adapted. Dispersal between the two habitats then begins, and we investigate under which conditions the niche width is maintained, that is, under which conditions the wide niche equilibrium is viable and locally stable. In the analytical model, solving Eq A5 assuming $N_1 = N_2$, we found population density and mean maladaptation, measured before selection, at the wide niche equilibrium:

$$\hat{N}_1 = \hat{N}_2 = f$$
$$\hat{\bar{G}}_1 = \hat{\bar{G}}_2 = \frac{m_\mathrm{t}\Delta}{1 - \frac{V_\mathrm{s}}{V_\mathrm{s}+V_\mathrm{g}}(1-2m_\mathrm{t})}, \tag{2}$$

where $m_\mathrm{t} = m_\mathrm{s} + \frac{1}{2}m_\mathrm{p}(1-2m_\mathrm{s})$ is the total gene flow, that is, the probability that a gene copy is dispersed to the other habitat via pollen or seed. Such a gene copy can originate from a dispersed pollen grain's contribution to a non-dispersed seed (probability $\frac{1}{2}(1-m_\mathrm{s})m_\mathrm{p}$), from a non-dispersed pollen grain's contribution to a dispersed seed (probability $\frac{1}{2}m_\mathrm{s}(1-m_\mathrm{p})$), or from an ovule's contribution to a dispersed seed (probability $\frac{1}{2}m_\mathrm{s}$). Note that, as found in Lopez et al. (2008) (see also Nagylaki, 1997; Hu and Li, 2001), maladaptation at the wide niche equilibrium depends on the total gene flow, and not on the relative contribution of pollen and seed dispersal (see however Kawecki (2003) for different results when reproductive outputs vary between patches). Eq 2 shows that higher genetic variance V_g allows better local adaptation (lower $\hat{\bar{G}}_i$). Conversely, dispersal induces maladaptation in each population at the wide niche equilibrium (Eq 2). The wide niche equilibrium is viable (i.e. exists) and is locally stable (see Appendix B) when

$$\Delta < \frac{1}{m_\mathrm{t}}\left(1 - \frac{V_\mathrm{s}}{V_\mathrm{s}+V_\mathrm{g}}(1-2m_\mathrm{t})\right)\sqrt{2(V_\mathrm{s}+V_\mathrm{g})\ln(fv)}. \tag{3}$$

For a specific combination of pollen and seed dispersal probabilities, Figure 1 shows the values of the genetic variance V_g for which the wide niche equilibrium is viable and locally stable, according to the analytical model: maintenance of a wide niche is impossible when V_g is either too small or too large. Too low V_g does not support response to selection that maintains sufficient adaptation of each population for them to persist (Eq 2). When V_g is too high, too few individuals survive selection for both populations to persist.

There are thus two critical values of the genetic variance, denoted $V_\mathrm{g}^{\mathrm{SCinf}}$ and $V_\mathrm{g}^{\mathrm{SCsup}}$, below which and, respectively, above which, it is not possible to maintain a wide niche in the presence of dispersal (SC superscript stands for Secondary Contact scenario). The values of $V_\mathrm{g}^{\mathrm{SCinf}}$ and $V_\mathrm{g}^{\mathrm{SCsup}}$ are the solutions for V_g of Eq 3 where the inequality is replaced by an equality. We were unable to solve Eq 3 analytically for the genetic variance V_g; we obtained the critical genetic variances numerically.

At the wide niche equilibrium, because of the ceiling form of density regulation we assume, seed dispersal does not affect population size after density regulation. The stability of the wide niche equilibrium is consequently not affected by the demographic effect of seed dispersal, and the critical genetic variances are determined by the genetic effect of seed and pollen dispersal. In other words, the values of $V_\mathrm{g}^{\mathrm{SCinf}}$ and $V_\mathrm{g}^{\mathrm{SCsup}}$ depend on total gene flow m_t, but not on the specific pollen and seed dispersal probabilities (Eq 3 depends on m_t only). In Appendix E we consider a continuous density regulation function (Beverton-Holt) for which the stability of the wide niche equilibrium depends on the demographic effect of seed dispersal. Although the values of $V_\mathrm{g}^{\mathrm{SCinf}}$ and $V_\mathrm{g}^{\mathrm{SCsup}}$ then depend on the specific pollen and seed dispersal probabilities (at constant total dispersal, increasing seed dispersal tends to stabilize the wide niche equilibrium), the results described below are qualitatively unchanged and quantitatively weakly affected (Appendix E).

The range of V_g for which a wide niche can be maintained (i.e. $V_\mathrm{g}^{\mathrm{SCsup}} - V_\mathrm{g}^{\mathrm{SCinf}}$) is reduced by total dispersal m_t. This is illustrated in Figure 2, which shows the variations of $V_\mathrm{g}^{\mathrm{SCsup}}$ and $V_\mathrm{g}^{\mathrm{SCinf}}$ as a function of pollen dispersal m_p (and corresponding total dispersal m_t), for different fixed values of seed dispersal m_s and habitat heterogeneity Δ. As total

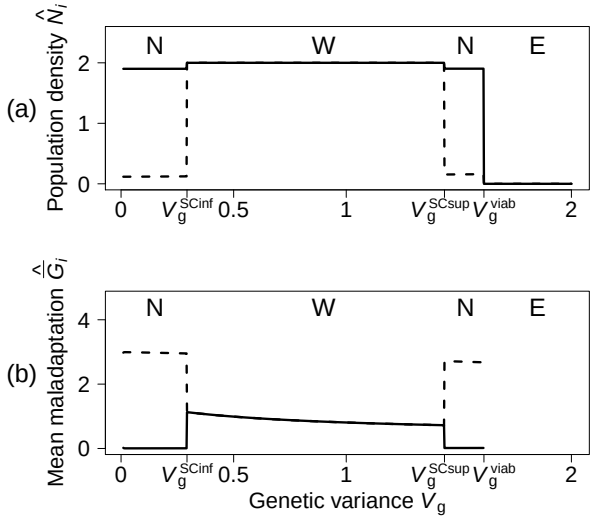


Figure 1: Population density $\hat{N}_i$ (panel (a)) and mean maladaptation $\hat{\bar{G}}_i$ (panel (b)) measured before selection at the evolutionary equilibrium as a function of the fixed genetic variance V_g for the secondary contact scenario. The niche of the species is initially wide; when niche contraction occurs, one population remains a source (solid line), the other one becomes a sink (dashed line). Letters W, N and E indicate the parameter region corresponding to the wide niche equilibrium, narrow niche equilibrium and to extinction of both populations respectively. Results are obtained by numerically iterating Eqs A1-A4 with almost symmetrical initial conditions ($N_1 = f$, $N_2 = f - 0.001$, $\bar{G}_1 = 0$, and $\bar{G}_2 = 0.001$), mimicking a small perturbation at secondary contact of the two populations. Parameter values: $m_s = 0.05$, $m_p = 0.2$, $\Delta = 3$, $1/\omega^2 = 1$, $f = 2$, $V_e = 1$.

gene flow m_t increases, there may be no value of V_g allowing maintenance of a wide niche (on panels (a) and (b) of Figure 2, V_g^{SCsup} and V_g^{SCinf} are not defined for $m_t > 0.182$).

As expected from previous literature (e.g. Ronce and Kirkpatrick, 2001), Figure 2 reveals that the range $V_g^{SCsup} - V_g^{SCinf}$ is reduced by increasing habitat heterogeneity and by stronger selection (panels (a)-(b) with $\Delta = 3$ versus panels (c)-(d) with $\Delta = 2$, and Appendix D).

When a wide niche is not maintained at secondary contact, niche contraction may occur (populations persist, but in a source-sink system), or both populations may go extinct. Figure 1 illustrates that there is a threshold value of the genetic variance, denoted V_g^{viab}, above which the narrow niche equilibrium is not viable. Excessive genetic variance depresses mean fitness, resulting in the extinction of both populations. We derived an approximation for V_g^{viab} assuming that, at the narrow niche equilibrium, close to extinction, the sink is a black-hole sink (i.e. no dispersal from the sink to the source; see Appendix C):

$$V_g^{viab} \approx \omega^2 f^2 (1 - m_s)^2 - V_s. \tag{4}$$

The approximation of V_g^{viab} does not depend on pollen dispersal m_p because (i) it assumes a black-hole sink, which implies that the source does not receive pollen from the sink, and (ii) we assume that pollen does not limit reproduction (all ovules in the source are fertilized). The accuracy of the approximation was checked by comparing it to the viability threshold found by numerical iteration of Eqs A1-A4. The accuracy was found to be generally good, although the approximation given by Eq 4 underestimates the value of V_g^{viab} at high seed dispersal (see e.g. Figure 2).

The critical variance V_g^{viab} decreases with increased seed dispersal (Eq 4, Figure 2).

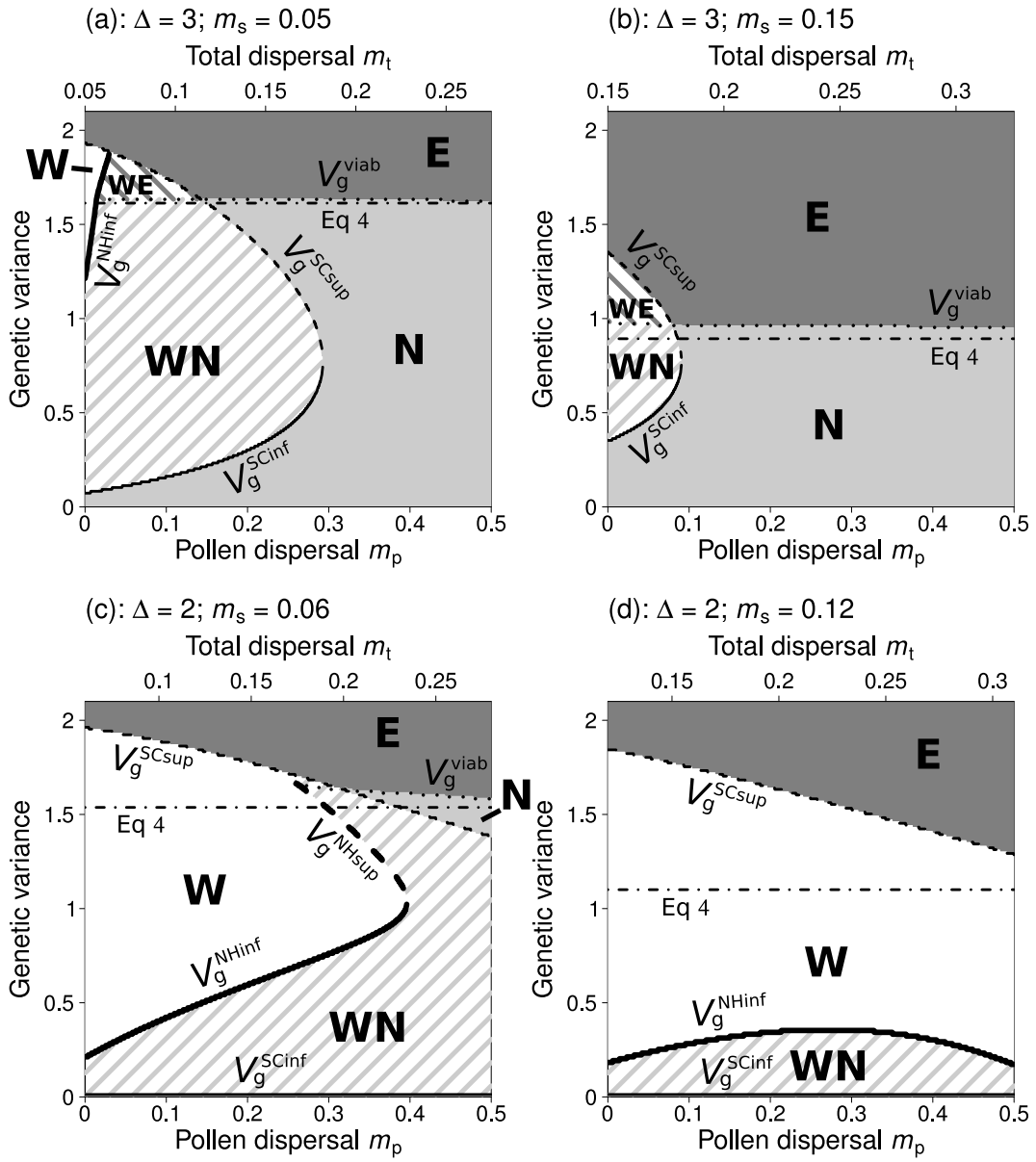


Figure 2: Critical genetic variances V_g^{SCinf}, V_g^{SCsup}, V_g^{NHinf}, V_g^{NHsup}, V_g^{viab} and its approximation by Eq 4, computed with the analytical model as a function of pollen dispersal m_p, for fixed values of seed dispersal m_s and habitat heterogeneity Δ. Solid regions indicate the parameter region where only one equilibrium is viable and locally stable: only the wide niche equilibrium (W), only the narrow niche equilibrium (N), or only extinction of both populations (E). Striped regions indicate bistabilities (WN and WE). Viability and stability of the wide niche equilibrium is determined from Eq 3. Viability and stability of the narrow niche equilibrium is determined from numerical iteration of Eqs A1-A4 with asymmetrical initial values corresponding to an empty sink and an adapted source ($N_1 = f$, $N_2 = 0$, $\bar{G}_1 = 0$, and $\bar{G}_2 = \Delta$). Parameter values: $1/\omega^2 = 1$, $f = 2$, $V_e = 1$.

Indeed, at the narrow niche equilibrium, most of the seeds dispersed from the source to the sink die. When seed dispersal increases, high genetic variance cannot be sustained because of the combined demographic loads due to the effects of selection and the loss of seeds to the sink through dispersal. Similarly, V_g^{viab} decreases when selection becomes stronger (Eq 4, Figure D1).

Figure 2 summarizes the parameter region where, depending on the value of the genetic variance V_g compared to the three critical genetic variances V_g^{SCinf}, V_g^{SCsup} and V_g^{viab}, the

analytical model predicts the maintenance of the wide niche equilibrium ($V_{\mathrm{g}}^{\mathrm{SCinf}} < V_{\mathrm{g}} < V_{\mathrm{g}}^{\mathrm{SCsup}}$) or not, and, in the latter case, if niche contraction or the extinction of both populations ($V_{\mathrm{g}} > V_{\mathrm{g}}^{\mathrm{viab}}$) is predicted.

3.1.2 Dynamics of genetic variance and consequences for niche width evolution

Figure 3 shows typical time series from our individual-based simulations with freely evolving genetic variance V_{g}, for two of the three possible outcomes in the secondary contact scenario. The genetic variance immediately increases at secondary contact, because of the initial genetic differentiation of the populations. Then, because dispersal quickly homogenizes the genotypic distribution between the two habitats, V_{g} tends to decrease, especially at high dispersal (Figure 3(d)). Then, the differentiation between the populations in each habitat increases in response to selection, so that the mean genetic variance increases again, until it reaches a quasi-stationary level denoted $\tilde{V}_{\mathrm{g}}$. For parameter values such that the critical genetic variances $V_{\mathrm{g}}^{\mathrm{SCinf}}$ and $V_{\mathrm{g}}^{\mathrm{SCsup}}$ exist, we found that $\tilde{V}_{\mathrm{g}}$ was higher than $V_{\mathrm{g}}^{\mathrm{SCinf}}$, but below or above $V_{\mathrm{g}}^{\mathrm{SCsup}}$, depending on parameter values.

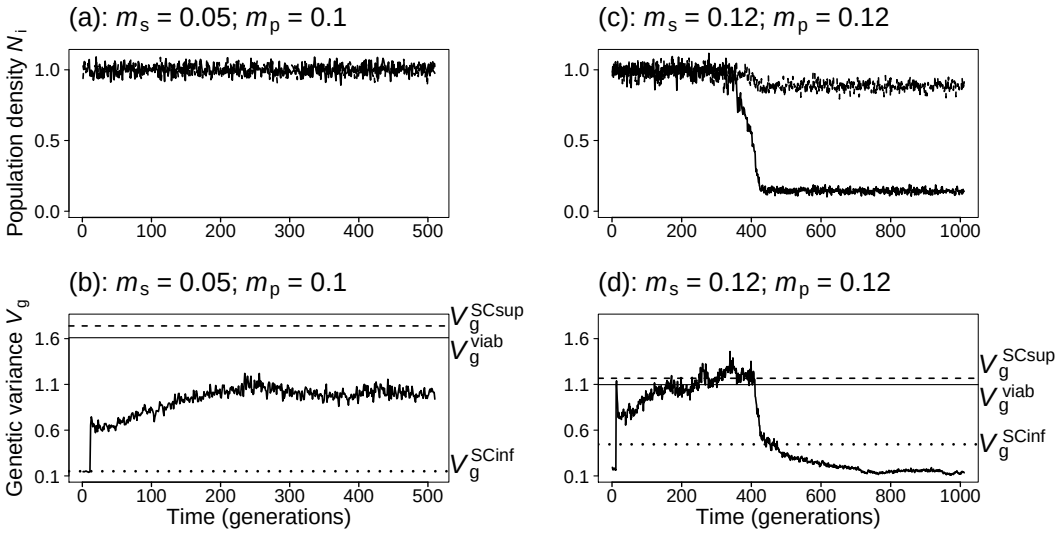


Figure 3: Typical time series (simulation model, secondary contact scenario) ending by the maintenance of the wide niche equilibrium (panels (a)-(b)) and by niche contraction (panels (c)-(d)). Panels (a) and (c) show population density before selection N_i in each habitat and panels (b) and (d) show the mean genetic variance of the populations before selection. Horizontal lines show the critical genetic variances $V_{\mathrm{g}}^{\mathrm{SCinf}}$ (dotted lines), $V_{\mathrm{g}}^{\mathrm{SCsup}}$ (dashed lines) and $V_{\mathrm{g}}^{\mathrm{viab}}$ (solid lines), computed with the analytical model for the parameter values used in these simulations. Parameter values: $K = 400$, $\Delta = 3$, $1/\omega^2 = 1$, $f = 2$, $\sigma^2 = 0.01$, $V_{\mathrm{e}} = 1$, $U = 0.1$, $L = 10$.

As predicted from the analytical model (Figure 2), the populations remain at the wide niche equilibrium as long as V_{g} remains lower than $V_{\mathrm{g}}^{\mathrm{SCsup}}$ (Figure 3(a)-(b)). In simulations where V_{g} becomes higher than $V_{\mathrm{g}}^{\mathrm{SCsup}}$ (i.e. $\tilde{V}_{\mathrm{g}} > V_{\mathrm{g}}^{\mathrm{SCsup}}$), which may occur after a long time, the populations quickly leave the wide niche equilibrium (Figure 3(c)-(d)). Such conditions, under which niche contraction is possible, correspond to high habitat heterogeneity, strong selection (Appendix D), and low to moderate total dispersal (Figure 2(a)-(b)). Once $V_{\mathrm{g}} > V_{\mathrm{g}}^{\mathrm{SCsup}}$, the growth rate of the populations was typically slightly below 1: the wide niche is lost when a stochastic demographic event induces a dispersal asymmetry large enough to result in the collapse of at least one of the two populations. This collapse is associated with a drastic decrease of the genetic variance.

In simulations where $\tilde{V}_g > V_g^{SCsup}$, we always observed that V_g became smaller than V_g^{viab}, as illustrated in Figure 3(c)-(d). The populations ultimately persist at the narrow niche equilibrium, i.e. niche contraction occurred.

Considering simulations with approximately the same quasi-stationary genetic variance $\tilde{V}_g$ but different values of V_g^{SCsup}, smaller than $\tilde{V}_g$, we found that niche contraction was more likely and more rapid for lower values of V_g^{SCsup} (Figure 4). This suggests that when V_g^{SCsup} is far from $\tilde{V}_g$, weak dispersal asymmetries are enough to destabilize the wide niche equilibrium.

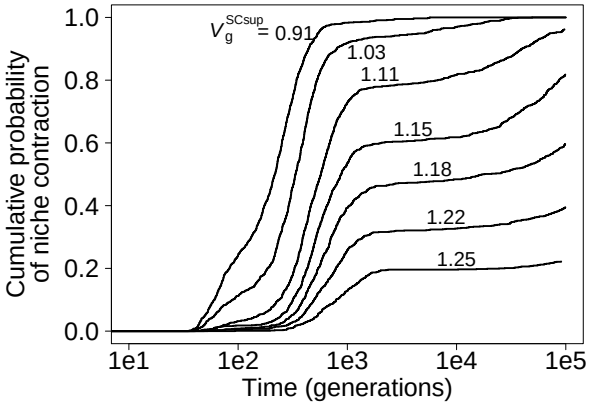


Figure 4: Cumulative distribution of the waiting time to the loss of the wide niche equilibrium (simulation model, secondary contact scenario). Each curve corresponds to a value of the critical genetic variance V_g^{SCsup} indicated next to it. The different V_g^{SCsup} are generated with different combinations of seed and pollen dispersal. In all simulations, the quasi-stationary genetic variance of the population $\tilde{V}_g$ is approximately the same, about 1.25. Parameter values: $K = 400$, $\Delta = 3$, $1/\omega^2 = 1$, $f = 2$, $\sigma^2 = 0.01$, $V_e = 1$, $U = 0.1$, $L = 10$.

Because V_g^{SCsup} decreases when m_t increases (Figure 2), and niche contraction is more likely and faster for smaller values of V_g^{SCsup} (Figure 4), unsurprisingly, we found that niche contraction occurs faster when total dispersal increases (Figure 5). In addition, Figure 5 shows that niche contraction proceeds more rapidly with increase in the proportion of pollen dispersal as a component of total gene flow. Dispersal through seeds can indeed delay niche contraction because, for the same number of gene copies dispersed, maladapted genes from immigrating seeds are more easily purged from the population than maladaptive alleles in heterozygotes formed from immigrating pollen. This is consistent with the higher genetic load with pollen dispersal observed by Lopez et al. (2008).

In simulations with parameter values such that there is no critical genetic variance V_g^{SCinf} and V_g^{SCsup} (high habitat heterogeneity, strong selection (Appendix D), and high total dispersal (Figure 2(a)-(b))), we observed that the populations always left the wide niche equilibrium, as predicted by the analytical model. We observed either niche contraction or extinction of both populations. The probability of an extinction of both populations shows a very sharp transition as seed dispersal increases (Appendix F, Figure F1). As expected from the analytical model, extinction occurs when the value of V_g^{viab} (which decreases when m_s increases, Eq 4) becomes lower than the genetic variance in the source population at the narrow niche equilibrium.

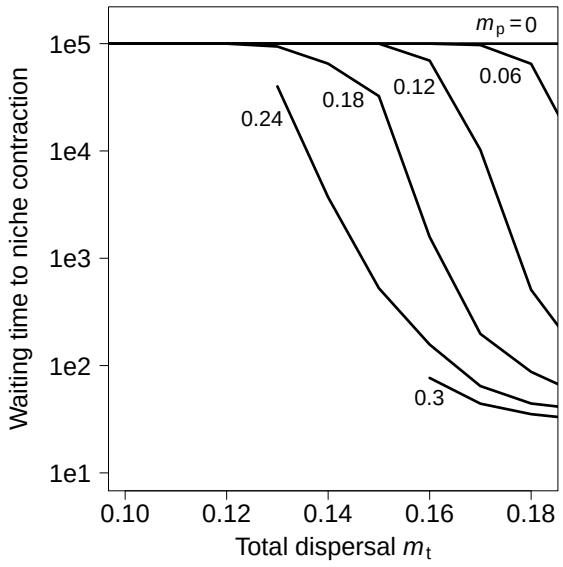


Figure 5: Mean number of generations computed from simulations before niche contraction as a function of total dispersal m_t (secondary contact scenario). Each curve corresponds to a fixed value of m_p. A waiting time of 10^5 generations indicates that the populations were still at the wide niche equilibrium when simulations were stopped. Parameter values: $K = 400$, $\Delta = 3$, $1/\omega^2 = 1$, $f = 2$, $\sigma^2 = 0.01$, $V_e = 1$, $U = 0.1$, $L = 10$.

3.2 New habitat scenario

3.2.1 Expected equilibrium at fixed genetic variance

In the new habitat scenario, the niche of the species is initially narrow: one habitat is a source where individuals are on average perfectly locally adapted and the other habitat is an empty sink where immigrants are strongly maladapted. By numerically iterating Eqs A1-A4 with initial values corresponding to this source-sink system ($N_1 = f$, $N_2 = 0$, $\bar{G}_1 = 0$, and $\bar{G}_2 = \Delta$), we can determine the parameter values for which the species' niche remains narrow, for which niche expansion occurs (i.e. the species' niche becomes wide, with both habitats as sources), and for which both populations go extinct.

For a specific combination of pollen and seed dispersal probabilities, numerical iteration of the analytical model shows that niche expansion occurs if the genetic variance is sufficiently large, but not too large (Figure 6). Increasing the genetic variance improves the response to selection in the sink, so that the sink population may eventually become sufficiently adapted to persist in its habitat. At very high genetic variance, however, the wide niche equilibrium is not stable (and possibly not viable) because selection removes too many individuals.

There are thus two critical values of the genetic variance between which niche expansion is possible. The lower, denoted V_g^{NHinf} (NH superscript stands for New Habitat scenario), is the threshold above which the narrow niche equilibrium is unstable while the wide niche equilibrium is viable, locally stable, and reachable from initial conditions corresponding to an empty sink and an adapted source. The larger critical genetic variance, denoted V_g^{NHsup}, is the threshold above which the wide niche equilibrium is unreachable from initial conditions corresponding to an empty sink and an adapted source. It is important to note that the wide niche equilibrium may be unreachable because it is inviable. In this case, the upper critical genetic variance for niche expansion is the critical variance V_g^{SCsup} already defined for the secondary contact scenario. We were unable to

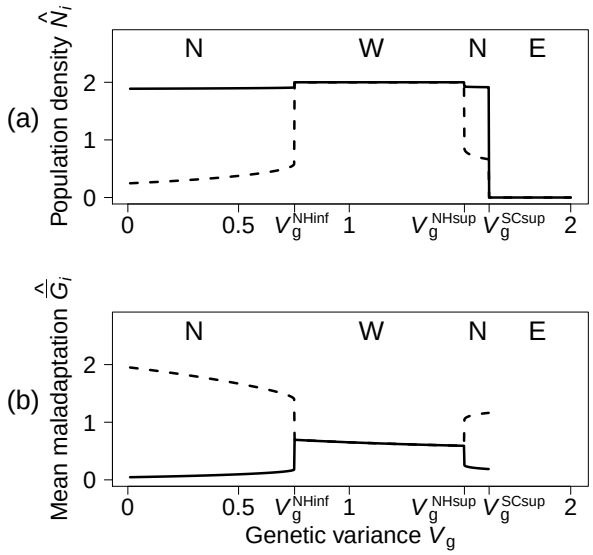


Figure 6: Population density $\hat{N}_i$ (panel (a)) and mean maladaptation $\hat{\bar{G}}_i$ (panel (b)) at the evolutionary equilibrium as a function of the fixed genetic variance V_g for the new habitat scenario. Initially, one habitat is a source (solid lines), the other one is a sink (dashed lines). Letters W, N and E indicate the parameter region corresponding to the wide niche equilibrium, narrow niche equilibrium and to extinction of both populations respectively. Results are obtained by numerically iterating Eqs A1-A4 with initial conditions corresponding to an empty sink and an adapted source ($N_1 = f$, $N_2 = 0$, $\bar{G}_1 = 0$, and $\bar{G}_2 = \Delta$). Parameter values: $m_\mathrm{s} = 0.06$, $m_\mathrm{p} = 0.3$, $\Delta = 2$, $1/\omega^2 = 1$, $f = 2$, $V_\mathrm{e} = 1$.

determine $V_\mathrm{g}^{\mathrm{NHinf}}$ and $V_\mathrm{g}^{\mathrm{NHsup}}$ analytically. We obtained them by numerical iteration of Eqs A1-A4. When $V_\mathrm{g}^{\mathrm{NHinf}}$ coincides with $V_\mathrm{g}^{\mathrm{NHsup}}$, or when there is no $V_\mathrm{g}^{\mathrm{NHinf}}$, niche expansion is not possible.

Consistent with previous literature (e.g. Ronce and Kirkpatrick, 2001), we found with the analytical model that there are values of the genetic variance allowing niche expansion for a large range of pollen and seed dispersal probabilities unless habitat heterogeneity is high and selection strong (Figure 2 and Appendix D).

The critical genetic variance $V_\mathrm{g}^{\mathrm{NHinf}}$ varies with pollen and seed dispersal (Figure 2). Whatever the proportion of pollen dispersing, increasing seed dispersal decreases $V_\mathrm{g}^{\mathrm{NHinf}}$. Because seeds contribute directly to population growth, initial demographic asymmetry between the habitats implies that seed dispersal directly increases the size of the sink population, which decreases demographic asymmetry. As a result, when m_s increases, the potential for adaptation is enhanced in the sink and compromised in the source, so that expansion of the niche can occur at lower genetic variance, i.e. $V_\mathrm{g}^{\mathrm{NHinf}}$ decreases.

At low seed dispersal, increasing pollen dispersal strongly increases $V_\mathrm{g}^{\mathrm{NHinf}}$ (Figure 2, panels (a) and (c)). Because pollen does not contribute to population growth, pollen dispersal does not directly affect population size. Pollen dispersal, however, increases maladaptation in the sink, which reinforces demographic asymmetries between the source and the sink population. The potential for adaptation is consequently compromised in the sink, so that niche expansion requires greater genetic variance, i.e. when m_p increases, $V_\mathrm{g}^{\mathrm{NHinf}}$ increases. As pollen dispersal increases, the genetic variance that would allow niche expansion may be very high. Too few individuals may then survive selection for the wide niche equilibrium to be reachable from initial conditions corresponding to an empty sink and an adapted source (Figure 2(c): $V_\mathrm{g}^{\mathrm{NHinf}}$ finally reaches $V_\mathrm{g}^{\mathrm{NHsup}}$), or even to be viable (Figure 2(a): $V_\mathrm{g}^{\mathrm{NHinf}}$ finally reaches $V_\mathrm{g}^{\mathrm{SCsup}}$).

For higher values of seed dispersal, the effect of pollen dispersal on $V_\mathrm{g}^{\mathrm{NHinf}}$ is weaker

(Figure 2(d)) because seed dispersal reduces the asymmetry in population size. In addition, increasing pollen dispersal first increases V_g^{NHinf}, then decreases V_g^{NHinf}. When pollen dispersal becomes high, pollen dispersal from the sink into the source also compromises adaptation of the source population to its local conditions. Pollen dispersed from the source has then a weaker maladaptive effect in the sink, and thus reduces demographic asymmetry. The potential for adaptation in the sink is consequently enhanced, so that niche expansion requires less genetic variance. This effect of increased pollen dispersal facilitating niche expansion appears only when seed dispersal is high enough: the sink population size is then sufficiently large, and gene flow from the sink to the source high enough, to generate significant maladaptation in the source.

The critical genetic variance V_g^{NHsup} also varies with pollen and seed dispersal: it decreases with pollen dispersal and increases with seed dispersal (Figure 2(c)-(d)).

Figure 2 summarizes the parameter region where, depending on the value of the genetic variance V_g compared to the four critical genetic variances V_g^{NHinf}, V_g^{NHsup}, V_g^{SCsup} and V_g^{viab}, the analytical model predicts that niche expansion is possible ($V_g^{\mathrm{NHinf}} < V_g < V_g^{\mathrm{NHsup}} \leq V_g^{\mathrm{SCsup}}$) or not, and, in the latter case, if populations are predicted to persist in a source-sink system or to go extinct ($V_g > V_g^{\mathrm{viab}}$). Note that there are parameter regions where the wide niche equilibrium is viable and locally stable, but not reachable from the initial conditions of the new habitat scenario (bistabilities indicated on Figure 2).

3.2.2 Dynamics of genetic variance and consequences for niche width evolution

For parameter values identified in the analytical model such that critical genetic variances allowing niche expansion exist (not too extreme habitat heterogeneity nor too intense selection, Appendix D), we found with simulations that a system initiated in a narrow niche state generally evolves to ultimately expand its niche. This can however occur after a very large amount of time. In some simulations the system was still at the narrow niche equilibrium after 10^5 generations. The shift from the narrow to the wide niche equilibrium is associated with changes in the genetic variance, which increases progressively during the period before the sink population reaches carrying capacity. Once the genetic variance in the sink population reaches the critical genetic variance V_g^{NHinf}, adaptation accelerates, and population growth in the new habitat is rapid. We observed that the genetic variance then stabilizes below the critical genetic variance V_g^{NHsup}. Figure 7 shows a typical time series illustrating these dynamics.

We found that the mean time to niche expansion depends on the difference between V_g^{NHinf} and the genetic variance V_g at the time dispersal to the new habitat begins. Higher values of parameters that increase the initial genetic variance in the source (U, σ^2, as well as V_e) decrease the time to adaptation to the sink (not shown). Figure 8 shows the cumulative distribution of waiting times to niche expansion for simulations with the same initial genetic variance but different values of V_g^{NHinf}; at lower values of V_g^{NHinf}, niche expansion proceeds more quickly. For high values of V_g^{NHinf}, niche expansion may take thousands of generations, making niche conservatism more likely on a biologically relevant time scale (environmental changes may for example occur before niche expansion can happen).

Because seed dispersal decreases V_g^{NHinf} (Figure 2), and niche expansion is faster for lower values of V_g^{NHinf} (Figure 8), seed dispersal accelerates niche expansion (Figure 9). Conversely, for most parameter values, pollen dispersal increases V_g^{NHinf} (Figure 2), and thus slows down niche expansion (Figure 9). Figure 9 also shows that for high seed and pollen dispersal, pollen dispersal accelerates niche expansion; pollen dispersal decreases

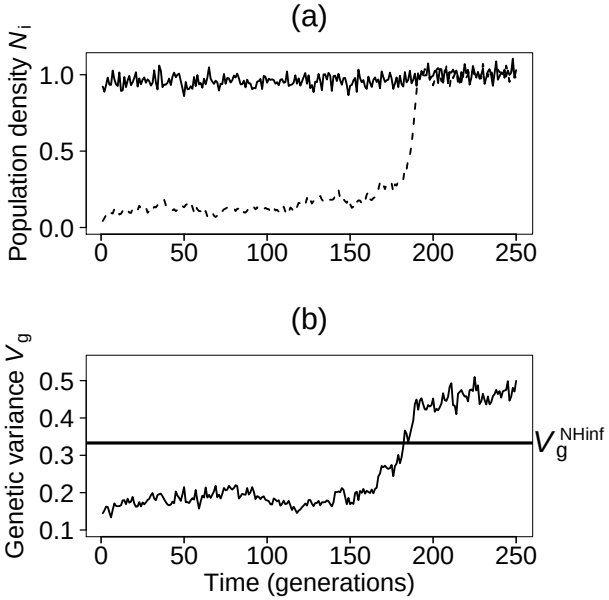


Figure 7: Typical time series from the simulation model for the new habitat scenario showing population density before selection N_i (panel (a)) and the mean genetic variance of the populations before selection (panel (b)). On panel (b), the horizontal line indicates the critical genetic variance V_g^{NHinf} (computed with the analytical model for the parameter values used in this simulation). With the parameter values used in this figure, the upper critical genetic variance for niche expansion is $V_g^{SCsup} = 1.934$. Parameter values: $m_s = 0.05$, $m_p = 0.05$, $K = 400$, $\Delta = 2$, $1/\omega^2 = 1$, $f = 2$, $\sigma^2 = 0.01$, $V_e = 1$, $U = 0.1$, $L = 10$.

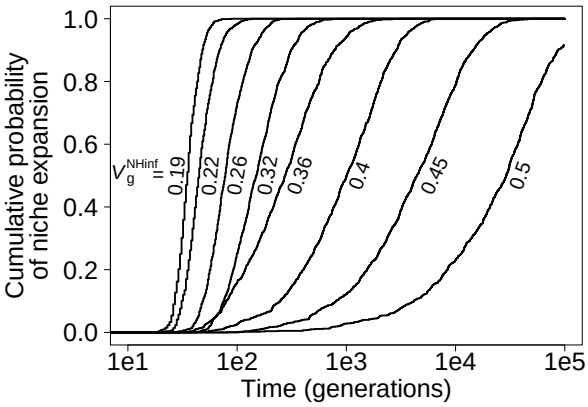


Figure 8: Cumulative distribution of the waiting time to niche expansion (simulation model, new habitat scenario). Each curve corresponds to a value of the critical genetic variance V_g^{NHinf} indicated next to it. The different V_g^{NHinf} are generated with different combinations of seed and pollen dispersal. In all simulations, at the time dispersal begins, the genetic variance of the population is about 0.21. Parameter values: $K = 400$, $\Delta = 2$, $1/\omega^2 = 1$, $f = 2$, $\sigma^2 = 0.01$, $V_e = 1$, $U = 0.17$, $L = 10$.

V_g^{NHinf} in this case (Figure 2).

In simulations with parameter values such that niche expansion was predicted as impossible by the analytical model (V_g^{NHinf} coincide with V_g^{NHsup}, or V_g^{NHinf} does not exist), the system was still at the narrow niche equilibrium after 10^5 generations, or both populations went extinct. As in the secondary contact scenario, extinction of both populations occurred when the genetic variance V_g of the source population was higher than V_g^{viab}, resulting in a sharp transition between narrow niche equilibrium and extinction

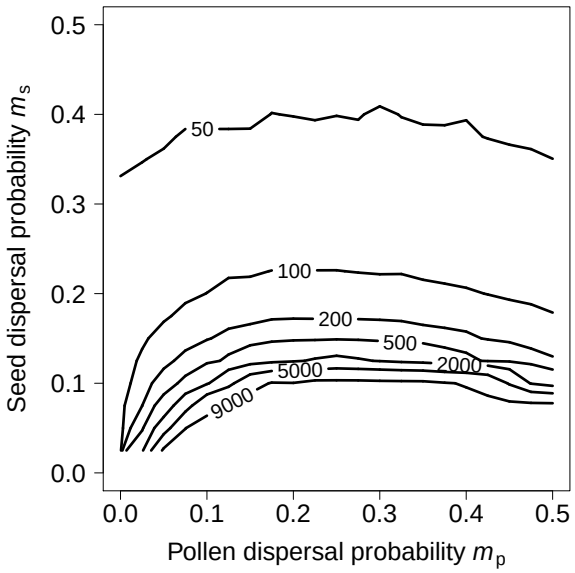


Figure 9: Mean number of generations computed from simulations before shift from narrow to wide niche equilibrium (new habitat scenario) as a function of pollen and seed dispersal probabilities. Parameter values: $K = 400$, $\Delta = 2$, $1/\omega^2 = 1$, $f = 2$, $\sigma^2 = 0.01$, $V_e = 1$, $U = 0.1$, $L = 10$.

as m_s increases (Appendix F, Figure F2).

4 Discussion

For organisms living in differentiated habitats, we find that enhancement of dispersal between the habitats can result in a shift from wide to narrow niche equilibrium or even in extinction. These shifts between equilibria are observed only when selection is severe and favours widely different phenotypes in the distinct habitats. Our model further shows that niche contraction is more likely and faster when total dispersal is high. Gene flow via pollen or seeds compromises local adaptation so that maintaining a viable population in both habitats may be impossible. Simulations show that, compared to gene flow through pollen, gene flow through seeds slows niche contraction, because seed dispersal generates a smaller dispersal load than pollen dispersal (see Lopez et al., 2008, for a detailed discussion of this phenomenon). Maladapted immigrant genes in seeds are more easily purged from the population than immigrant pollen genes, whose effects are partly masked in hybrids. However, our models also show that the higher the seed dispersal, the higher the probability of general extinction. As seed dispersal increases, and results in higher mortality of dispersing seeds, population viability depends on genetic variance being low enough to allow numerous individuals to survive selection.

Our results show that adaptation to the novel habitat requires sufficient genetic variance. When habitat heterogeneity and selection are not too strong, we found an increase of the genetic variance after dispersal to the new habitat begins, as also found in Barton (2001). This increase may take hundreds or thousands of generations when gene flow takes place predominantly via pollen dispersal. Antonovics et al. (2001) likewise found that, the greater the dispersal into a novel habitat via seeds, the faster the adaptation, whereas increasing dispersal via pollen impeded adaptation. As in Holt et al. (2003), the sink population remains maladapted until a shift to an adapted state occurs. Our results show that this shift occurs only after a minimal genetic variance is reached; adaptation

per se then proceeds quite rapidly. At high pollen dispersal, however, it can take so long to reach the higher critical genetic variance, that the environment may change before adaptation to the new niche can proceed fully. The immediate genetic effect of pollen dispersal is to increase maladaptation in the sink, and thus to reduce population size in the sink. This reduces the efficacy of selection, even though the pollen-mediated gene flow increases the genetic variation in the sink. Gene flow via pollen predominates in many plant populations, as suggested by the differentiation among populations for molecular markers transmitted via seeds versus pollen (reviewed in Petit et al., 2005). Thus, by incorporating effects of pollen dispersal, our models help to address the challenge raised by Bradshaw (1991) of accounting for the prevalence of failures of plants to adapt to novel, harsh habitats, such as soils contaminated by heavy metals.

However, for plants whose seeds disperse more profusely than pollen, we find that adaptation to a selectively distinct habitat can proceed relatively quickly, in tens of generations. Because seeds contribute directly to population size, seed dispersal directly increases the size of the sink population (as animal dispersal does, see e.g. Holt and Gaines, 1992; Kawecki, 1995; Holt, 1996a,b; Holt and Gomulkiewicz, 1997a; Ronce and Kirkpatrick, 2001; Holt et al., 2003). As a result, the potential for adaptation is enhanced in the sink and compromised in the source, so that relatively little genetic variation is required for adaptive expansion of the species' niche. Plants having seeds with wings or other appendages that facilitate wind dispersal, or those whose seeds are frequently transported by animals may thus be especially likely to rapidly expand their niche under the circumstances of our models.

Though we have motivated this study by focusing on plants, we expect analogous conclusions to apply to other organisms for which male gametes and zygotes disperse (but not female gametes), and male gametes do not limit reproduction. Our conclusions may thus apply to some haplodiploid organisms, such as wasps with both male (haploid) and female (diploid) dispersal, or fungi dispersing both as haploids and diploids. In the case of animals having sex-specific dispersal rates, Kawecki (2003) showed that the fitness in absolute sink habitats is enhanced by female-biased dispersal but reduced by male-biased dispersal. This result relies on the fact that dispersal of both sexes has genetic consequences, but in the absence of parental care and sperm limitation, only females contribute directly to population growth. This situation is analogous to pollen (male) and seed (female) dispersal and explains the qualitative convergence of Kawecki (2003)'s results and ours. Quantitative differences are however expected because of important differences in the reproductive biology. In particular, pollen is haploid, whereas, apart from haplodiploids, male animals are not, and seeds carry alleles from the gametes produced by both parents, whereas when female animals disperse before mating, their progeny receive their paternal genetic complement from males encountered after dispersal. Kawecki (2003) concluded that the ecological niche of species with female-biased dispersal should be broader than that of species with male-biased dispersal. Similarly, according to our results, we expect the ecological niche of species dispersing predominantly via seeds to be wider than that of species dispersing predominantly via pollen. With individual based simulations on a linear gradient, Butlin et al. (2003) also found that, at constant total dispersal, increasing the mating area (a circle around a female where she chooses a male for mating, which is analogous to pollen dispersal) makes the evolutionary equilibrium switch from an unlimited range to a limited range.

We know of no empirical study for which all of the components of our models have been estimated, clearly a challenging task. However, the new habitat scenario of our models may bear on the adaptation to metal-contaminated soil of some grass species from adjacent pasture populations (Bradshaw, 1991). For two species of those that have

evolved tolerance to the highly toxic metal-contaminated soils and that persist on the mine tailings, *Anthoxanthum odoratum* and *Agrostis tenuis*, gene flow via pollen between the populations on the different soils has declined greatly due to evolution of differences in flowering phenology (McNeilly and Antonovics, 1968), which have persisted for four decades (Antonovics, 2006), and to evolution of higher selfing rates on the mine tailings (Antonovics, 1968). In the case of *Agrostis tenuis*, McNeilly (1968) demonstrated ongoing seed dispersal between the two habitats. Our models suggest that such reductions of the exchange of pollen, while seeds continue to disperse, may have favored adaptation of these otherwise highly outcrossing, wind-pollinated plants to the contaminated soils. It remains to be tested whether high pollen dispersal explains the failure of adaptation for those many species that have not colonized such extreme habitats. For many invasive plants, pollen receipt is restricted via self-pollination and seeds are widely dispersed (Sakai et al., 2001), a combination that our models shows is conducive to evolutionary niche expansion.

Our findings concerning the scenario of secondary contact between diverged populations indicate that adaptation to both habitats will be maintained under many circumstances, including differences in selection that are moderate and gene flow that comprises at least some seed dispersal. This conclusion, which accounts for both demography and genetics, reinforces the view of Frankham et al. (2011), based on genetic considerations alone, that outbreeding depression threatening population persistence should be expected under only restricted conditions, including when divergent populations occupy habitats that are selectively extremely different. Accordingly, these authors advocate for management that augments gene flow between isolated populations more often than it is currently practiced. Our findings tend to support this recommendation, particularly whenever gene flow is predominantly via seeds. When habitat differences are very strong, too high seed dispersal may however lead to extinction in both habitats and should be avoided.

Our main conclusions about the effect of seed and pollen dispersal on niche evolution appear to be robust to variation of several assumptions of our model. We ran simulations with mutations drawn from a truncated normal distribution with maximal mutation effect of 0.1Δ (rather than unbounded distribution), and found that this restriction on mutation effect size did not preclude adaptation in the sink (results not shown). This indicates that adaptation in the sink did not require mutations with extreme effects (in contrast to what previous authors have concluded, under different assumptions, e.g. Holt and Gomulkiewicz, 1997b; Kawecki, 2000; Holt and Barfield, 2011; Yeaman and Whitlock, 2011). Further analyses would be necessary to study the effect of seed versus pollen dispersal on the evolution of genetic architecture during adaptation. The order of life history events is known to influence the likelihood and speed of niche evolution in animal models (Ronce and Kirkpatrick, 2001; Kawecki, 2008; Holt and Barfield, 2011). We analyzed our models with a variant life cycle (density regulation before selection). We found that niche expansion was less likely and took longer when regulation occurs before selection, and niche contraction more likely and faster (results not shown). Our qualitative conclusions remain however unaffected: a wider niche is generally predicted for species dispersing seeds rather than pollen. Likewise, our models assume a ceiling form of density regulation which eliminates density-dependence effects at low density. Using a continuous density regulation function (Beverton-Holt), we checked that our results are robust with respect to a moderately strong form of density dependence: niche contraction and niche expansion take only slightly longer with Beverton-Holt regulation (see Appendix E).

If negative density-dependence is strong in low density sink habitats, the demographic effect of seed dispersal could be globally negative, lowering the mean fitness of the populations, and preventing niche expansion. Similarly, our models assume that pollen is not limiting and, consequently, pollen dispersal has no direct demographic consequences,

while it is not unusual in nature for pollen influx to contribute directly to seed production, particularly in marginal populations (Richards, 2000; Wagenius, 2004; Knight et al., 2005). Pollen limitation may lead to an Allee effect: sufficient pollen dispersal would then be required to increase local population size, and allow the evolution or maintenance of a wide niche. Conditions of severe density-regulation or pollen limitation would likely weaken our prediction of a wider niche for species dispersing seeds rather than pollen. A detailed analysis would be necessary to clarify the respective effect of seed and pollen dispersal on niche width evolution under such conditions.

Finally, our conclusions may be altered by consideration of additional factors, such as the genetic consequences of selfing (Charlesworth and Charlesworth, 1995; Charlesworth, 2003), or the effect of gene flow on inbreeding depression (Lopez et al., 2009; Ronce et al., 2009). We should also mention that our models assume constant pollen and seed dispersal rates. Dispersal is, however, a life-history trait subject to evolution, as predicted by models (e.g. van Valen, 1971; Balkau and Feldman, 1973; McPeek and Holt, 1992; Ravigné et al., 2006) and as demonstrated by the various pollen and seed dispersal rates observed in natural populations (Ouborg et al., 1999; Petit et al., 2005). Because of its maladaptive effect, pollen dispersal could be selected against. Additional investigations would be required to determine the conditions under which pollen dispersal rate, as a trait under selection, might decrease enough to allow for niche expansion.

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Appendices

A Changes in mean phenotype and population size along the life cycle

Here, we detail the change of $\bar{G}_i$ and N_i over one generation in the analytical model. We use the superscript $^{(k)}$ to denote variables after step k of the life cycle. Assuming a Gaussian distribution of genotypic values before selection and a Gaussian probability of survival to selection, selection reduces population density and mean maladaptation, so that after selection:

$$\begin{aligned} N_i^{(1)} &= \bar{W}_i N_i \\ \bar{G}_i^{(1)} &= \left(\frac{V_\mathrm{s}}{V_\mathrm{s}+V_\mathrm{g}}\right)\bar{G}_i \end{aligned} \tag{A1}$$

where $\bar{W}_i$ is defined by Eq 1. Density regulation does not change the mean maladaptation level, and assuming a ceiling form of density regulation, we can write:

$$\begin{aligned} &N_i^{(2)} = 1 \quad \text{if} \quad N_i^{(1)} > 1, \qquad N_i^{(2)} = N_i^{(1)} \quad \text{otherwise} \\ &\bar{G}_i^{(2)} = \bar{G}_i^{(1)} \end{aligned} . \tag{A2}$$

We assume no survival cost to pollen and seed dispersal, and no habitat choice. The proportion of ovules in habitat i fertilized by pollen originating from the habitat j ($j \neq i$) is thus

$$m_{\mathrm{p}i} = \frac{m_\mathrm{p} N_j^{(2)}}{(1-m_\mathrm{p})N_i^{(2)} + m_\mathrm{p} N_j^{(2)}}.$$

Assuming that pollen is not limiting, after syngamy, population density and mean maladaptation in habitat i are

$$\begin{aligned} N_i^{(3)} &= f N_i^{(2)} \\ \bar{G}_i^{(3)} &= \bar{G}_i^{(2)} + \tfrac{1}{2} m_{\mathrm{p}i}(\Delta - \bar{G}_i^{(2)} - \bar{G}_j^{(2)}) \end{aligned} . \tag{A3}$$

Similarly, the proportion of adults in habitat i arising from seeds produced in habitat j ($j \neq i$) is

$$m_{\mathrm{s}i} = \frac{m_\mathrm{s} N_j^{(3)}}{(1-m_\mathrm{s})N_i^{(3)} + m_\mathrm{s} N_j^{(3)}}.$$

After seed dispersal, population density and mean maladaptation in habitat i are

$$\begin{aligned} N_i^{(4)} &= (1-m_\mathrm{s})N_i^{(3)} + m_\mathrm{s} N_j^{(3)} \\ \bar{G}_i^{(4)} &= \bar{G}_i^{(3)} + m_{\mathrm{s}i}(\Delta - \bar{G}_i^{(3)} - \bar{G}_j^{(3)}) \end{aligned} . \tag{A4}$$

The evolutionary equilibrium is found either by solving

$$\begin{aligned} N_i^{(4)} &= N_i \\ \bar{G}_i^{(4)} &= \bar{G}_i \end{aligned} \tag{A5}$$

or by iterating Eqs A1-A4.

B Viability and stability of the wide niche equilibrium

Here, we derive the conditions in the analytical model for the wide niche equilibrium to be viable and locally stable. We use the notation defined in Appendix A. The wide niche equilibrium is found by solving Eq A5 assuming $N_1 = N_2$. We can easily see from Eqs A1-A4 that, when $N_1 = N_2$, the population can be viable only if $N_1^{(1)} > 1$ and $N_2^{(1)} > 1$ (i.e. both habitats are saturated after selection). Consequently, the wide niche equilibrium is found by solving Eq A5 assuming $N_1 = N_2$ and $N_i^{(1)} > 1$. Under these two conditions, the changes in population density and maladaptation over one generation can be written as:

$$\begin{aligned} N_1^{(4)} &= f \\ N_2^{(4)} &= f \\ \bar{G}_1^{(4)} &= \alpha\bar{G}_1 + m_\mathrm{t}\left(\Delta - \alpha(\bar{G}_1 + \bar{G}_2)\right) \\ \bar{G}_2^{(4)} &= \alpha\bar{G}_2 + m_\mathrm{t}\left(\Delta - \alpha(\bar{G}_1 + \bar{G}_2)\right) \end{aligned} \tag{B1}$$

where $m_\mathrm{t} = m_\mathrm{s} + \frac{1}{2}m_\mathrm{p}(1 - 2m_\mathrm{s})$, $\alpha = V_\mathrm{s}/(V_\mathrm{s} + V_\mathrm{g})$ and $V_\mathrm{s} = \omega^2 + V_\mathrm{e}$. Solving Eq A5 with the above expressions leads to the wide niche equilibrium described by Eq 2. If $N_i^{(1)}$, evaluated at the wide niche equilibrium, is greater than 1, then the wide niche equilibrium is viable. Evaluating this condition leads to the viability condition given by Eq 3.

The wide niche equilibrium is locally stable if populations return to this equilibrium after infinitesimally small perturbations around it. When the wide niche equilibrium is viable, populations at this equilibrium are above the carrying capacity after selection in both habitats, i.e. $N_i^{(1)} > 1$, so that for small enough perturbations of population size before selection, $N_i^{(1)}$ is still more than 1. Considering such perturbations, their dynamics are given by the Jacobian matrix of Eqs B1, evaluated at the wide niche equilibrium defined by Eq 2:

$$\begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & \alpha(1-m_\mathrm{t}) & -\alpha m_\mathrm{t} \\ 0 & 0 & -\alpha m_\mathrm{t} & \alpha(1-m_\mathrm{t}) \end{bmatrix}. \tag{B2}$$

The four eigenvalues of the Jacobian are 0, 0, α and $\alpha(1 - 2m_\mathrm{t})$. From its definition, $0 < \alpha < 1$. Because we consider values of m_p and m_s less or equal to 0.5, $0 \leq \alpha(1-2m_\mathrm{t}) < 1$. The absolute value of all eigenvalues of the Jacobian B2 are thus less than 1 for all the parameter values used in our analysis. Therefore, when the wide niche equilibrium is viable, it is necessarily also locally stable. The condition given by Eq 3 is thus the condition for the wide niche equilibrium to be both viable and locally stable.

The above derivation ensures the local stability of the wide niche equilibrium against perturbations small enough so that both habitats remain saturated after selection. The wide niche equilibrium is however not globally stable (otherwise, the evolution of niche width would have been impossible). In particular, a system initially at the wide niche

equilibrium will not always return to the wide niche equilibrium if it is subject to perturbations large enough to cause population size to be below carrying capacity after selection in at least one of the two habitats. We define the maximal perturbation size, p^*, as the maximal proportion of individuals killed by the perturbation before selection in one of the two habitats such that a population initially at the wide niche equilibrium returns to this equilibrium after the perturbation (computed by iterating Eqs A1-A4, thus assuming a constant genetic variance). Figure B1 shows p^* for different combinations of pollen and seed dispersal: the wide niche equilibrium is stable against moderately large perturbations for a large range of parameter values, in particular when pollen dispersal is not high.

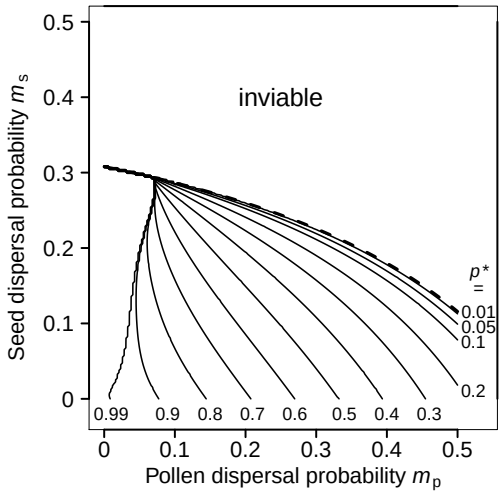


Figure B1: Isoclines of maximal perturbation size p^* as a function of pollen and seed dispersal probabilities. The dashed line corresponds to the viability limit of the wide niche equilibrium (Eq 3); above it, the wide niche equilibrium is inviable. Parameter values: $\Delta = 2.4$, $1/\omega^2 = 1$, $V_g = 0.68$, $f = 2$, $V_e = 1$.

C Approximation for viability of the narrow niche equilibrium

Here, we derive an approximation for V_g^{viab}, the critical genetic variance above which the narrow niche equilibrium is not viable. We use the notation defined in Appendix A. Our approximation relies on the assumption that, at the narrow niche equilibrium, close to extinction, the sink is a black-hole sink: there is no dispersal from the sink to the source.

Let us assume that population 1 is the source and population 2 is the sink. Assuming a black-hole sink implies that no individual survives selection in the sink, i.e. $N_2^{(1)} = 0$. We can easily see from Eqs A1-A4 that under such condition the source population can be viable only if $N_1^{(1)} > 1$ (i.e the source is above carrying capacity after selection). Solving Eq A5 assuming $N_1^{(1)} > 1$ and $N_2^{(1)} = 0$ leads to the following expressions for population density and mean maladaptation, measured before selection, at the equilibrium:

$$\hat{N}_1 = f(1 - m_s)$$
$$\hat{N}_2 = f m_s$$
$$\hat{\bar{G}}_1 = 0$$
$$\hat{\bar{G}}_2 = \Delta$$

If $N_1^{(1)}$, evaluated at the above equilibrium, is greater than 1, then the narrow niche equilibrium is viable. Evaluating this condition leads to the viability condition given by Eq 4.

D Effects of habitat heterogeneity and intensity of selection

D.1 Secondary contact scenario

Considering the analytical model, we can conclude from Eq 3 that the wide niche equilibrium is viable and locally stable when habitat heterogeneity Δ is below the threshold

$$\Delta^{\max} = \frac{1}{m_{\mathrm{t}}} \left(1 - \frac{V_{\mathrm{s}}}{V_{\mathrm{s}} + V_{\mathrm{g}}}(1 - 2m_{\mathrm{t}})\right) \sqrt{2(V_{\mathrm{s}} + V_{\mathrm{g}}) \ln(fv)}$$

where $V_{\mathrm{s}} = \omega^2 + V_{\mathrm{e}}$, $m_{\mathrm{t}} = m_{\mathrm{s}} + \frac{1}{2} m_{\mathrm{p}}(1 - 2m_{\mathrm{s}})$ and $v = \sqrt{\omega^2/(V_{\mathrm{s}} + V_{\mathrm{g}})}$. Indeed, at the wide niche equilibrium, the genetic load increases with Δ (Eq 2), so that when Δ is above $\Delta^{\max}$, the wide niche equilibrium is not viable. No value of the genetic variance may allow maintaining a wide niche as Δ increases (e.g. Figure D1(a), no $V_{\mathrm{g}}^{\mathrm{SCsup}}$ and no $V_{\mathrm{g}}^{\mathrm{SCinf}}$ for $\Delta > 3.84$).

When habitat heterogeneity Δ increases, the range of genetic variances $V_{\mathrm{g}}^{\mathrm{SCsup}} - V_{\mathrm{g}}^{\mathrm{SCinf}}$ allowing the species to persist in a wide niche state is reduced (Figure D1). The lower critical genetic variance, $V_{\mathrm{g}}^{\mathrm{SCinf}}$, increases with Δ. Indeed, the higher Δ, the more genetically different the populations in the two habitats before secondary contact, the more maladaptive the gene flow at secondary contact. Conversely, the higher critical genetic variance, $V_{\mathrm{g}}^{\mathrm{SCsup}}$, decreases with Δ. Indeed, the higher Δ, the more maladaptive the gene flow at secondary contact, the higher its demographic cost.

Similarly, there is a maximal value for $1/\omega^2$ (thus, a maximal intensity of selection) above which the wide niche equilibrium is not viable, because selection then removes too many individuals. In addition, $V_{\mathrm{g}}^{\mathrm{SCinf}}$ increases with $1/\omega^2$ and $V_{\mathrm{g}}^{\mathrm{SCsup}}$ decreases with $1/\omega^2$: the range of genetic variances $V_{\mathrm{g}}^{\mathrm{SCsup}} - V_{\mathrm{g}}^{\mathrm{SCinf}}$ allowing the populations to persist in a wide niche state is reduced as the strength of selection increases (Figure D1, comparison of the two panels).

The critical variance $V_{\mathrm{g}}^{\mathrm{viab}}$ above which the narrow niche equilibrium is not viable is approximately independent of Δ (Eq 4 is exactly independent of Δ, because it assumes that the sink is a black-hole sink, i.e. all seeds that disperse to the sink die, independently of their maladaptation in the sink). The viability of the population depends on the number of individuals surviving selection in the source: $V_{\mathrm{g}}^{\mathrm{viab}}$ decreases when selection becomes stronger (Eq 4, Figure D1).

Note that Figure D1 is drawn for a specific combination of pollen and seed dispersal probabilities. The qualitative effect of Δ and $1/\omega^2$ on the critical genetic variances is not altered when changing the value of m_{s} and m_{p} (not shown).

D.2 New habitat scenario

Considering the analytical model, Figure D1 shows that habitat heterogeneity Δ increases the critical genetic variance $V_{\mathrm{g}}^{\mathrm{NHinf}}$, until no value of V_{g} allows niche expansion ($V_{\mathrm{g}}^{\mathrm{NHinf}}$ and $V_{\mathrm{g}}^{\mathrm{SCsup}}$ coincide). Indeed, increasing habitat heterogeneity increases maladaptation in the sink, which reinforces demographic asymmetries between the source and the sink

population. The potential for adaptation is thus reduced in the sink, so that niche expansion requires greater genetic variance.

Similarly, $V_{\mathrm{g}}^{\mathrm{NHinf}}$ increases with $1/\omega^2$ (Figure D1, comparison of the two panels), until reaching $V_{\mathrm{g}}^{\mathrm{SCsup}}$. Increasing selection indeed reduces population size in the sink, which strengthens demographic asymmetries between the source and the sink populations, so that niche expansion requires greater genetic variance.

Note that Figure D1 is drawn for a specific combination of pollen and seed dispersal probabilities. The qualitative effect of Δ and $1/\omega^2$ on $V_{\mathrm{g}}^{\mathrm{NHinf}}$ is not altered when changing the value of m_{s} and m_{p} (not shown).

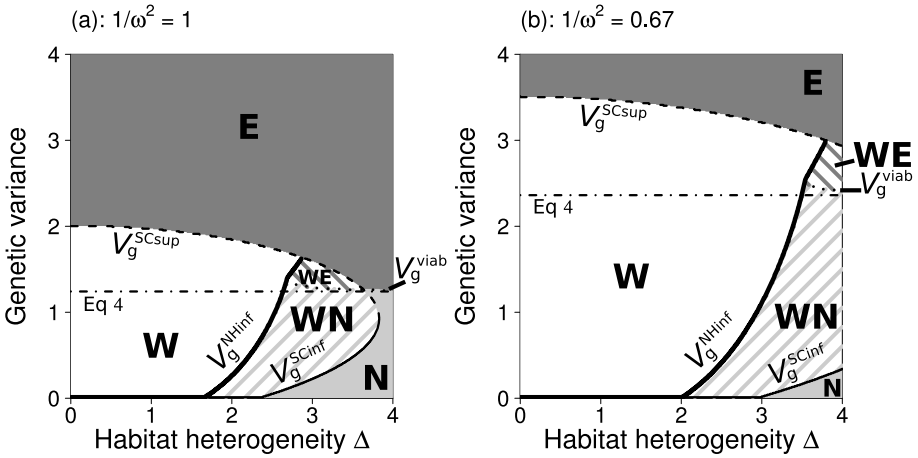


Figure D1: Critical genetic variances $V_{\mathrm{g}}^{\mathrm{SCinf}}$, $V_{\mathrm{g}}^{\mathrm{SCsup}}$, $V_{\mathrm{g}}^{\mathrm{NHinf}}$, $V_{\mathrm{g}}^{\mathrm{viab}}$ and its approximation by Eq 4, computed with the analytical model as a function of habitat heterogeneity Δ, for two different intensities of selection (panel (a): $1/\omega^2 = 1$; panel (b): $1/\omega^2 = 0.67$). With the parameter values used in this figure, the upper critical genetic variance for niche expansion is $V_{\mathrm{g}}^{\mathrm{SCsup}}$. Solid regions indicate the parameter region where only one equilibrium is viable and locally stable: only the wide niche equilibrium (W), only the narrow niche equilibrium (N), or only extinction of both populations (E). Striped regions indicate bistabilities (WN and WE). Viability and stability of the wide niche equilibrium is determined from Eq 3. Viability and stability of the narrow niche equilibrium is determined from numerical iteration of Eqs A1-A4 with asymmetrical initial values corresponding to an empty sink and an adapted source ($N_1 = f$, $N_2 = 0$, $\bar{G}_1 = 0$, and $\bar{G}_2 = \Delta$). Parameter values: $m_{\mathrm{s}} = 0.1$, $m_{\mathrm{p}} = 0.05$, $f = 2$, $V_{\mathrm{e}} = 1$.

E Results with Beverton-Holt density regulation

In this appendix, we analysed the two scenarios with a continuous density regulation function (Beverton-Holt) instead of the ceiling regulation used in the main text. All assumptions of the models remain unchanged, except Eq A2 which becomes:

$$\begin{aligned} N_i^{(2)} &= \frac{N_i^{(1)}}{1 + (f-1)N_i^{(1)}} \\ \bar{G}_i^{(2)} &= \bar{G}_i^{(1)} \end{aligned} \quad . \tag{E1}$$

E.1 Secondary contact scenario

At the wide niche equilibrium, population density and mean maladaptation, measured before selection, are:

$$\hat{N}_1 = \hat{N}_2 = \hat{N} = \frac{f\hat{\bar{W}} - 1}{(f-1)\hat{\bar{W}}}$$
$$\hat{\bar{G}}_1 = \hat{\bar{G}}_2 = \hat{\bar{G}} = \frac{m_\mathrm{t}\Delta}{1 - \frac{V_\mathrm{s}}{V_\mathrm{s}+V_\mathrm{g}}(1-2m_\mathrm{t})}$$

where $\hat{\bar{W}} = v\exp\left(-\frac{\hat{\bar{G}}^2}{2(V_\mathrm{s}+V_\mathrm{g})}\right)$, $v = \sqrt{\omega^2/(V_\mathrm{s}+V_\mathrm{g})}$, $V_\mathrm{s} = \omega^2 + V_\mathrm{e}$ and $m_\mathrm{t} = m_\mathrm{s} + \frac{1}{2}m_\mathrm{p}(1-2m_\mathrm{s})$. Solving $\hat{N} > 0$ leads to the viability condition for the wide niche equilibrium defined by Eq 3 (i.e. same viability condition as with ceiling regulation). The stability condition is however not given by Eq 3 (i.e. different stability condition from the ceiling regulation model). We were unable to find it analytically; we found numerically the critical genetic variances $V_\mathrm{g}^{\mathrm{SCinf}}$ and $V_\mathrm{g}^{\mathrm{SCsup}}$.

By contrast to the model with ceiling regulation, in the model with Beverton-Holt regulation the values of the critical genetic variance $V_\mathrm{g}^{\mathrm{SCinf}}$ and $V_\mathrm{g}^{\mathrm{SCsup}}$ depend not only on total gene flow m_t, but also on the specific pollen and seed dispersal probabilities: the range $V_\mathrm{g}^{\mathrm{SCsup}} - V_\mathrm{g}^{\mathrm{SCinf}}$ reduces as the proportion of pollen dispersal as a component of total gene flow increases (Figure E1).

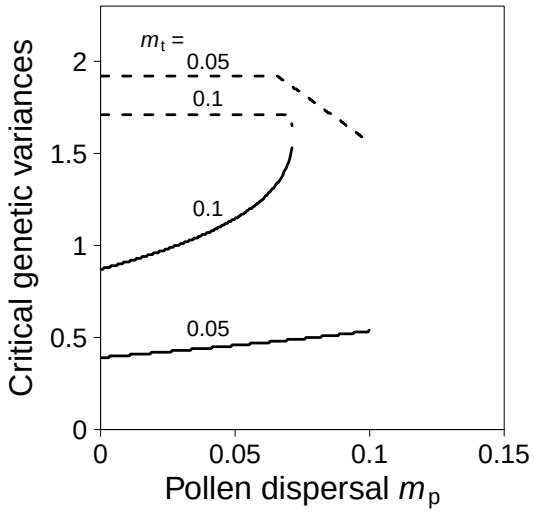


Figure E1: Critical genetic variances $V_\mathrm{g}^{\mathrm{SCsup}}$ (dashed curves) and $V_\mathrm{g}^{\mathrm{SCinf}}$ (solid curves), computed with the analytical model as a function of pollen dispersal m_p, for fixed values of total dispersal m_t indicated next to each curve. Density regulation is done according to the Beverton-Holt function defined by Eq E1. Parameter values: $\Delta = 3$, $1/\omega^2 = 1$, $f = 2$, $V_\mathrm{e} = 1$.

The range of genetic variance for which a wide niche can be maintained (i.e. $V_\mathrm{g}^{\mathrm{SCsup}} - V_\mathrm{g}^{\mathrm{SCinf}}$) is reduced compared to the model with ceiling regulation, especially at high pollen dispersal (Figure E2, compared to Figure 2).

The critical genetic variance for the viability of the narrow niche equilibrium, $V_\mathrm{g}^{\mathrm{viab}}$, computed assuming that the sink is a black-hole sink, leads to Eq 4 (i.e. same approximation as with ceiling regulation).

Despite the effect of Beverton-Holt regulation on the critical genetic variances $V_\mathrm{g}^{\mathrm{SCsup}}$ and $V_\mathrm{g}^{\mathrm{SCinf}}$, Figure E3 shows that the waiting times to niche contraction are weakly affected by Beverton-Holt regulation compared to ceiling regulation.

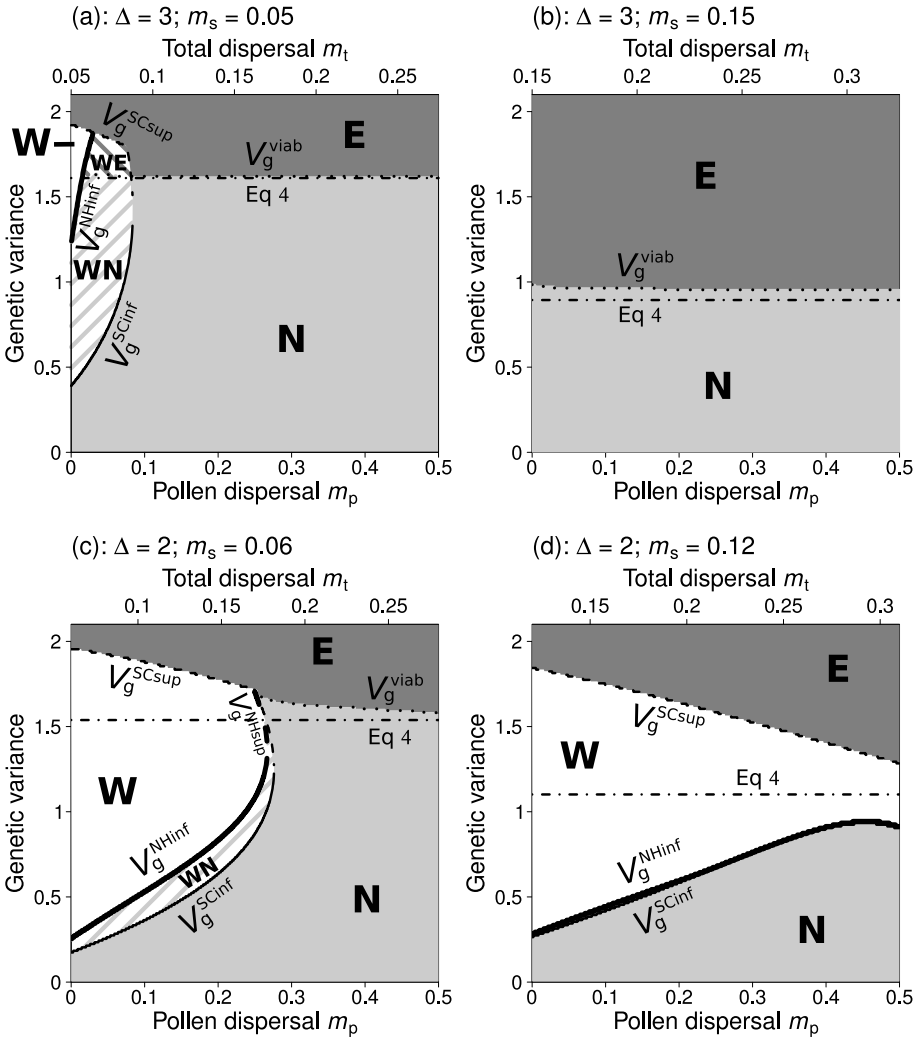


Figure E2: Critical genetic variances V_g^{SCinf}, V_g^{SCsup}, V_g^{NHinf}, V_g^{NHsup}, V_g^{viab} and its approximation by Eq 4, computed with the analytical model as a function of pollen dispersal m_p, for fixed values of seed dispersal m_s and habitat heterogeneity Δ. Density regulation is done according to the Beverton-Holt function defined by Eq E1. Solid regions indicate the parameter region where only one equilibrium is viable and locally stable: only the wide niche equilibrium (W), only the narrow niche equilibrium (N), or only extinction of both populations (E). Striped regions indicate bistabilities (WN and WE). Viability and stability of the wide niche equilibrium is determined from numerical iteration of the analytical model with almost symmetrical initial conditions ($N_1 = \dfrac{fv-1}{(f-1)v}$, $N_2 = \dfrac{fv-1}{(f-1)v} - 0.001$, $\bar{G}_1 = 0$, and $\bar{G}_2 = 0.001$), mimicking a small perturbation at secondary contact of the two populations. Viability and stability of the narrow niche equilibrium is determined from numerical iteration of Eqs A1-A4 with asymmetrical initial values corresponding to an empty sink and an adapted source ($N_1 = \dfrac{fv-1}{(f-1)v}$, $N_2 = 0$, $\bar{G}_1 = 0$, and $\bar{G}_2 = \Delta$). On panel (d) V_g^{SCinf} and V_g^{NHinf} are very close and indistinguishable on the graph. Parameter values: $1/\omega^2 = 1$, $f = 2$, $V_e = 1$.

E.2 New habitat scenario

The critical genetic variance V_g^{NHinf} above which niche expansion is possible is increased by Beverton-Holt regulation, compared to ceiling regulation (Figure E2, compared to Figure 2). In addition, the range of parameter values for which V_g^{NHinf} exists is reduced, especially at high pollen dispersal.

Despite these differences, Figure E4 shows that the waiting times to niche expansion

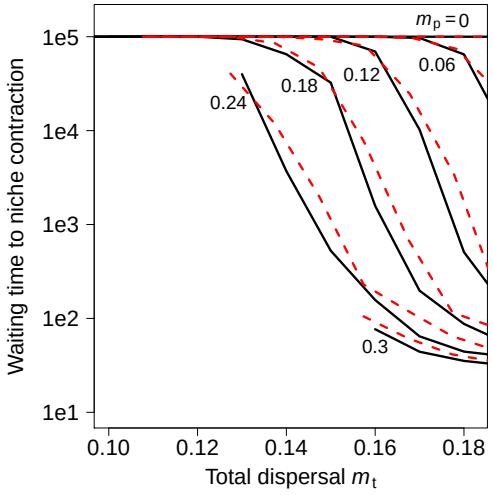


Figure E3: Mean number of generations computed from simulations before niche contraction as a function of total dispersal m_t (secondary contact scenario). Solid black curves: ceiling regulation (same data as in Figure 5). Red dashed curves: Beverton-Holt regulation (Eq E1). Each curve corresponds to a fixed value of m_p. A waiting time of 10^5 generations indicates that the populations were still at the wide niche equilibrium when simulations were stopped. Parameter values: $\Delta = 3$, $1/\omega^2 = 1$, $f = 2$, $\sigma^2 = 0.01$, $V_e = 1$, $U = 0.1$, $L = 10$. Ceiling regulation: $K = 400$. Beverton-Holt regulation: $K = 800$. Note that K is chosen such that population size after density regulation is similar in the model with ceiling regulation and in the model with Beverton-Holt regulation. Demographic stochasticity is consequently of the same order of magnitude.

are only very longer with Beverton-Holt regulation compared to ceiling regulation.

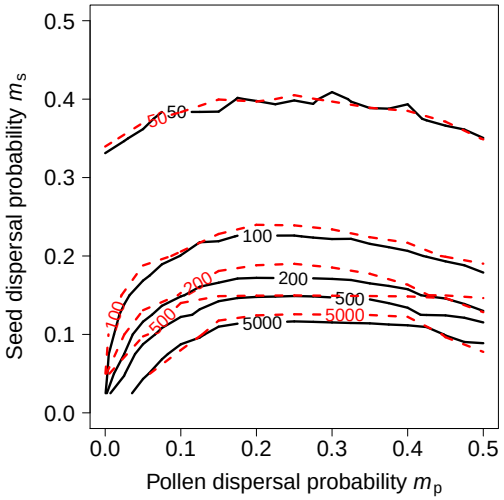


Figure E4: Mean number of generations computed from simulations before shift from narrow to wide niche equilibrium (new habitat scenario) as a function of pollen and seed dispersal probabilities. Solid black curves: ceiling regulation (same data as in Figure 9). Red dashed curves: Beverton-Holt regulation (Eq E1). Parameter values: $\Delta = 2$, $1/\omega^2 = 1$, $f = 2$, $\sigma^2 = 0.01$, $V_e = 1$, $U = 0.1$, $L = 10$. Ceiling regulation: $K = 400$. Beverton-Holt regulation: $K = 800$. Note that K is chosen such that population size after density regulation is similar in the model with ceiling regulation and in the model with Beverton-Holt regulation. Demographic stochasticity is consequently of the same order of magnitude.

F Extinction of both populations

F.1 Secondary contact scenario

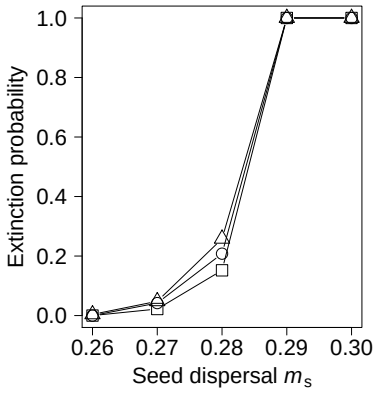


Figure F1: Probability of extinction of both populations as a function of seed dispersal probability m_s (simulation model, secondary contact scenario). Parameter values are such that maintenance of a wide niche is predicted impossible by the analytical model (no critical genetic variance V_g^{SCinf} and V_g^{SCsup}): $m_p = 0.05$ (squares), 0.1 (circles), 0.15 (triangles), $K = 400$, $\Delta = 3$, $1/\omega^2 = 1$, $f = 2$, $\sigma^2 = 0.01$, $V_e = 1$, $U = 0.1$, $L = 10$. With these parameter values, for m_s between 0.26 and 0.3, V_g^{viab} drops from 0.1904 to 0. When extinction occurs, the time to extinction is very short, a few dozen of generations.

F.2 New habitat scenario

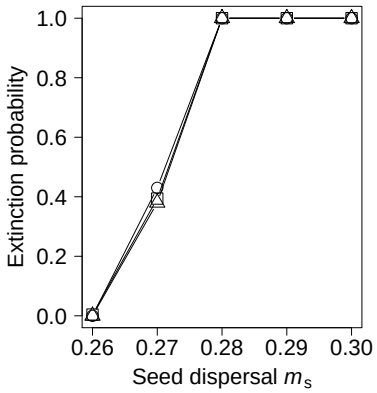


Figure F2: Probability of extinction of both populations as a function of seed dispersal probability m_s (simulation model, new habitat scenario). Parameter values are such that niche expansion is predicted impossible by the analytical model (no critical genetic variance V_g^{NHinf}): $m_p = 0.05$ (squares), 0.1 (circles), 0.15 (triangles), $K = 400$, $\Delta = 3$, $1/\omega^2 = 1$, $f = 2$, $\sigma^2 = 0.01$, $V_e = 1$, $U = 0.1$, $L = 10$. With these parameter values, for m_s between 0.26 and 0.3, V_g^{viab} drops from 0.1904 to 0.