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Gene flow and the limits to natural selection

Thomas Lenormand

In general, individuals who survive to reproduce have genotypes that work relatively well under local conditions. Migrating or dispersing offspring elsewhere is likely to decrease an individual's or its offspring's fitness, not to mention the intrinsic costs and risks of dispersal. Gene flow into a population can counteract gene frequency changes because of selection, imposing a limit on local adaptation. In addition, the migrant flow tends to be higher from densely populated to sparsely populated areas. Thus, although the potential for adaptation might be greatest in poor and sparsely populated environments, gene flow will counteract selection more strongly in such populations. Recent papers, both theoretical and empirical, have clarified the important role of migration in evolution, affecting spatial patterns, species ranges and adaptation to the environment; in particular, by emphasizing the crucial interaction between evolutionary and demographic processes.

Thomas Lenormand
CEFE – CNRS, 1919 route de Mende, 34293 Montpellier cedex 05, France.
e-mail: thomas.lenormand@cefe.cnrs-mop.fr

Under natural selection, individuals tend to adapt to their local environmental conditions, resulting in a pattern of LOCAL ADAPTATION (see Glossary). Local adaptation can occur if the direction of selection changes for an allele among habitats (antagonistic environmental effect), but it might also occur if the intensity of selection at several loci that are maintained as polymorphic by recurrent mutations covaries negatively among habitats. These two possibilities have been clearly identified in the related context of the evolution of senescence but have not have been fully appreciated in empirical and theoretical studies of local adaptation [1,2].

The interaction between directional selection and gene flow

When an allele with antagonistic environmental effects is maintained at a MIGRATION–SELECTION EQUILIBRIUM, gene flow changes allele frequencies in a direction opposite to natural selection, and each population is suboptimally adapted: that is, there is a MIGRATION LOAD. For small amounts of migration, this load per locus approximately equals the migration rate. If the migration rate is large compared with selection, the polymorphism is lost. When such GENE SWAMPING occurs, the alleles with the best average reproductive success across all populations tend to become fixed; therefore there is no local adaptation

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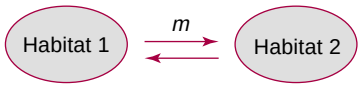
Box 1. Migration–selection model with two demes and two alleles

Figure I illustrates a model [a] where allele *A* has an advantage *s* over allele *a* in habitat 1 and a disadvantage αs in habitat 2. A fraction *m* (the migration rate) of individuals from each habitat is exchanged each generation. With antagonistic environmental effect ($\alpha > 0$), *A* is favorable in one habitat and *a* in the other. If migration is strong enough relative to selection (*m*/*s* larger than a critical value), the allele with the largest fitness averaged over both habitats will tend to become fixed (e.g. it will be *A* if $0 < \alpha < 1$ and $s > 0$). The critical value for *m*/*s* is $\alpha / |1-\alpha|$, which increases when the two alleles are on overall equally fit (α close to 1). If *m*/*s* is less than this critical value, a polymorphism is maintained by a balance between migration and selection. If *m*/*s* is greater than this critical value, the best overall allele 'swamp' the other.

Fig. I



Relative fitness of allele	*A*	1+*s*	1–αs
	a	1	1

If α is negative then *A* is either deleterious or advantageous in both habitats, and recurrent mutation is necessary to maintain a polymorphism. I considered here only the interaction between allelic effect and the environment. However, fitness can also depend on the interaction between alleles at the same or different loci. For instance, if selection occurs on diploids, marginal over- or underdominance can produce other types of stable polymorphism among habitats.

Reference

a Bulmer, M.G. (1972) Multiple niche polymorphism. *Am. Nat.* 106, 254–257

even though selection varies over space. Gene swamping occurs even in simple models, where population density is not affected by the amount of selection or by the amount of dispersal (SOFT SELECTION models). In the simplest possible model that exhibits gene swamping, there are two habitats, soft selection, one locus with antagonistic environmental effect and three parameters: (1) the amount of migration *m* (assuming that migration is symmetrical); (2) the intensity of selection *s* in one habitat; and (3) the ratio of selection coefficients between the two habitats α (Box 1). Gene swamping occurs if $m/s > \alpha/(1-\alpha)$ for α chosen such that $\alpha < 1$ [3]. Thus, if the selection coefficients do not perfectly balance between habitats ($\alpha < 1$), gene swamping occurs in a large portion of the parameter space.

This basic model can be elaborated introducing an asymmetry in density or migration between the two habitats; this tends to favor the allele that is advantageous in the high density, low immigration deme [3]. Another important illustration of gene swamping comes from models of a continuous habitat with a constant density of individuals. An allele that is beneficial in a pocket of the environment might decrease in frequency and be lost even where it has a local selective advantage (Box 2). This type of model shows that there are two crucial combinations of parameters determining migration–selection equilibria: one integrating the relative spatial scale of migration and selection and the other combining the different asymmetries among habitats.

Evidence for migration load and gene swamping

Mayr's observation [4] that phenotypic divergence is often correlated with the degree of isolation is consistent with the idea that gene flow keeps divergence in check by opposing the effect of natural selection. This can also be explained by the action of genetic drift or by variation of selection pressure with distance. Evidence for a migration load comes from more precise studies. The study of gene frequency patterns across a sharp transition in the environment (e.g. presence or absence of heavy metals in habitats of *Agrostis tenuis* and *Anthoxantum odoratum* [5]) often results in the observation of smooth clines ([6] for other examples). The width of these clines is typically larger than the environmental transition and can be used to estimate the opposing effects of selection and gene flow (i.e. $\sigma/\sqrt{s}$; Box 2). Transplantation experiments can also provide a good estimate of the opposing effects of dispersal on selection provided that the scale of the transplantation is carried out at the scale of dispersal (this requires an independent measure of dispersal [7,8]). Further evidence for a migration load is that the degree of local adaptation (measured experimentally) correlates with the degree of isolation of populations [9,10]. More directly, temporal variation in the parameters (gene flow and selection pressure) generates variation in local adaptation that is consistent with a migration–selection model [11,12].

Gene swamping is an extreme form of migration load. However, it is much more difficult to demonstrate because it requires one to show, in the absence of a genetic polymorphism, that the genetic potential for local adaptation exists. Gene swamping can be inferred, however, if local adaptation occurs only in sufficiently large or isolated environments. For instance, *Culex* mosquitoes in Corsica do not exhibit insecticide resistance in spite of the occurrence of the selection pressure and the presence, at low frequency, of resistance genes that have spread elsewhere. This situation is best explained by gene swamping, because the insecticide-treated area in this region is small compared with the dispersal distance of *Culex* [13].

When the environment changes with distance

The maladaptive effects of dispersal depend strongly on how selection varies over space. In models where dispersal is a function of distance, one has to specify how selection varies with distance. This specification has an enormous impact on the theoretical predictions. For instance, in a model of Felsenstein [14] where the optimum for a quantitative trait varies

Box 2. Environmental pockets of adaptation

Figure I shows the frequency pattern (dashed line) of an allele *A* that has an additive fitness advantage of *s* in a 1D environmental 'pocket' of size 2*a* and is deleterious elsewhere by an amount αs. The model allows the density (dotted line) outside the environmental pocket to be τ times the density inside the pocket. Gene flow is measured by σ, the standard deviation of parent–offspring distance measured along one dimension. At equilibrium, the maximum frequency of the beneficial allele within the environmental pocket, p_{max}, depends on two combinations of the parameters (Fig. II) [a,b]. The first combination (y axis on Fig. II) measures the relative scales of the spatial heterogeneity (a) and of the 'characteristic length' ($\sigma/\sqrt{s}$), which weighs the strength of selection relative to gene flow. The second combination (x axis on Fig. II) is a measure of overall asymmetry between habitats (ratio of selection coefficients and ratio of densities outside/inside the pocket). Figure II is read as a contour-plot for p_{max}. The blue area corresponds to the parameter space where gene swamping occurs: allele *A* is absent ($p_{max} = 0$) from the geographical pocket in spite of its local selective advantage. Gene swamping occurs if the relative scales parameter is small and the asymmetry parameter large. The density ratio outside/inside the pocket (τ) strongly magnifies the ratio of selection coefficients outside/inside the pocket (α): the asymmetry parameter is $\tau^2\alpha^{1/2}$, which indicates that α plays the same role as the fourth power of τ. However, the asymmetry between habitats has a limited impact on local adaptation: p_{max} is almost independent from the x axis when the asymmetry parameter is > ~3 (e.g. the curve $p_{max} = 0$ tends toward $\pi/2$). The parameter combination leading to a maximum frequency p_{max} between 0 and 0.5 is quite narrow (green area): allele *A* is likely to be either absent or present at high frequency.

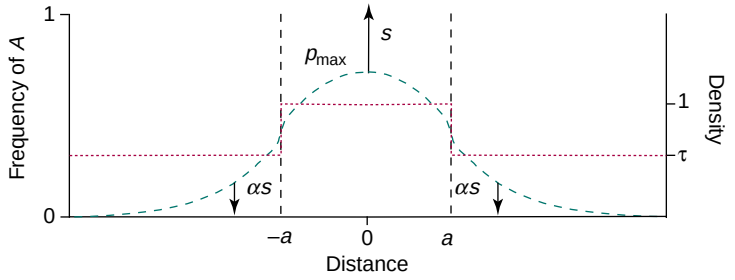


Fig. I TRENDS in Ecology & Evolution

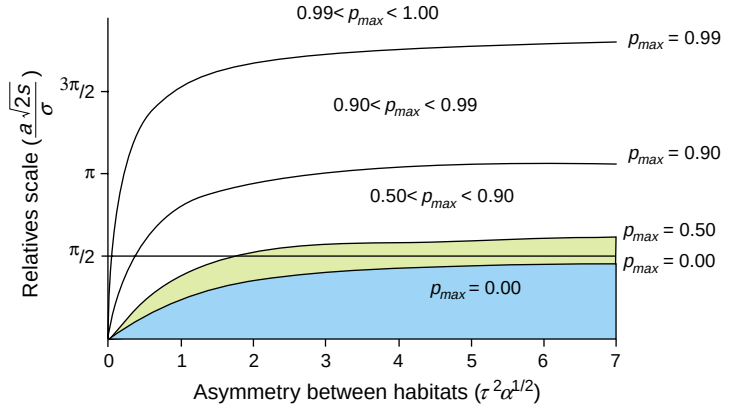


Fig. II TRENDS in Ecology & Evolution

The above results for one locus can be extended to a quantitative trait under stabilizing selection. Each underlying locus can be viewed as a locus with alleles with a small effect that exhibits antagonistic pleiotropy over space. As a consequence, a quantitative trait with additive variance V_a under stabilizing selection of intensity *s* cannot significantly track environmental change under a critical geographical scale, which is proportional to a 'characteristic length' $\sigma/\sqrt{(V_a s)}$ [c].

Reference

a Nagylaki, T. (1975) Conditions for the existence of clines. *Genetics* 80, 595–615

b Nagylaki, T. (1978) Clines with asymmetric migration. *Genetics* 88, 813–827

c Slatkin, M. (1978) Spatial patterns in the distributions of polygenic characters. *J. Theor. Biol.* 70, 213–228

linearly along a single dimension (with constant and equal densities everywhere and homogeneous dispersal), the population mean of the trait matches perfectly the optimum regardless of the amount of migration. This extreme result is obtained because, in any location, the genetic and phenotypic change caused by migrants from the right and the left cancel each other out exactly. Perfect matching is not observed, however, if the environment is finite or if the gradient is nonlinear, or if the underlying genes do not contribute additively to the phenotype. The repeated occurrence of wide genetic clines on different continents has been used to infer that selection pressures vary with distance (e.g. continental body size clines in *Drosophila subobscura* [15]). The migration load occurring within these clines has not been estimated but can be substantial because of linkage disequilibria (i.e. when the environmental gradient and dispersal are strong) or edge effects (peripheral population or populations at the edge of wide clines are likely to exhibit maladaptation because of a relatively low density [16]).

The effect of genetic parameters, life cycle and mating system

Several other important biological parameters influence the impact of migration. First, an allelic effect might depend on the presence of other alleles at the same (dominance) or different (epistasis) locus, as well as on the environment. These two types of interaction have a similar and potentially large effect on migration–selection equilibria. For instance, the range of parameters where gene swamping occurs is much larger for alleles whose local advantage is recessive [17], especially when segregation occurs between migration and selection within the life cycle. Marginal underdominance because of different dominance coefficients among environments could lead to migration–selection equilibria that are locally stable but that are dependent on initial conditions. In these cases, gene swamping could occur very rapidly following a large perturbation in allele frequencies (e.g. following a bottleneck).

Second, migration produces associations between the selected alleles. Within-loci associations, often measured by heterozygote deficit, are removed efficiently by segregation at meiosis. However, between-loci associations, often measured by linkage disequilibria, are only partially removed by recombination at meiosis. For alleles whose effects covary positively over space, migration creates a local excess of extreme genotypes in fitness (i.e. positive linkage disequilibrium). Recombination tends to reduce this disequilibrium and thus also the variance in fitness. As a consequence, the efficacy of selection is lower and the migration load higher with high recombination rates [18–21], especially when recombination occurs between migration and selection within the life cycle. Consequently, the migration load will tend to be higher in models assuming many

Box 3. Evolution out of a niche

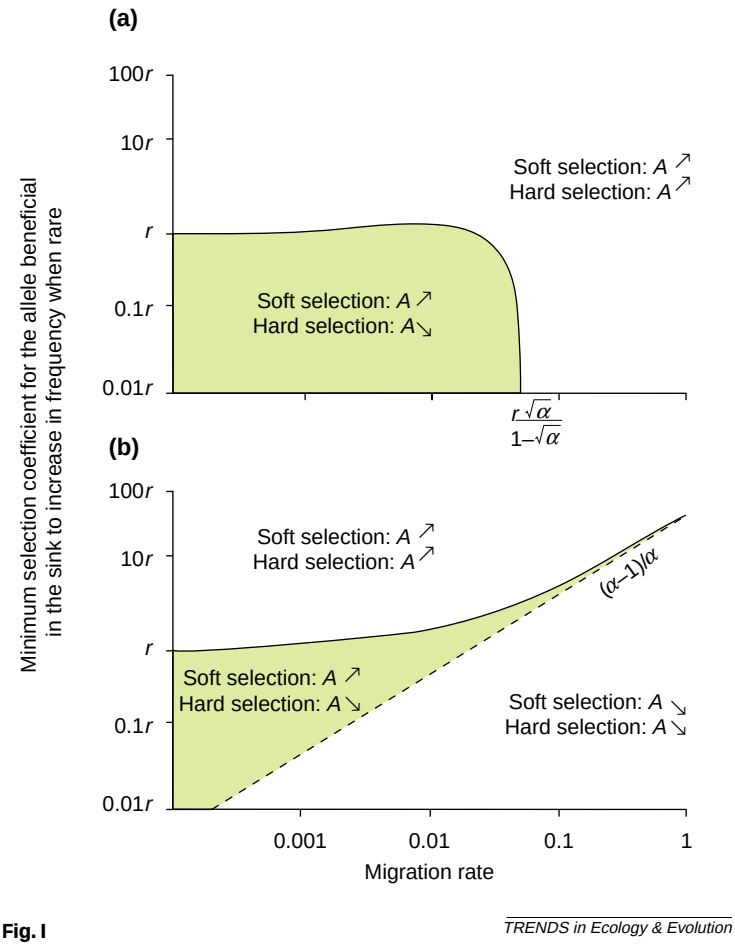


Fig. I

Here, I consider the fate of an allele *A* that increases the intrinsic growth rate in habitat 1, *r*, by an amount *s* but reduces intrinsic growth rate in habitat 2, *r′*, by an amount αs (Table I).

The two habitats are connected by migration at rate *m* per individual. In the absence of *A*, only habitat 2 has a positive growth rate ($-r<0$ and $r'>0$) and is self sustaining (the population in habitat 1 is maintained by the recurrent immigration from habitat 2). For simplicity, regulation by density occurs only in habitat 2. I compare this hard selection model [a] with the soft selection model presented in Fig. I in Box 1, where population sizes in habitats 1 and 2 are considered constant and equal.

In Fig. I, the solid and dashed curves indicate the minimum selection intensity (y axis) required for the allele *A* to increase when rare for a given migration (x axis) and a given ratio of selection coefficients α under hard and soft selection, respectively.

Figure Ia considers the case where *A* is more advantageous in habitat 1 than disadvantageous in habitat 2 ($\alpha<1$), whereas Fig. Ib considers the opposite case ($\alpha>1$). When *A* is, on average, advantageous (Fig. Ia), it always increases in frequency when rare in the soft selection case. It might even fix if migration is high enough (Box 1). However, this is no longer true with hard selection: *A* is lost in the green area. With hard selection, *A* increases in frequency only if it is beneficial enough to compensate for the negative growth rate in habitat 1, especially when migration is so low that only a small fraction of the total population is exposed to selection in habitat 1. When *A* is, on average, deleterious (Fig. Ib), it increases in frequency when rare in the soft selection case only if migration is low enough (i.e. above the dashed line, see Box 1). However, as in Fig. Ia, with hard selection and low migration, allele *A* also has to be beneficial enough to compensate for the negative growth rate in habitat 1. In both Fig. Ia and b, the conditions under which *A* increases in frequency are more stringent under hard than under soft selection (because *A* is the allele beneficial in the bad habitat) and the difference between hard and soft selection decreases when the intensity of selection (*s*) or migration (*m*) increases.

Table I

		Habitat 1	Habitat 2
Intrinsic rate of increase	*a*	$-r$	r'
	A	$-r+s$	$r'-\alpha s$

References

a Kawecki, T.J. (2000) Adaptation to marginal habitats: contrasting influence of the dispersal rate on the fate of alleles with small and large effects. *Proc. R. Soc. London B Biol. Sci.* 267, 1315–1320

unlinked loci of small effects (e.g. quantitative genetics models [22,23]) than in single-locus models [17,24].

The mating system can also strongly modify the impact of migration. When locally adapted genotypes tend to have a greater mating success, the genetic differences among populations are accentuated, which generally reduces the migration load. For instance, sexual selection might increase dramatically the reproductive success of locally adapted males [25]. Assortative mating could also increase the mating success of the most common genotypes, because they encounter suitable mates more often. This mating system will tend to favor the locally adapted genotypes, because they are likely to be common where they are favored. However, assortative mating can prevent local adaptation whenever the locally adapted genotype happens to be rare (e.g. when a mutation arises [25], in situations close to gene swamping, or simply by chance [26]). In addition, the mating system might not change the mating success of individuals but only the genetic correlations between mates, which thus affects the variance in fitness. For instance, positive assortative mating (e.g. because of an assortment trait, such as flowering time [26], behavioral imprinting [27] or selfing [28]) will, in general, increase the variance in fitness and thus reduce the migration load. Overall, any parameter that increases the efficacy of selection, by increasing either the relative fitness of locally adapted genotypes or the local genetic variance in fitness, will reduce the migration load.

Relaxing soft selection hypotheses

When the assumptions of soft selection are relaxed (i.e. under HARD SELECTION), the qualitative impact of gene flow on adaptation is less straightforward; introducing more realistic demography within local adaptation models has interesting, but complicated, consequences. For clarity, I separate the interaction

between mean fitness and density and the interaction between migration and density, although both effects occur simultaneously (Box 3).

Migration meltdown: linking density and mean fitness

When local density is a monotonically increasing function of mean fitness, a positive feedback loop can occur that magnifies the effect of gene swamping: populations with a lower-than-average density receive a proportionately higher-than-average influx of maladapted alleles, their mean fitness decreases, which reduces their density further, and so on. Thus, asymmetries in gene flow can become more extreme over time, which destabilizes migration–selection equilibria. For example, differences in density occurring by chance (e.g. following an environmental perturbation) can arbitrarily cause one allele to swamp another. This MIGRATION MELTDOWN is similar to the feedback loop occurring in mutation meltdown models [2], and indeed the two might work in concert. Migration meltdown might also be enhanced by a mating system that confers a mating advantage to the most common genotype [25].

Migration meltdown has been proposed as an explanation for constraints on species range [22]. In this model, a quantitative trait whose optimum changes with distance is considered. Because density decreases when mean fitness decreases, a migration meltdown occurs at the species boundary whenever migration is too large. The same process occurs in models considering the evolution of ecological specialization. An accidental change in population size or habitat availability can be followed by a migration meltdown, resulting in a niche switch or reduction [23].

Direct evidence for a migration meltdown is almost entirely absent and probably very difficult to obtain. Studies of the butterfly *Euphydryas editha* in California [29] document that a niche switch between the two main host plants of this butterfly occurred after an unusual summer frost, which killed all the larvae on the preferred host plant *Collinsia*. In the subsequent years, the butterflies consistently reproduced on the other host plant *Pedicularis* in spite of normal weather conditions. The reason for this switch could be because recolonization of *Collinsia* is difficult (the life cycle on *Pedicularis* occurs later in the season, which creates a temporal barrier to migration) but it might also be due to genetic changes that were induced by the frost [23].

Migration rescue: the movement of individuals

When gene flow results directly from a movement of individuals (e.g. seed dispersal but not pollen dispersal when pollen is not limiting), asymmetric gene flow can have demographic consequences. With all individuals having the same probability of migrating, habitats of lower-than-average density will have a net increase in density after dispersal and vice versa. By decreasing density differences among habitats, the migration of individuals opposes the demographic effects of a migration meltdown [23]. Net immigration can compensate for the reduction in density caused by maladaptation to local conditions. Whether the demographic or genetic effect of migration dominates depends ultimately on the strength of the relationship between density and adaptation, about which we often know very little. It is this type of contradictory effect that has to be balanced in agronomy or conservation biology when some populations are artificially supplemented with maladapted individuals for a demographic reason [30,31].

At one extreme, dispersal can sustain some populations at non-zero densities that would otherwise go extinct [32]. This situation could have profound evolutionary consequences, because it might allow the niche of a species to evolve. Dispersal brings individuals into new environments and therefore makes adaptation to these new conditions possible. This effect, known as 'evolution-out-of-a-niche' [24,33,34], is particularly important for alleles that confer an advantage in the new habitat that is not great enough for the population to be self-sustaining in that habitat without immigration. This kind of allele can spread only with very high rates of dispersal (Box 3).

A genetic rescue effect can also occur because dispersal increases genetic variance, which is necessary for adaptation but which might otherwise be absent in small peripheral populations. This effect has been quantified by Gomulkiewicz *et al*. [34], who showed that local adaptation can proceed faster with intermediate migration rates. Dispersal can also reduce the deleterious effects of inbreeding acting in small peripheral populations [35], although this effect might be weak compared with the migration load owing to local adaptation [28].

The interaction of gene flow and disruptive selection

A migration load can also be generated by interactions among alleles without any effect of the environment: immigrant alleles might simply not work well with combinations of local alleles. This occurs when there are alternative adaptive peaks within a single habitat. Such a disruptive selection pattern can be caused by underdominance, strong epistasis or, more generally, selection against rare genotypes [36]. The transition between directional and disruptive selection is gradual and depends on the relative depth of the 'adaptive valley' and the altitude difference between the peaks. Moreover, both types of selection can lead to the same pattern (e.g. the same migration–selection equilibria [37]) and have been often grouped and referred to as outbreeding depression or hybrid breakdown [38]. Their relative importance has largely been evaluated (and debated) in the context of hybrid zones using crosses and transplants [39]. However, decomposing the factors causing outbreeding depression within species could be achieved by the same method, which would be useful for evaluating the effect of gene flow on adaptation.

Glossary

Gene swamping: loss of the genetic variance at a locus under selection because gene flow is too high. *Sensu stricto*, gene swamping cannot occur when genetic variance is maintained by recurrent mutations (e.g. in quantitative genetic models). However, the concept can used *sensu lato* to describe situations where there is no significant response to selection because gene flow is too high. This occurs when the spatial scale of environmental heterogeneity is below a 'characteristic length' [a] equal to $\sigma/\sqrt{s}$ (Box 2).
Hybrid sink effect: self-reinforcing process in which immigration causes mating between two subspecies, which produces unfit hybrids, which decreases local density, which increases immigration rate and so on.
Local adaptation: a better average performance of individuals born in the habitat in which the measure is done, compared with the performance of immigrants.
Migration load: decrease in mean fitness of a population because of immigration. This occurs because the phenotypic mean of the population is different from the local optimum value.
Migration meltdown: self-reinforcing process in which immigration brings locally maladapted alleles, which decreases local density, which increases immigration rate and so on.
Migration–selection equilibrium: stable polymorphic equilibrium in which the frequency change caused by migration cancels out the frequency change caused by selection.
Soft and hard selection: soft selection refers to models where population density is not affected by the amount of selection or by the amount of dispersal; hard selection refers to any other cases.

Reference

a Slatkin, M. (1978) Spatial patterns in the distributions of polygenic characters. *J. Theor. Biol.* 70, 213–228

One of the most controversial debates in evolutionary biology has been the role of gene flow in adaptation under disruptive selection between at least two fitness peaks of different height (a process referred to as the shifting balance or SBP [40]). Gene flow plays a crucial role in shifting balance models; it has to be both low enough for a peak shift to occur (so that drift enables alleles to cross the 'valley') and high enough for a high-fitness peak to spread [41]. Thus, adaptation by the SBP, if it occurs, requires rather intermediate levels of gene flow. Demographic effects of migration might help or hinder the SBP. If deme density increases with mean fitness, demes near the high fitness peak might contribute more to the migrant pool [42] and spread more rapidly. However, a similar process to the migration meltdown, the HYBRID SINK EFFECT [43], can decrease local gene flow in demes at the boundary between populations on different peaks (because such demes will tend to fall in the fitness valley between the peaks). A local reduction in gene flow (a barrier) can dramatically slow the spread of an adaptive peak. The presence of a barrier increases the effect of disruptive selection because migrants are rarer on the other side of the barrier and are thus more heavily selected against. Barriers can therefore prevent the spread of a high fitness peak [43]. Overall, gene flow can severely limit adaptation when there is a significant amount of disruptive selection whenever migration rates are too high, too low or too irregular over space. That hybrid zones often coincide in space with natural barriers is consistent with this last effect [44].

Indirect effect of the migration load

The fitness load because of migration can be very high in a heterogeneous environment, which creates a strong indirect selection pressure to suppress it. The migration load can be alleviated by a genetic amelioration process [45], whereby the effects of genes are modified directly (e.g. by increased dominance [46] or decreased deleterious pleiotropic effects [47]).

This migration load also generates a weak indirect selection pressure that favors any trait that reduces gene flow between habitats (e.g. increased assortative mating, increased habitat choice, reduced movement rate, etc.), a mechanism which works in the same way as reinforcement. However, these traits can be classified into two categories depending on whether they have to differ between habitats to reduce gene flow (one- versus two-allele mechanisms [48]). For instance, habitat preference can be caused by habitat-specific alleles (e.g. an individual might prefer habitat A because it carries allele *A* (a two-allele mechanism)) or by alleles that are not habitat specific (e.g. an allele which causes individuals to prefer the habitat where they were born (a one-allele mechanism)). The indirect selection pressures caused by the migration load tend to be weak and thus might, in general, be insufficient to oppose gene flow and produce divergence among populations of a reinforcement trait [26], although there is evidence that it can occur [49]. As a consequence, one-allele mechanisms (e.g. reduced dispersal [28,50], increased plasticity [1,51,52], reduced recombination [20,21], imprinting-based mating or habitat preferences) might be more likely to evolve to reduce the migration load than are two-allele mechanisms.

These different processes can occur only if there is a migration load. However, most of them cannot operate if there is gene swamping. For instance, the amelioration process cannot occur if the polymorphism at the selected loci is swamped by gene flow. In this case, the rate of adaptation is determined by the rate of appearance of beneficial mutations that have few deleterious pleiotropic effects in the different habitats. To some extent, gene flow might prevent evolution by mutations of small effect; assuming that a small beneficial effect in one place is more easily swamped by deleterious effects elsewhere, spatial heterogeneity in selection might therefore bias upward the average effect of alleles that underlie adaptation [53].

Dispersal evolution

Dispersal, which is the ultimate source of migration load, is selected against in a heterogeneous habitat. Gene flow is therefore only a proximate factor limiting adaptation, the ultimate factors being the selective forces (or constraints) that favor dispersal. Dispersal might simply be unavoidable, but it can also be directly favored for different reasons. First, dispersal can evolve because the environment varies in time and space (to escape crowding [54] or as a risk-spreading strategy [55]). Second, dispersal could evolve as an altruistic trait to reduce kin competition [56]. Third, dispersal could be favored to escape the fitness depression caused by mating among relatives when there are many deleterious recessive mutations [57]. Fourth, gene flow could be favored because it

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accelerates the spread of advantageous mutations. These different forces are likely to interact [58], and there is no global theoretical synthesis that includes all of them.

Conclusion

Gene flow tends to oppose the effects of local selection and thus limits adaptation. However, it can also replenish the local population and local genetic variation, which are both pre-requisites for evolution by natural selection. The relative importance of these effects is not yet clear. A more thorough evaluation of the spatial scale of the heterogeneity of selection among habitats and of the type of selection (directional or disruptive) is necessary to evaluate the role of gene flow on adaptation. Finally, manipulating adaptation by controlling gene flow might have simple and direct applications, either to disrupt local adaptation of pests to pesticide [19] or to maintain local adaptation of agronomical or endangered species [31,59].

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