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E-Article

Expansion Load and the Evolutionary Dynamics of a Species Range

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abstract: Expanding populations incur a mutation burden, the so-called expansion load. Using a mixture of individual-based simulations and analytical modeling, we study the expansion load process in models where population growth depends on the population's fitness (i.e., hard selection). We show that expansion load can severely slow down expansions and limit a species' range, even in the absence of environmental variation. We also study the effect of recombination on the dynamics of a species range and on the evolution of mean fitness on the wave front. If recombination is strong, mean fitness on front approaches an equilibrium value at which the effects of fixed mutations cancel each other out. The equilibrium rate at which new demes are colonized is similar to the rate at which beneficial mutations spread through the core. Without recombination, the dynamics is more complex, and beneficial mutations from the core of the range can invade the front of the expansion, which results in irregular and episodic expansion. Although the rate of adaptation is generally higher in recombining organisms, the mean fitness on the front may be larger in the absence of recombination because high-fitness individuals from the core have a higher chance to invade the front. Our findings have important consequences for the evolutionary dynamics of species ranges as well as on the role and the evolution of recombination during range expansions.

Keywords: range expansion, invasion, genetics, mutation load, expansion load, genetic drift.

Introduction

The ranges of all species have fluctuated during their history. Shifts in the boundaries of species ranges can result from a variety of ecological and evolutionary processes (MacArthur 1972), and now occur owing to rapid climatic or environmental changes (e.g., Thomas et al. 2001; Parmesan 2006; Yamano et al. 2011; Pateman et al. 2012). Understanding the dynamics of species range limits has important applications, for instance, for predicting the expansions of invasive species (Alexander and Edwards 2010) and expected responses to climate change (Parmesan et al. 2005; Sekercioglu et al. 2008).

There has been a strong focus on identifying and understanding the ecological and evolutionary processes that cause range expansions, range shifts, or contractions (e.g., Sexton et al. 2009). More recently, the effects of range expansions on several evolutionary processes have been empirically and theoretically studied. These processes include the evolution of dispersal (Lindström et al. 2013; Lombaert et al. 2014), life-history traits (Burton et al. 2010), or cooperation (Datta et al. 2013). The growing appreciation of the consequences of dynamic range margins on the ecology (Brown et al. 2013), population genetics (e.g., Excoffier et al. 2009), and behavior (Lindström et al. 2013) of species has changed our views about several evolutionary processes, such as the evolution of dispersal (Shine et al. 2011) and the spatial structuring of biodiversity (Waters et al. 2013) or genetic diversity (Excoffier et al. 2009).

In expanding populations, individuals that arrive first in new habitats are likely ancestors of later generations living in the same area (Moreau et al. 2011), and the processes at range margins allow neutral genetic variants to quickly spread into new territories (Klopfstein et al. 2006). This phenomenon—called gene surfing—has been documented in several species, including humans (Moreau et al. 2011), tortoises (Graciá et al. 2013), and experimental microbial populations (Hallatschek and Nelson 2010), and it has been invoked in the explanation of patterns of genetic diversity in humans (Hofer et al. 2009).

Positively or negatively selected variants can also surf (Travis et al. 2007; Lehe et al. 2012), which may increase the ability to explore complex fitness landscapes (Burton and Travis 2008). If multiple deleterious mutations cosegregate, deleterious mutations can accumulate during range expansions and create a so-called expansion load (Peischl

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et al. 2013).These findings may help explain many interesting phenomena, such as a reduced potential to adapt to novel environments (Pujol and Pannell 2008) and patterns of intraspecific variation in inbreeding depression (Pujol et al. 2009) and mutation load (Peischl et al. 2013).

Although the expansion load is transient, it can prevail for thousands of generations if selection is soft (Peischl et al. 2013). Under soft selection (sensu Wallace 1975), deleterious mutations do not affect a population ability to reproduce and survive. In that case, expansion load can increase indefinitely without causing a mutation meltdown (Lynch et al. 1995) or affecting a species ability to expand its range. This is in clear contrast with models of hard selection, where demographic parameters—such as growth rates and carrying capacities—do depend on the genetic composition of the population. When hard selection is operating, we would therefore expect two key differences in the expansion dynamics. First, there should be an absolute limit on the mutation load: individuals with too many deleterious mutations will not be able to survive or reproduce, independently of the fitness of conspecifics. Second, because the efficiency of selection on the wave front increases with decreasing growth rate (Hallatschek and Nelson 2010; Peischl et al. 2013), there should be a negative feedback between the dynamics of expansion load and the expansion process itself. It is, however, unclear how such a feedback would affect the evolutionary dynamics of mutation load across the range of an expanding species.

We extend here the model developed by Peischl et al. (2013) to allow for growth rates and carrying capacities to depend on the local population's mean fitness. Using a mixture of individual-based simulations and analytical approximations, we investigate the effect of hard selection on expansion load and the evolutionary dynamics of a species range. We study the effect of recombination on the dynamics of the mutation load itself and on adaptation at the edge of expanding populations.

Models and Results

Simulation Model

Life Cycle. We model a population of diploid monoecious individuals that occupy discrete demes located on a one- or two-dimensional grid (the stepping stone model; e.g., Kimura and Weiss 1964). Selection is uniform in space. Generations are discrete and nonoverlapping. Mating within each deme is random: mating pairs are formed by randomly drawing individuals (with replacement) according to their relative fitness, and each pair produces a single offspring. This process is repeated N_j' times for deme j, where N_j' is the total number of offspring in the next generation. The number of offspring per individual is therefore approximately Poisson distributed. Individuals then migrate to adjacent demes with probability m per generation. Migration is homogeneous and isotropic with reflecting boundaries.

Selection. Each gamete carries k_d (k_b) new deleterious (beneficial) mutations, where k_d (k_b) is drawn from a Poisson distribution with mean u_d (u_b). We denote the genome-wide mutation rate $u = u_d + u_b$. Mutations are randomly distributed over n independently segregating regions. Within these regions, sites are assumed completely linked, and each new mutation falls on a new site (infinite site model). This model should be a good approximation for the evolution of sexual populations with linear chromosomes (Weissman and Hallatschek 2014). We denote by $\varphi_d = u_d/u$ the probability that a new mutation is deleterious and by $\varphi_b = 1 - \varphi_d$ the probability that it is beneficial. We assume that mutation effects are drawn from the same distribution of fitness effects for all individuals (independently from their current fitness) and that beneficial and deleterious mutations have symmetric effects s and $-s$, respectively. Note that the effect of using more complex distributions of fitness effects is examined in appendix E (appendixes A–F available online). Fitness effects are multiplicative, such that the relative fitness of an individual is given by $w = \prod_i (1 + s_i)$, where s_i is the selection coefficient associated with the ith mutation (i.e., there is no dominance or epistasis). Mean fitness of deme j is denoted $\bar{w}_j$. Whenever it is clear from the context, we will simply write $\bar{w}$ and omit the dependence of mean fitness on j. In the following, we will use subscript f to indicate properties of demes that are on the front of the species range.

Population Dynamics and Absolute Fitness. Population growth of a deme depends on both its density and mean fitness. The expected number of offspring in the next generation produced by the N_j adults in deme j is

$$N_j^* = R_j \bar{w}_j N_j. \tag{1}$$

R_j is the deme's basic (geometric) growth rate, which captures the effects of density dependence:

$$R_j = \frac{R_0}{1 + (R_0 \bar{w}_j - 1) N_j / K_j},$$

where R_0 is the fundamental growth rate. K_j is the deme's carrying capacity, which we also assume depends on mean fitness:

$$K_j = \min\left(K_0 \bar{w}_j, K_{\max}\right).$$

The actual number of offspring, N_j', is then drawn from a Poisson distribution with mean N_j^*.

According to the growth equation (1), the population grows logistically if $R_j > 1$, and it declines if $R_j < 1$. Cases

where fitness affects either carrying capacities or growth rates (but not both) are discussed in appendix F.

Simulation Setup. We performed individual-based simulations in one- or two-dimensional habitats. Initially, the five left-most demes (or five left-most columns in two-dimensional simulations) are at carrying capacity, and all other demes are empty. The expansion starts after a burn-in phase of $10K_0$ generations to reach mutation-selection-drift equilibrium. Unless stated otherwise, we assumed that 90% of all nonneutral mutations were deleterious, which seems conservative (Eyre-Walker and Keightley 2007). Under this assumption, the mean fitness is expected to decrease on the expansion front for a wide range of reasonable parameter combinations (Peischl et al. 2013). We mainly focus here on a single parameter combination for illustrative purposes. Additional simulation results for different parameter combinations are shown in "Dynamics of Mean Fitness on the Wave Front" and in appendix E.

Evolution of Mean Fitness and Dynamics of Expansion. Figure 1 illustrates our main results. It shows two representative examples of the evolution of the mean fitness in a population expanding in a one-dimensional habitat, with and without recombination. In both cases, we see a very strong contrast in the rate of adaptation between core and peripheral populations.

In asexual organisms, the dynamics of the range expansion shows an episodic pattern (fig. 1*A*). The progressive accumulation of deleterious mutations slows down the expansion and may even stop it when front populations suffer from mutation meltdown (Lynch et al. 1995). Eventually, fitter individuals from the core invade the front, which allows the population to initiate a new expansion, but the load builds up again, and the process begins anew. The result is a pulse-like process of repeated colonization and extinction, causing the species range to grow in an irregular way.

With recombination, expansion load also gradually slows down the speed of the advancing wave front (approximately until $t = 2{,}000$ generations in fig. 1*B*). Rather than stopping completely, the front eventually progresses at a slower but constant rate (approximately when $t > 2{,}000$ generations in fig. 1*B*). The rate of adaptation in the core is much higher with than without recombination (cf. fitness in fig. 1), as expected from fundamental theory (e.g., Felsenstein 1974).

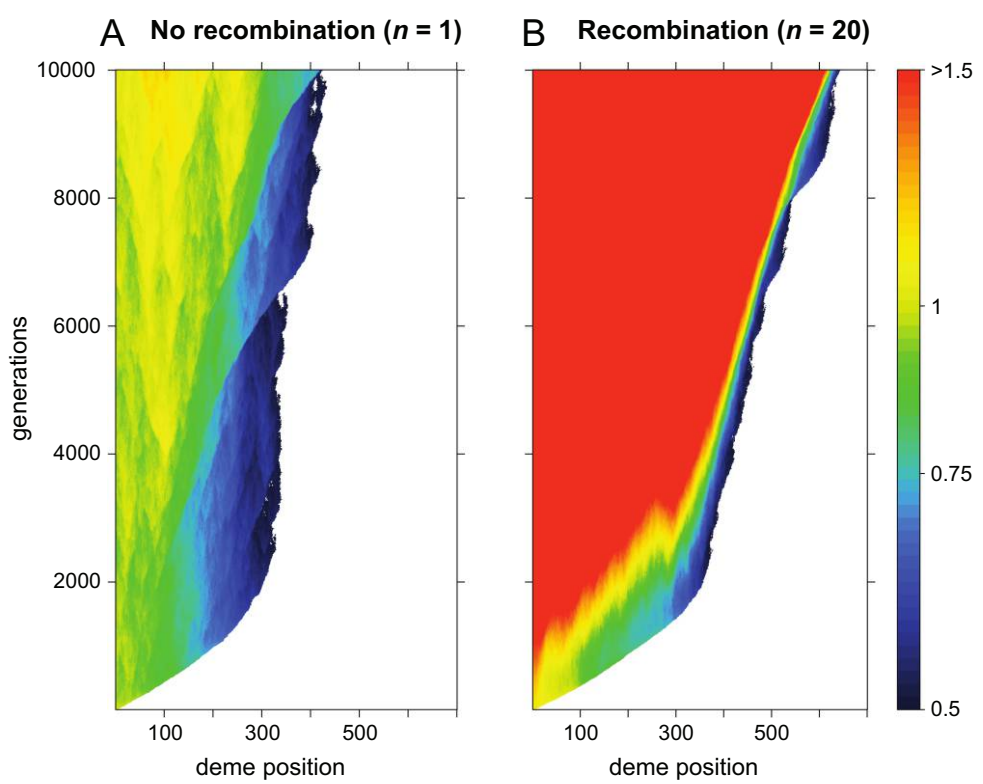

Figure 1: Evolution of the mean fitness during a range expansion. Fitness is normalized to the mean fitness at the onset of the expansion. *A*, Nonrecombining species. *B*, Species with $n = 20$ freely recombining regions. Parameter values: $s = 0.005$, $m = 0.05$, $K_0 = 100$, $R_0 = 2$, $u = 0.05$, $\varphi_d = 0.9$, $K_{max} = 200$.

Figure 1 shows an example where recombination leads to (slightly) higher mean fitness on the wave front and hence enables the species to expand at a faster rate than without recombination (see also fig. 2*A*). This is, however, not always the case. In the absence of recombination, high-fitness migrant individuals from the core may invade the front, spread into empty territories, and temporarily restore a higher mean fitness at the wave front (see figs. 1*A*, 2). If selection is weak, these invading lineages provide only a slight increase in mean fitness (red line in fig. 2*A*). If selection is sufficiently strong, however, invading lineages from the wake of the wave (carrying a lower number of deleterious mutations) can lead to a substantial increase in front mean fitness (colored lines fig. 2*B*) and hence accelerate the expansion over long periods. Contrastingly, high-fitness migrants rarely invade the front in presence of recombination, since the effects of beneficial mutations are rapidly diluted when these migrants mate with local individuals (fig. 1*B*).

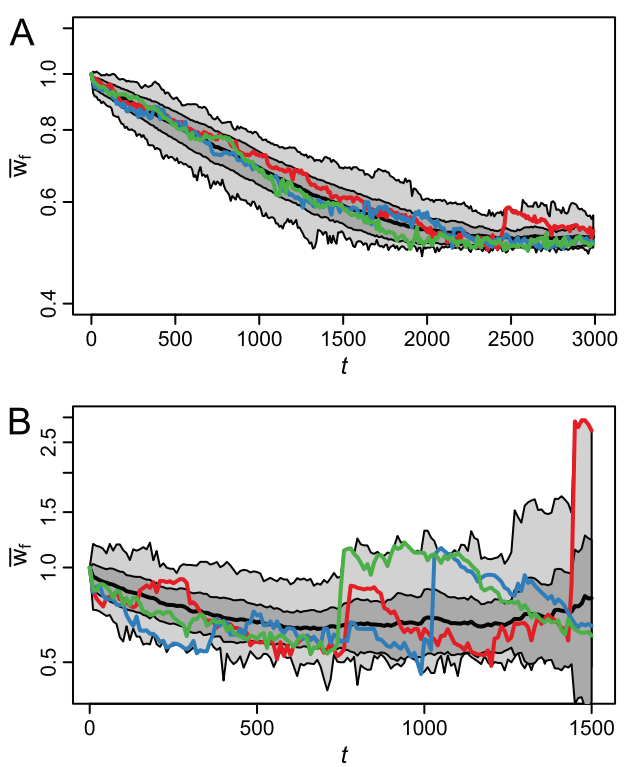

Figure 2: Evolution of the mean fitness at the front of a range expansion for nonrecombining species. Colored lines show mean fitness on the wave front for three replicates. Solid black lines indicate the mean over 50 simulations. Shaded areas show 1 SD (dark gray) and the minimum and maximum of all observed values (light gray). *A*, Simulation parameters are as in figure 1*A* ($s = 0.005$). *B*, Similar to *A* except that $s = 0.025$.

Effect of Recombination. Without recombination, colonization of new habitats is sometimes triggered by fit migrants from the wake of the wave (generation 6,000 in fig. 1*A*). With recombination, however, it is unclear how gene flow influences the wave front dynamics seen in figure 1*B*. We therefore recorded the spatial origin of mutations carried by individuals living on the wave front. We categorize mutations into two classes: mutations that first occurred in an individual living on the wave front and mutations that first occurred in an individual living in the wake of the wave. We define the wave front as the set of demes that have been colonized within the past five generations. This definition of the wave front is arbitrary, but other choices yield qualitatively and quantitatively very similar results. We denote by L the total number of deleterious mutations carried by individuals living on the wave front and by L_f the number of deleterious mutations that first occurred in an individual that lived on the wave front and that are now carried by individuals living on the front. We can then define the relative contribution of mutations originating from the wave front by

$$D_f = \frac{L_f}{L}.$$

Thus, D_f can be considered as the fraction of mutation load on the front that arose from the expansion process. Analogously, we define B_f as the fraction of beneficial mutations that originated on the wave front. We interpret D_f and B_f as measures of the relative contribution of ancestors that lived on the wave front to the mean fitness of individuals currently living on the front. If D_f (or B_f) is close to 0, most of mutation load (or adaptation) on the wave front is due to migrants from the core. In contrast, if D_f (B_f) is close to 1, most of the genetic variation contributing to mutation load (adaptation) can be traced back to ancestors who lived on the wave front.

Figure 3 shows the evolution of D_f and B_f. Initially, both D_f and B_f increase because the front moves so fast that it is essentially isolated from the core, and new mutations steadily accumulate on the wave front. Recombination has essentially no effect on the front of the expansion during this stage of the expansion process because diversity on the front is extremely low. Note that B_f increases much more slowly than D_f because the deleterious mutations rate is larger than the beneficial mutation rate. When the expansion reaches its equilibrium speed around 2,000 generations, both D_f and B_f reach a maximum and then start to decrease. The fact that both D_f and B_f decrease implies that mutations from the core have invaded the front. This invasion is facilitated by the slowdown of the expansion. Importantly, this means that at equilibrium, the dynamics of the expansion process is diffusive rather than discrete.

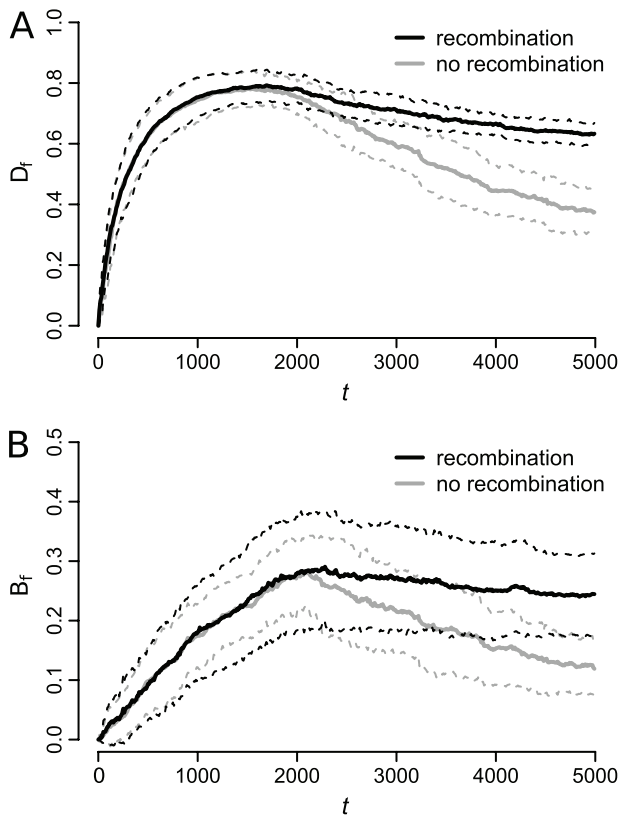

Figure 3: Evolution of the fraction of deleterious (*left*) and beneficial (*right*) mutations that originate on the wave front. Solid lines show the means, and dashed lines indicate ± 1 SD. Parameters are as in figure 1.

The decrease of D_f and B_f is more pronounced in the absence of recombination (fig. 3), implying that recombination prevents the establishment of both deleterious and beneficial core mutations on the wave front. Recombination reduces the rate of hitchhiking of deleterious mutations whose ancestry can be traced to core populations, leading to the differences observed in figure 3. Recombination also reduces the rate at which core beneficial mutations invade the front, because it breaks apart the genome of potential core invaders and dilutes the selective advantage of invading lineages. This explains the observed behavior of B_f in figure 3*B*.

Two-Dimensional Expansions. We also performed simulations in a two-dimensional habitat of 10×500 demes (fig. 4). In general, these lead to patterns similar to the one-dimensional case. The two main differences are that in two-dimensional habitats the expansion load is less severe (fig. E1*A*; figs. E1–E4, F1–F6 available online), and recombination has a weaker effect on the evolution of D_f and B_f (cf. figs. 3*A*, 3*B*, E1*B*, E1*C*). These two differences are probably due to the larger effective population size on the wave front in two-dimensional expansions, which increases the efficiency of selection and leads to a slower buildup of the expansion load and to a higher fitness at equilibrium. In contrast to one-dimensional expansions, there is a variation in fitness on the front of two-dimensional expansions, and demes on the wave front can be invaded by fitter individuals coming from demes that are either in the core or on the wave front as well. This explains why the difference between recombining and nonrecombining organisms is less pronounced than in one-dimensional expansions (cf. figs. 3, E1*B*, E1*C*).

Analytical Model

To better understand the dynamics of expansion load, we next derive results in a simplified model of expanding populations in one dimension (Peischl et al. 2013). We focus on the dynamics of wave front and ignore gene flow between interior demes. This assumption makes mathematical analysis feasible and (as we shall see) yields a good approximation for the model simulated above (Peischl et al. 2013).

Individuals are diploid and monoecious. Generations are discrete and nonoverlapping, and mating within each deme is random. As before, mutations have symmetric effects $\pm s$ and enter the population at rate u_b and u_d, respectively. Fitness effects are multiplicative; that is, there is no dominance or epistasis. We assume that new mutations are uniformly distributed over n loci. We assume that these loci are in linkage equilibrium, as is approximately the case if recombination is strong. We scale relative fitness with respect to the ancestral population such that $\bar{w}_f = 1$ at the onset of the expansion.

Demes are arranged along a one-dimensional uniform habitat. Let $d_f(t)$ denote the deme on the wave front at generation t. If $d_f(t)$ is at carrying capacity, F founder individuals move from deme $d_f(t)$ to deme $d_f(t) + 1$. Migration between other demes is ignored. The newly colonized deme grows geometrically at rate R until it reaches its carrying capacity K. We denote the time it takes to reach carrying capacity by T. Selection acts through differential viability only during this growth phase. As before, we set $K = K_0\bar{w}$ and $R = R_0\bar{w}$ in each deme, where K_0 and R_0 are constant across demes. Furthermore, we assume that F is proportional to K such that we can write $F = F_0\bar{w}_f$ where F_0 is the number of founders if $\bar{w}_f = 1$. The time T to reach carrying capacity is then independent of K; therefore, mutation load slows down the expansion independently of its effect on carrying capacities. We note that our model can be readily applied to other models of hard selection or population growth, for instance, when mean

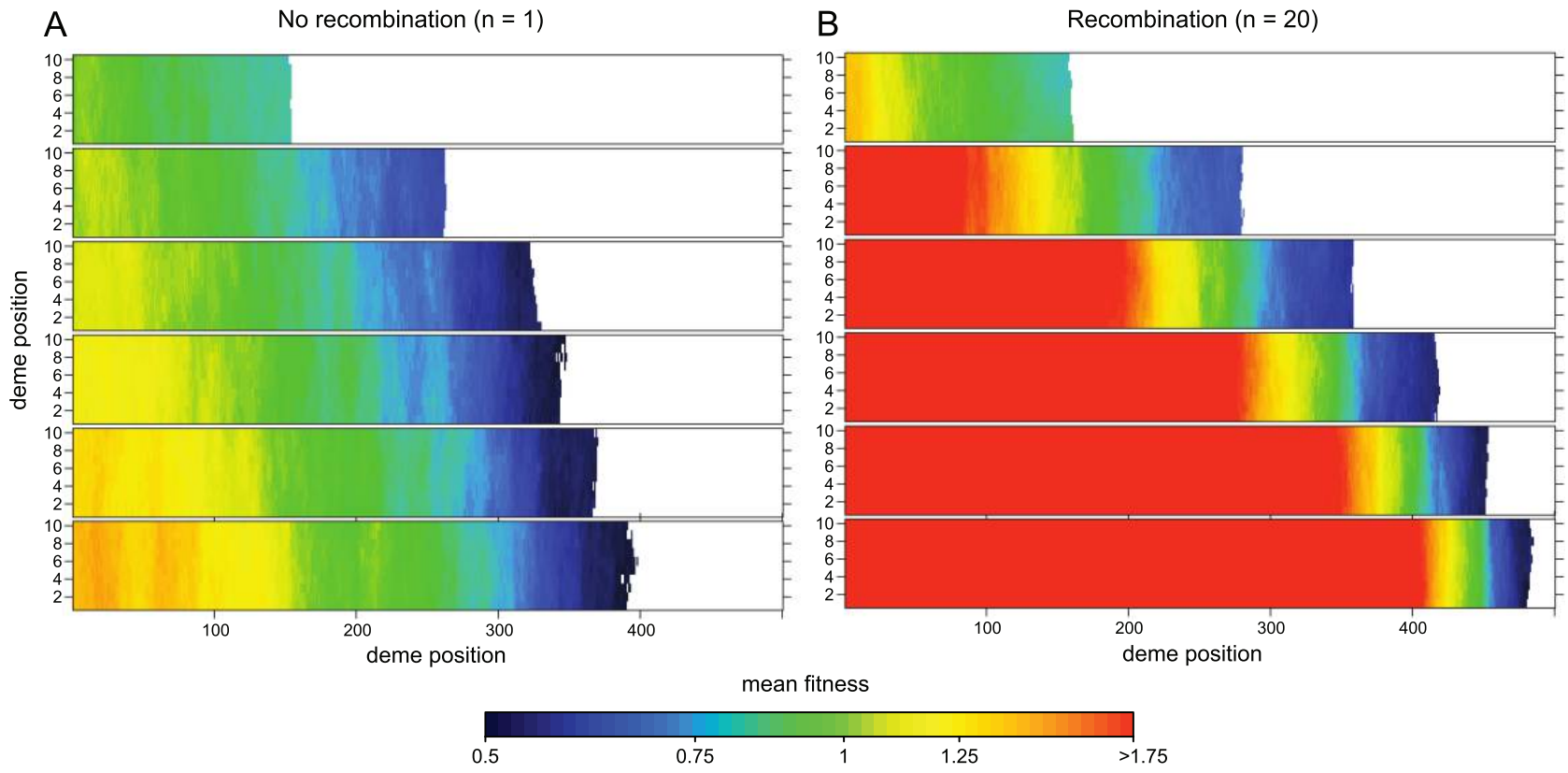

Figure 4: Evolution of mean fitness during a range expansion in a two-dimensional habitat. *A*, Nonrecombining species. *B*, Species with 20 recombining regions. Snapshots are shown at $t =$ 800, 1,600, 2,400, 3,200, 4,000, and 4,800 generations after the onset of the expansion (from top to bottom). Parameter values are as in figure 1.

fitness affects growth rates or carrying capacities but not both (see app. F).

In this model, the wave front is simply the deme at the edge of the species range, and we can study the dynamics of the wave front by simply tracking the dynamics of allele frequencies in this deme $d_f(t)$. The resulting model is similar in spirit to a recent model of serial founder effects (Slatkin and Excoffier 2012) and to models of repeated bottlenecks in single panmictic populations (Wahl and Gerrish 2001). The dynamics of our model is illustrated in figure 5.

Probability of Fixation. Peischl et al. (2013) derived the probability of fixation of new mutations on the expanding wave front under this model but assuming soft selection (and using a different parameterization). Note that a mutation may be fixed on the front but at intermediate frequencies or absent elsewhere. Consider a mutation that first occurs as a single copy in deme $d_f(t)$, and let τ denote the age (in generations) of this deme. In appendix A, we show that the probability of fixation of this mutation under hard selection is approximately

$$p(s,\tau) \approx \frac{\exp\left[-4FTsx_0(\tau)\right]-1}{\exp\left(-4FTs\right)-1}, \tag{2}$$

where $x_0(\tau)$ is the expected frequency of the mutation when the deme $d_f(t)$ reaches carrying capacity. Note that the probability of fixation depends on factors such as population size (via F) and growth rates (via T), which in turn depend on the population mean fitness. If mean fitness increases on the wave front, both R and K also increase. Higher growth rates decrease the efficiency of selection on the front because the time T during which selection occurs decreases. On the other hand, larger carrying capacities increase the efficiency of selection on the front. A detailed derivation of equation (2) can be found in appendix A.

Evolution of the Mean Fitness on the Wave Front. We make here the simplifying assumption that mutations are

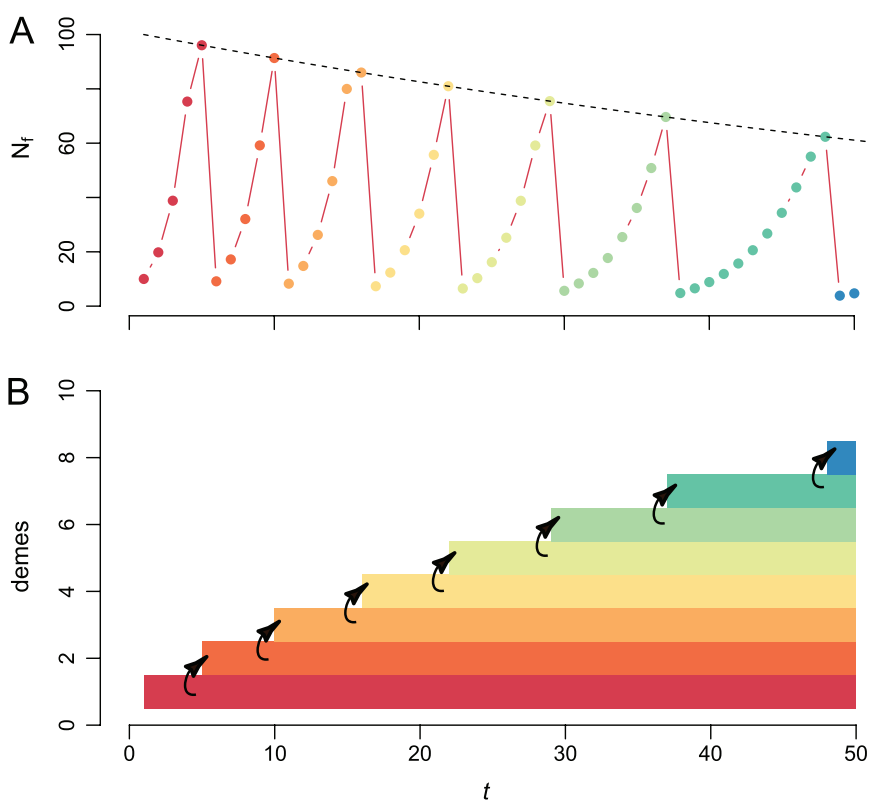

Figure 5: Sketch of the demographic dynamics of the analytical model. *A*, Circles show the population size on the wave front, N_f, as a function of time. Each color corresponds to a particular deme in *B*. When the deme on the front reaches carrying capacity, a new deme is colonized, and we follow its density. The dashed line shows the carrying capacity on the front, which is proportional to mean fitness. Note that the time T it takes to colonize demes increases with decreasing mean fitness. *B*, Sketch of the colonization process. Empty demes are white, and colonized demes are different colors. Arrows indicate colonization events.

either fixed or lost within a single generation after they appear with probability $p(s, \tau)$ and $1 - p(s, \tau)$, respectively. We can then approximate the mean fitness in the leading deme at time t by

$$\bar{w}_f(t) \approx \prod_{i=1}^{t} [1 + \sigma(t)]^n, \tag{3}$$

where $\sigma(t)$ is the expected change in relative mean fitness caused by mutations that establish at a locus in generation t (for details on the derivation, see app. B). For the sake of simplicity, the dependence on τ is omitted in equation (3) (see also eq. [C2]).

Equation (3) shows that the mean fitness increases if $\sigma > 0$ and decreases if $\sigma < 0$. Under soft selection, σ is constant for a given set of parameters, and the mean fitness on the wave front either increases or decreases geometrically (Peischl et al. 2013). Contrastingly, in our model of hard selection, σ depends on $\bar{w}_f$ (see app. B), which may change over time. Increasing mean fitness leads to higher growth rates, which decrease the efficiency of selection on the wave front and hence also σ. On the other hand, mean fitness increases carrying capacities, which increases both the efficiency of selection on the front and the influx of mutations and, consequently, also σ.

Dynamics of Mean Fitness on the Wave Front. Unfortunately, equation (3) is too complex to obtain an explicit analytical approximation for $\bar{w}_f(t)$, but we can use it to predict the evolution of mean fitness numerically. Note that there is no migration rate in our analytical model. To compare the analytical results with stochastic simulations, we set the number of founders to $F = Km/2$, where m is the migration rate used in the simulations (see also Peischl et al. 2013).

Figure 6 compares our numerical solution resulting from equation (3) to results from simulations done under the more complex model, where demes grow logistically and gene flow can occur every generation between all occupied demes. The expected mean fitness on the wave front decreases gradually and approaches an equilibrium value at which the establishment of new deleterious and beneficial mutations occur at the same rate (fig. 7). Theory and simulations are in good agreement unless m is large (e.g., when $m = 0.2$; fig. 6*E*, 6*F*). This makes sense since our analytical model ignores gene flow between core and front demes. Our approximation is conservative since it underestimates expansion load if m is large (e.g., when $m = 0.2$; fig. 6*E*, 6*F*). One might expect that large migration rates would reduce isolation between the front and the core, which would make selection more efficient. However, high rates of migration also flatten the profile of the expanding wave front (Fisher 1937), which decreases the effective population size at the front and thus increases the strength of drift.

We can better understand the dynamics of the expansion load by considering separately beneficial and deleterious mutations. Figure 7*A* shows the evolution of the average number of deleterious and beneficial mutations carried by individuals living at the front of the expansion. Initially, deleterious mutations accumulate at a higher rate than beneficial mutations, resulting in a decrease of the mean fitness. Because the expansion slows down over time, selection becomes more efficient on the wave front (Hallatschek and Nelson 2008), and after some time, an equilibrium is reached, and deleterious mutations are established at the same rate as beneficial mutations (fig. 7*B*). Mean fitness then remains (on average) constant over time (cf. fig. 6).

Conditions for Expansion Load. Peischl et al. (2013) derived a simple condition for the occurrence of expansion load in terms of the fraction of mutations that are deleterious. We can readily generalize this condition to our model of hard selection (for details, see app. B). We find that at time t, the wave front mean fitness will decrease if the fraction of mutations that are deleterious exceeds a threshold:

$$\varphi_d > \frac{1}{\exp(-4FTs) + 1}. \tag{4}$$

If $4FTs \ll 1$, it further simplifies to

$$\varphi_d > \frac{1}{2} + FTs.$$

In plain words, adaptation at the front of range expansions is possible only if the population expands very slowly, if founder sizes are large, or if beneficial mutations have very large effects and/or occur sufficiently frequent relative to deleterious mutations. Evaluating equation (4) for the parameters used in figure 1 yields that mean fitness should decrease at the front of the expansion if $\varphi_d > 0.57$. This is in very good agreement with our simulations, in which we determine the critical value at $\varphi_d \approx 0.55$ (fig. 8).

Equilibrium Mean Fitness. We next assume that equation (4) is satisfied at the onset of the expansion, and so expansion load builds up. Assuming weak selection, such that we can ignore second- and higher-order terms in s, a simple approximation for the equilibrium mean fitness at the wave front is

$$\tilde{\bar{w}} \approx \frac{1}{R_0}\left\{1 + 2\frac{F_0}{R_0}\frac{\log(K_0/F_0)}{\log[\varphi_d/(1-\varphi_d)]}s + O(s^2)\right\} \tag{5}$$

(app. C; see also app. F for the equilibrium mean fitness in other models of hard selection). Note that $1/R_0$ is the mean

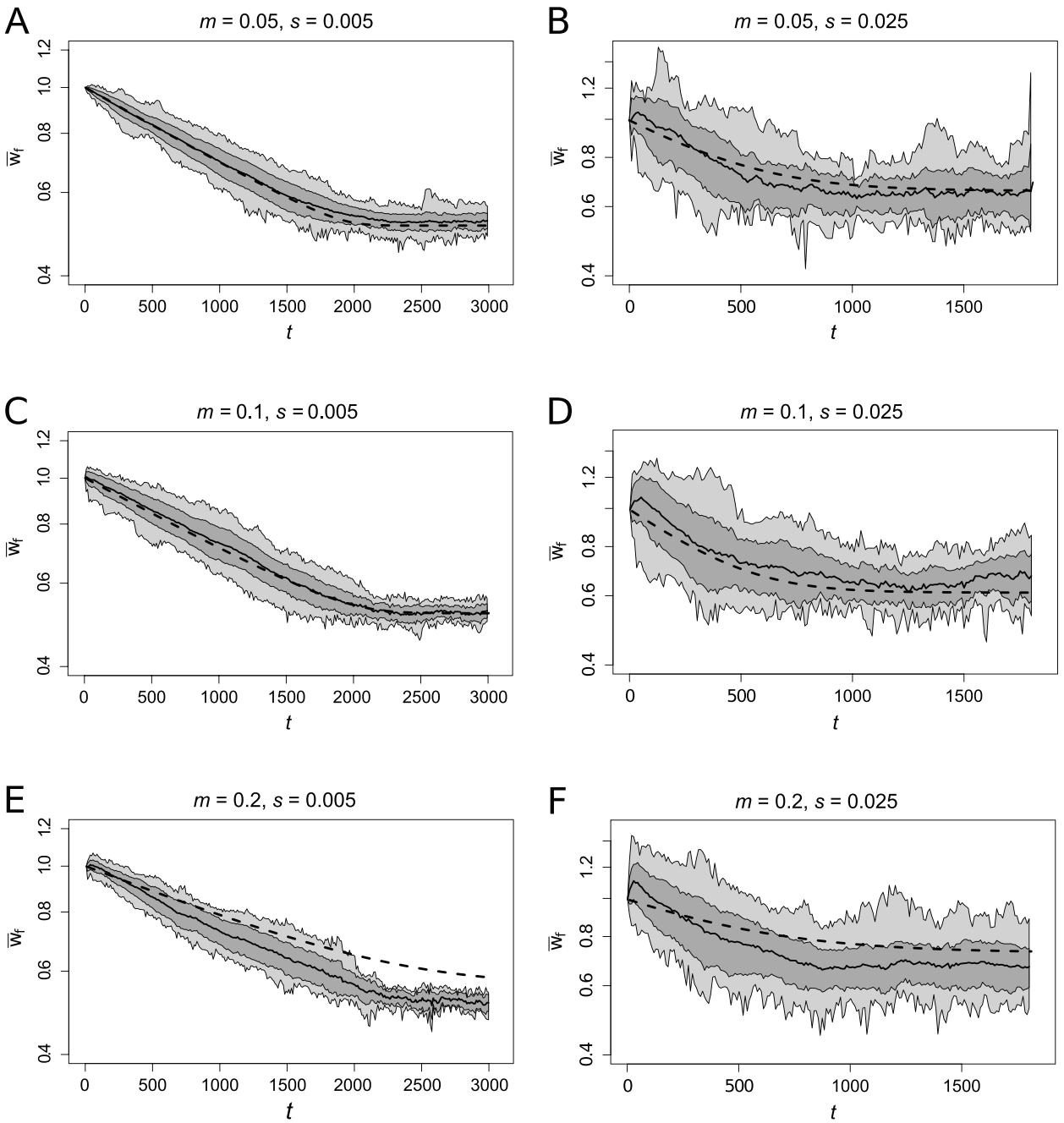

Figure 6: Evolution of the mean fitness on the wave front of an expanding population. The solid line shows the mean over 50 simulations. The shaded areas show 1 SD (dark gray) and the minimum and maximum of all observed values (light gray). The dashed line shows the analytical prediction from equation (3). Parameter values: $K_0 = 100$, $R_0 = 2$, $u = 0.05$, $n = 20$, $K_{\max} = 200$ (in simulations). The analytical results were evaluated using $F = Km/2$ and $T = \log(K/F)/\log(R)$.

(relative) fitness threshold above which the population growth rate remains positive. If the mean fitness on the front of the expansion falls below $1/R_0$, the expansion stops until migrants from the core bring new beneficial variants and increase the fitness, allowing for a new round of expansion (cf. fig. 1*A*). Thus, if mutational effects are small, the equilibrium mean fitness at the front is close to—but larger than—the critical threshold for extinction (fig. 6). Recall that we assumed that mutations evolve independently to avoid the mathematical complications that arise

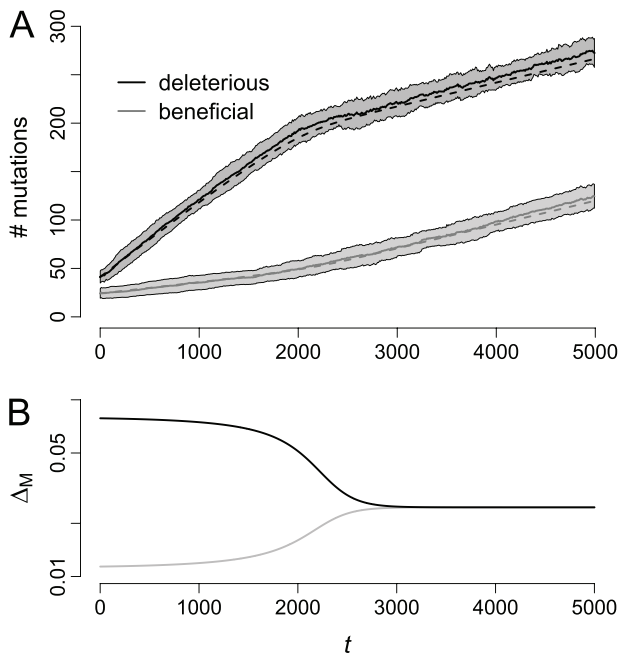

Figure 7: *A*, Evolution of the number of beneficial and deleterious mutations on the wave front. The solid line shows the mean over 50 simulations. The shaded areas show 1 SD. The dashed line shows the analytical prediction from equation (3). *B*, Analytical prediction for the rate of change of the number of deleterious (black) and beneficial (gray) mutations, denoted Δ_M. Parameter values: $s = 0.005$, $m = 0.05$, $K_0 = 100$, $R_0 = 2$, $u = 0.05$, $n = 20$, $K_{max} = 200$ (in simulations).

from clonal interference. This is a plausible approximation if recombination is strong.

The equilibrium mean fitness also determines the equilibrium expansion speed, which is given by approximately $[2m\log(R_0\tilde{\bar{w}})]^{1/2}$ (Skellam 1951). Because $\log(R_0\tilde{\bar{w}}) = O(s)$ as $s \to 0$, it implies that for small s values, the expansion speed at equilibrium is on the same order as the rate $(2ms)^{1/2}$ at which beneficial mutations spread through the core (Fisher 1937; Skellam 1951). For instance, for the parameter values used in figure 1, the equilibrium speed of the expansion is about twice the rate at which beneficial mutations spread through the core.

The simple form of equation (5) allows us to gain some insight into the dependence of $\tilde{\bar{w}}$ on the parameters of the model (for details, see app. C). The equilibrium mean fitness at the wave front decreases with R_0 and φ_d, and increases with K_0, F_0, and s. This effect of R_0 shows that a mutation that increases growth rate will be counteracted by a subsequent increase in mutation load. If the number of founders increases with increasing migration rate m, it follows that $\bar{w}_f$ also increases with increasing m. Consequently, mutations that increase dispersal rates or distances mitigate the effects of expansion load. Thus, an interesting prediction of our model of hard selection is that dispersal rates could evolve positively and growth rates negatively during range expansions.

Discussion

Studies of the geographical distributions of species have received constant interest since the early days of evolutionary biology (e.g., Darwin 1859; MacArthur 1972; Sexton et al. 2009). Nevertheless, the ecological and evolutionary factors that determine the limits of species ranges remain poorly understood (Gaston 2009). In some cases, sharp transitions in the environment impose clear limits to a species range, but for many species, the habitat at the range edge does not differ greatly from the habitat in the range core (Gaston 2009). This raises questions about which factors other than changes in the environment influence the dynamics of species ranges.

In this study, we investigated the effect of expansion load—that is, the mutation load that accumulates at the front of expanding populations (Peischl et al. 2013)—on the evolutionary dynamics of species ranges. We considered the case where population growth depends on the mean fitness; that is, selection is hard (sensu Wallace 1975). We found that deleterious mutations readily accumulate at the front of expanding populations, which slows the expansion down (fig. 1). This outcome differs qualitatively from the case of soft selection (Peischl et al. 2013). Moreover, the slowing down of the expansion makes selection on the wave front more efficient (see eq. [2]), and the rate at which mutation load builds up also slows down (fig. 6). Depending on the amount of recombination, this results in an expansion that approaches an equilibrium speed (fig. 1*B*; eq. [5]) or proceeds in pulsed expansions and extinctions (fig. 1*A*).

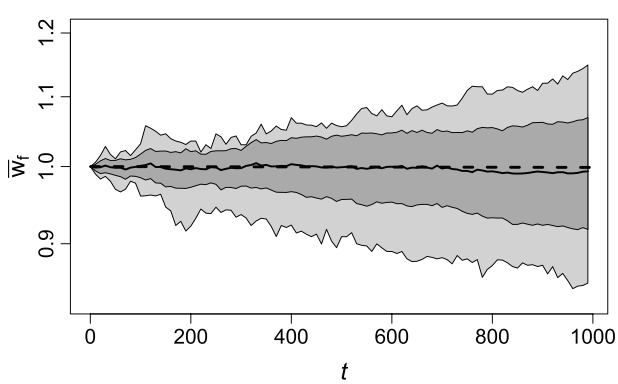

Figure 8: Evolution of mean fitness on the front of an expanding population. Parameter values are as in figure 1*B*, except that $\varphi_d = 0.55$. Color code is as in figure 6. The thin black line indicates $\bar{w}_f = 1$.

Which outcome occurs depends critically on the amount of recombination. Strong recombination increases the rate of adaptation in the wake of the expansion wave (fig. 1; see also Felsenstein 1974), but it reduces the rate at which beneficial mutations invade the wave front (fig. 3). Without recombination, the rate of adaptation in the core is relatively slow, but we observed repeated invasions of the wave front by high-fitness lineages from the core (fig. 3). In the absence of recombination, the rate of adaptation in the core is relatively slow, but we observed repeated invasions of the wave front by high-fitness lineages from the core (see figs. 1*A*, 2). If selection is strong, the invasion of the front by high-fitness individuals can temporarily restore the fitness on the wave front (fig. 2). This may have interesting consequences for the evolution of sex and recombination during and after range expansions. For instance, in organisms that can reproduce both sexually and asexually, we would predict higher rates of clonal reproduction in marginal populations. In obligatory sexual organisms, recombination modifiers such as inversions could facilitate the spread of beneficial variants from the core of the species range into marginal populations or new habitats.

In this article, we study models in which a population's mean fitness affects both its growth rate and its carrying capacity. Our main results extend to models in which fitness affects either growth rates (figs. F1–F3) or carrying capacities (figs. F4–F6) but not both. If mean fitness affects only growth rates, the expansion load builds up more slowly, and mean fitness at equilibrium is higher (fig. F1). That is because larger population sizes at the wave front make selection more efficient. If mean fitness affects only carrying capacities but not growth rates, the dynamics of mean fitness are similar to the case of soft selection (figs. F4–F6; see also Peischl et al. 2013) because the expansion dynamics are mainly determined by population growth rates (Skellam 1951). In contrast to soft selection, however, mutation load can drive populations at the leading edge to extinction (fig. F5; see also Lynch et al. 1995).

Interestingly, expansion load in asexual organisms can decrease the population size of demes in the wake of the wave to the point that deleterious mutations accumulate by a process known as Muller's ratchet (Haigh 1978). Populations can therefore collapse hundreds or thousands of generations after they have been at the wave front (fig. F5*A*).

Genetic differentiation between populations in our model is determined by gene flow between demes and founder effects during colonization. F_{ST} between front and core demes for neutral genes reached high values during the expansion (>30%; fig. E4). After the expansion, F_{ST} then quickly leveled off to values between 0.1 and 0.2 for demes located 100 demes apart. These final F_{ST} values are in line with those commonly observed in many organisms.

Migration rates were assumed constant across populations and did not evolve in our analysis. We showed that increasing migration rates or distances would not only accelerate the expansion but also mitigate the negative effects of expansion load (see fig. 6 or eq. [5]). Our results therefore suggest that selection against deleterious mutations could drive the evolution of higher dispersal during range expansions. Note that this is different from the process of spatial sorting (Shine et al. 2011), which does not invoke selection. Higher growth rates could also accelerate the expansion (Fisher 1937; Skellam 1951) but our results show that this will be counteracted by relaxed selection on the wave front and an accumulation of deleterious mutations (see eq. [5]). We thus predict that dispersal rates could evolve positively and traits linked to growth rates negatively during range expansions.

These predictions are met in the famous invasive cane toads in Australia. They evolved increased dispersal during their expansion (Phillips et al. 2010), and fast-dispersing toads from the invasion front may suffer higher mortality (Phillips et al. 2008; but see Phillips 2009). They often develop spinal arthritis (Brown et al. 2007), which could be due to some genetic defects.

Most of our results are based on a very simple distribution of fitness effects (DFE) with just two categories of mutations (beneficial and deleterious) and symmetric fitness effects. As shown in appendix D, our results extend to more complicated distributions of fitness effects (see fig. E3). A key requirement for expansion load to happen is that deleterious mutations occur more frequently than beneficial ones. Under our analytical model, equation (4) provides the exact conditions for the occurrence of expansion load in terms of the fraction of deleterious mutations among nonneutral mutations. Roughly speaking, if mutations have small effects, expansion load occurs if the fraction of deleterious mutations is larger than $1/2 + FTs$, where F is the size of founder populations, T is the number of generations between founder events, and s is the strength of selection.

Throughout this study, we focused on DFEs where the fraction of deleterious mutations remains constant, but it would be interesting to extend our results to models of stabilizing selection (e.g., Fisher 1930), where the DFE depends on the genetic composition of the population and hence changes over time. Our analytical results suggest that expansion load should be less severe in such cases, because the fraction of mutations with detrimental effects decreases if the population moves away from the optimal position in the fitness landscape (Martin and Lenormand 2006). On the basis of equations (4) and (5), we also expect expansion load to be less severe if the fraction of beneficial mutations stays constant but large-effect mutations become more common over time.

Changes in the DFE could also result from spatial or temporal variation in the environment (Bank et al. 2014). Our results could be certainly extended to temporarily changing environments by considering appropriately weighted averages of selective pressures (e.g., Peischl and Kirkpatrick 2012), and we believe that our results should remain qualitatively valid in this scenario. It seems likely that expansion load and the depletion of functional genetic variation during range expansions (Pujol and Pannell 2008) could prevent adaptation to novel environments that are encountered during an expansion. However, it has also been shown that the ability to explore complex fitness landscapes is increased during range expansions (Burton and Travis 2008), which could prove to be useful during invasion of new habitats. If the environment changes in both space and time, species can either adapt or shift their range to escape extinction (Davis and Shaw 2001). Previous studies highlighted the importance of range margins as a source of de novo mutations to adapt to environments that change in space and time (e.g., Kirkpatrick and Peischl 2013). These results are, however, based on models that ignore the stochastic effects at shifting range margins. Our results suggest that, like range expansions, range shifts should also have intrinsic costs in terms of an increased mutation burden, which may affect the role of range margins during adaptation to novel environmental conditions.

Our results show that intrinsic factors can limit a species range even in the absence of environmental variation. This complements classical explanations about species range limits due to the exhaustion of additive variance preventing further adaptation (e.g., Pujol and Pannell 2008) or to a flow of maladapted alleles from the range core (Kirkpatrick and Barton 1997). The view that species limits can be driven by intrinsic factors is supported by a recent review of transplant experiments showing that even though performance declined in about 75% of all considered cases, about 26% of studies showed self-sustained transplants from the range core beyond range limits (Hargreaves et al. 2014). This pattern is even more striking when focusing on geographic range limits (in contrast to altitudinal range limits), where transplants beyond the range limits showed positive growth rates in about 83% of all considered cases. Unfortunately, most of the experiments focused solely on individuals from the range core, and the comparison between the performances of transplants from different parts of the species range would yield interesting insights and could potentially provide support for the conclusions from our models.

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