Limits to adaptation along environmental gradients
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Abstract

Why do species not adapt to an ever-wider diversity of conditions, gradually expanding their geographic range? Haldane proposed that in a spatially heterogeneous environment, a species may be unable to adapt and expand its range because gene flow from the centre swamps populations at the margins, preventing their adaptation. Yet, current deterministic theory states that such a sharp limit to a species’ range only forms when genetic variance is fixed: when it can evolve, there is no such limit. We show that genetic drift can generate a sharp margin to a species’ range, by reducing genetic variance below the level needed for adaptation to spatially variable conditions. Dimensional arguments and separation of ecological and evolutionary time scales reveal a simple threshold that predicts when adaptation at the range margin fails. Two observable parameters describe this threshold: i) the effective environmental gradient, which can be measured by the loss of fitness due to dispersal to a different environment, and ii) the efficacy of selection relative to genetic drift. The key parameters were derived based on dimensional arguments and separation of ecological and evolutionary time-scales; the prediction is confirmed by simulations. Our theory predicts when a sharp range margins may form even in the absence of abrupt changes in the environment. Furthermore, it implies that gradual worsening of conditions across a species’ habitat may suddenly lead to range fragmentation – as adaptation to a wide span of conditions within a single species becomes impossible. This theory, which explains when genetic drift leads to a collapse of adaptation, has implications for conservation, response to climate change and management of biological invasions.

Author Summary

Why do species not adapt to an ever wider range of conditions, gradually expanding their geographic range? Haldane proposed that when the environment varies across space, a sharp limit to a species’ range forms due to “swamping” by gene flow which creates a positive feedback between low population size and maladaptation. Yet, current deterministic theory states that such a sharp limit to species’ range only forms in the presence of genetic constraints, such as fixed genetic variance. We show that there is an intrinsic limit to adaptation – and hence a sharp limit to a species’ range, which arises even when genetic variance can evolve. Just two observable parameters describe when adaptation fails: i) the effective environmental gradient, which can be measured by the loss of fitness due to dispersal to a different environment and ii) the efficacy of selection relative to genetic drift. The theory predicts sharp range margins even in the absence of abrupt changes in the environment. Furthermore, it implies that gradual worsening of conditions across a species’ habitat may suddenly lead to range fragmentation – as adaptation to a wide span of conditions within a single species becomes impossible.
Introduction

Why a species’ range sometimes ends abruptly, even when the environment changes smoothly across space, has interested ecologists and evolutionary biologists for many decades [1–6]. Haldane [2] proposed that when the environment is spatially heterogeneous, a species may be unable to adapt and expand its range because gene flow from the centre swamps the populations at the range margins, preventing their adaptation. Theory [7] showed that when genetic variance is fixed, adaptation indeed fails if the environment changes too steeply across space: the population remains well adapted only in the centre of the range, and gene flow swamps variants adapted to the margins, preventing range expansion. This limit to adaptation is dependent on local levels of genetic variance remaining unchanged.

Current theory states that when genetic variance can evolve, there is no sharp limit to a species’ range [8]. Genetic variance is generated by gene flow across a phenotypic gradient, so that adaptation to environmental differences is feasible until the fitness cost of maladapted variants becomes too high (i.e., when mean Malthusian fitness (growth rate) declines below zero). However, experimental evidence suggests that low genetic variance coupled with high gene flow [9,10] can prevent adaptation at the edge of a species’ range [11–13]. Thus, there is clear evidence that low genetic variation may limit adaptive range expansion. This may be because genetic drift reduces local variance [14,15] and hence the potential of the population to adapt [16]. Yet, studies of range expansion with genetic drift are few, and limited to simulations [17,18]. Here we ask if a sharp and stable margin to a species’ range arises due to the effect of genetic drift on the genetic variance.

Methods

We predict that the limit to a species’ range arises due to influences of genetic drift, when range expansion requires adaptation in a phenotypic trait. Our model assumes that the environment varies steadily across space, and that there is a fitness cost associated with deviation from the spatially changing optimal value of the phenotypic trait. To describe the system, we model jointly the evolution of i) population size and ii) trait mean and its variance via change in allele frequencies. Crucially, we include both genetic and demographic stochasticity. The model is first outlined at a population level, in terms of coupled stochastic differential equations (Text S1). Using this formalisation, we can reduce the number of effective parameters to four dimensionless parameters. Further, if we separate time-scales of ecology (fast) from evolution (slow), we can then predict the key driving parameters.

We test the predictive power of the derived key parameters using individual based simulations. Simulated individuals are distributed among demes that form a one-dimensional habitat, with the phenotypic optimum varying along the habitat, and experience a life cycle consisting of selection, mutation, recombination, and then dispersal. Every generation, each individual mates with a partner drawn from the same deme, with probability proportional to its fitness, to produce a number of offspring drawn from a Poisson distribution with mean of $\text{Exp}[r]$, where $r$ is the individual’s Malthusian fitness in continuous time. Individual fitness declines due to deviation of
the phenotypic trait \((z)\) from the optimum and due to crowding (the fitness is density-dependent). The trait is determined by a number of additive di-allelic loci, which permits genetic variation to evolve. All parameters are described in Table 1; for full description of the model see Text S1.

An illustration of a simulation with weak genetic drift \((N\sigma\sqrt{s} \gg 1, \text{see [19, 20]})\) is given in Fig. 1, where population size within a dispersal distance \(\sigma\) is given by \(N\sigma\), and \(s\) is the strength of selection per locus. \(N\) represents the local population size within each deme, which corresponds to population density in continuous space. With weak genetic drift, genetic variance \(V_G\) is generated primarily by gene flow due to mixing of genes from individuals with different phenotypes, well adapted to the diverse environments.

**Results**

**Scaling and separation of time scales**

We now proceed by reducing the number of parameters in the model (all parameters are defined in Table 1). First, we define the strength of density dependence by the rate at which deviations from the equilibrium population density \(\hat{N}\) are corrected – i.e. \(r^* \equiv -\frac{\partial}{\partial N} \frac{\partial N}{\partial t} \mid_{N=\hat{N}} [7]; \hat{N}\) assumes population is well adapted in its trait mean. We can then rescale space, time, trait, and population density (see Text S1 for more details) to obtain four dimensionless parameters that fully describe the system: neglecting mutation and assuming linkage equilibrium between loci. Linkage equilibrium (i.e., neglecting covariance between allele frequencies) is a good approximation for this model: whereas migration across the habitat generates positive linkage disequilibrium between loci, stabilising selection drives negative disequilibrium, and these cancel unless selection per locus is strong (see [21] and Text S2).

Two dimensionless parameters carry over from the phenotypic model of [7]: i) the load due to genetic variance around the optimum, scaled by \(r^*\), \(A = V_G/(r^*V_S)\); and, ii) the effective environmental gradient \(B = \frac{b_0}{r^*\sqrt{2V_S}}\). Two additional parameters come from including stochasticity: iii) the efficacy of population regulation relative to demographic stochasticity \(N\sigma\sqrt{r^*}\); and, iv) the efficacy of selection relative to the strength of genetic drift \(N\sigma\sqrt{s}\). Note that the dimensionless parameters describe a full set that defines the system, rather then all being fixed quantities: \(A\) evolves over long time-scales; whereas population density \(N\) evolves fast to its equilibrium value \(\hat{N}\).

Selection per locus \(s\) is typically much smaller than the rate of return to equilibrium population density \(r^*\) (see [22] and [23, Appendix D]). This has two important consequences. First, the ecological dynamics \(\partial N/\partial T\) are much faster than the evolutionary dynamics \(\partial p/\partial T\) of the individual loci. Second, the effect of fluctuations due to genetic drift (scaling with \(1/(N\sigma\sqrt{s})\)) is expected to dominate over the effect of demographic fluctuations, that rise with rise with \(1/(N\sigma\sqrt{r^*})\): see Text S1 and Fig. S1. Genetic drift slowly degrades adaptation at each locus (Fig. S2), reducing the variance, which then leads to a fast collapse of adaptation and local population size \(N\): the species’ range contracts from the margins or disintegrates.
Threshold for collapse of adaptation

We simulated the basic model with a linear gradient, assuming equal allelic effects $\alpha$ (equal phenotypic change due to substitution at every locus). Parameters were drawn at random from distributions consistent with our knowledge of the range of parameters expected in nature and ensuring that without genetic drift, all ranges would expand (see Fig. S3 and [23, Discussion]). This is denoted the “random set” of 1000 runs. We also performed additional runs to test if the threshold obtained from the linear gradient is robust to relaxing the model assumptions, namely that a stable range margin forms when i) the environmental gradient varies across space or ii) when the carrying capacity is non-uniform. Last, we tested stability of the threshold iii) when the allelic effects $\alpha_i$ are exponentially distributed.

Fig. 2 shows the results with a linear environmental gradient. The effective environmental gradient, $B$, and the efficacy of selection relative to the strength of genetic drift, $N\sigma\sqrt{\bar{s}}$, suffice to describe the threshold for the collapse of adaptation. This is because the genetic variance evolves primarily in response to $N\sigma\sqrt{\bar{s}}$ and $B$ and the effect of demographic stochasticity $N\sigma\sqrt{\bar{r}}$ is relatively weak. When $B \geq 0.15 N \sigma \sqrt{\bar{s}}$, genetic drift strongly degrades adaptation to a steeply changing environmental optimum and the species’ range contracts. The constant 0.15 is obtained as the best fitting threshold for the data in Fig. 2.

When the steepness of environmental gradient varies across space, this threshold, $B \sim 0.15 N \sigma \sqrt{\bar{s}}$, indicates where a stable range margin forms (Fig. 3). Without genetic drift, the genetic variance would steadily inflate, gradually reducing local population size due to increasing number of maladapted individuals (see dashed lines in Fig. 3D). With genetic drift, the variance is pushed below the level necessary to maintain adaptation, the trait mean abruptly fails to match the optimum and sharp margin to the range forms. Similarly, a sharp range margin forms when $B \geq 0.15 N \sigma \sqrt{\bar{s}}$, if carrying capacity declines across the habitat for extrinsic reasons (Fig. S4). When the allelic effects $\alpha_i$ are exponentially distributed, range expansion slows down progressively around the threshold (Figs. S5 and S6), described by $B^* = 0.15 N \sigma \sqrt{\bar{s}}$, where $\bar{\sigma} \equiv \frac{\sigma}{\pi}$ and $\bar{\sigma}$ is the mean allelic effect. For the population to expand further beyond the threshold, positively selected alleles with increasingly large effect need to arise (Fig. S7), yet these become ever rarer.

The threshold $B \sim 0.15 N \sigma \sqrt{\bar{s}}$ can be rephrased to a form that is closely related to Haldane’s cost of selection [24], which gives the number of selective deaths required to secure a substitution in a single locus. Considering uniform selection in a single population, it follows that adaptation fails when there are too few births to offset death due to selection. With spatially varying environments, adaptation fails when too many substitutions per unit distance are needed per (roughly speaking) birth in the population: $b/\alpha \geq 0.15 N \bar{r}^*$ (Fig. S8).

In the absence of genetic drift, low dispersal can enhance adaptation by reducing swamping by gene flow [8,9] – but with genetic drift, this is no longer true. To a first approximation, both the efficacy of selection relative to genetic drift, $N \sigma \sqrt{\bar{s}}$ and the effective environmental gradient $B = \frac{b\sigma}{\sqrt{2\pi}r^*}$ increase at same rate with dispersal. Only a weak dependence on dispersal remains...
via \( r^* \) (because \( \dot{N} = K \frac{r^*}{r_m} \)), which favours low to intermediate dispersal: see Fig. S1. This beneficial effect of intermediate dispersal [17] is much weaker than in one-locus models [25, 26], where gene flow only decreases fitness indirectly via density dependence.

In a purely deterministic phenotypic model with fixed genetic variance, there exists a “critical gradient”, above which the trait mean fails to track the spatially changing optimum, and the population is well adapted only in the centre of its range [7, p.6]. We show that when genetic variance evolves due to selection, dispersal and genetic drift, population characteristics will move towards the critical gradient \( B_c \sim \sqrt{2A} \) [23, Fig. 3] as the environmental gradient increases. The population will abruptly lose genetic variation and suffer demographic collapse when the critical gradient is crossed (Fig. 4).

**Discussion**

In summary, we have found a general threshold for a sharp range margin that forms when adaptation is maintained by many loci of small effect. This threshold exists in the absence of any genetic constraints, such as insufficient genetic variance [7, 27] or rigid fitness trade-offs between traits [28]. The threshold is set by the relative magnitude of the total fitness load due to dispersal versus the strength of genetic drift relative to the selection at each locus – hence, it readily generalises to many traits. In general, the loci underlying a particular trait will have a distribution of selection coefficients. We have demonstrated that a sharp range margin forms not only when all loci have equal allelic effects (i.e., the trait changes by a fixed value due to every substitution), but also when allelic effects are exponentially distributed. With exponentially distributed allelic effects, the mean selection coefficient gives a good estimate for the expected range margin, because clines at weakly selected loci are degraded by genetic drift (Fig. S7 and [20]), reducing the genetic variance, and positively selected alleles with large effects arise only rarely.

We explain when a sharp margin to a species’ range may form, even when there is no abrupt change in the environment. Furthermore, the theory implies that gradual worsening of conditions across a species’ habitat may suddenly lead to a range fragmentation, when adaptation to a wide span of conditions within a single species becomes impossible. This has implications for conservation [29], response to climate change [30] and management of biological invasions [31].

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References


Figure 1. Illustration of the individual-based model at the limit of weak genetic drift. Deterministic predictions [8] are shown by dashed lines. The species’ range keeps expanding over time (arrows) so that ultimately all available habitat would be filled. (A) Trait mean $\bar{z}$ matches the optimum trait value $\theta = b x$ - shown for the starting population (light blue) and after 5000 generations (dark blue). The spread of the trait values $z$ for all individuals is shown with dots. Further figures are at generation 5000. (B) Local population size is close to the predicted $\hat{N} = K r^s / r_m = K (1 - \frac{\sigma b}{2 \sqrt{V_S} r_m})$ at the centre (dashed line). (C) Clines for allele frequencies are shown by thin black lines, displaced to the side (for visibility) are two of the many predicted clines (dashed), with cline width $w_s = 4 \sigma / \sqrt{2S}$ and cline spacing $\alpha / b$. (D) Total genetic variance is shown in blue, the linkage-equilibrium component in black; the dashed line gives the prediction $V_G = V_{LE} = b \sigma \sqrt{V_S}$ (see also [8, p. 378]). There must be $b / \alpha$ clines per unit distance so that the trait mean matches the optimum and each cline (allele frequency across space) contributes genetic variance of $V_{G,1} = \alpha \sigma \sqrt{V_S}$. The contribution from mutation is negligible. Parameters (defined in Table 1): $b = 0.1$, $\sigma^2 = 1/2$, $V_S = 2$, $r_m = 1.025$, $K = 300$, $\mu = 10^{-6}$. ✫
Figure 2. Species’ range starts to contract when the effective environmental gradient is steep relative to the efficacy of selection relative to genetic drift: $B \gtrsim 0.15 N\sigma \sqrt{s}$, where $B = \frac{b\sigma}{r^* \sqrt{2V}}$. This threshold is shown by a dashed line (notice the log-log scale). The dots represent all simulation runs from the “random set” - the rate of expansion increases from light to dark blue and rate of range contraction increases from orange to red. Open dots indicate fragmented species’ ranges (Figure S10). Grey dots denote populations for which neither expansion nor collapse was significant (where no range change was included in 98% confidence interval). Predicted local population size given by the parameter $\hat{N} = Kr^*/r_m$ is used on the y-axis rather than the measured $N$; measuring $N$ does not change the threshold or its accuracy, assuming density is measured where population is well adapted. The ranges of the underlying (unscaled) parameters were in the following intervals: $b = [0.01, 1.99]$, $\sigma = [0.5, 4.8]$, $V_s = [0.006, 8.4]$, $K = [4, 185]$, $r_m = [0.27, 2]$ and $\alpha = [0.01, 0.39]$. This gives the selection coefficient per locus in the interval of $s = [3 \cdot 10^{-4}, 0.66]$, with median of 0.007. Mutation rate is small so its direct contribution to genetic variance is negligible, yet it is large enough to sustain expansion of a species’ range over the total time of 5000 generations: $\mu = [10^{-8}, 8 \cdot 10^{-5}]$ per locus and generation. The number of genes is between 7 and 3971. The parameter distributions are detailed in Fig. S3.
Figure 3. With a steepening environmental gradient, a stable range margin forms when $B \gtrsim 0.15N\sigma \sqrt{s}$ (red dots). (A) The gradient in trait mean follows the environmental optimum (dashed line) until the predicted threshold is hit (red dots) – then the population density starts to drop off rapidly. (B) As the environmental gradient steepens, local population density declines, and due to genetic drift, adaptation fails for a much shallower gradient than the deterministic prediction for evolving variance (dashed line). (C) Three representative clines are shown in black, other clines form the gray background. (D) Close to the threshold, genetic variance, especially its linkage equilibrium component $V_{LE}$, fails to increase fast enough to match the steepening environmental gradient, and adaptation fails. Formulae for the predicted dashed lines (B, D) are in Fig. 1. Parameters: gradient in the central habitat $b = 0.12$; $\sigma^2 = 1/2$, $V_S = 1/2$, $r_m = 1.06$, $K = 53$, $\mu = 2 \cdot 10^{-7}$. The trait and its mean is shown also for the initial population, all other subfigures depict the population after 100 000 generations, whilst it stops expanding after 40 000 generations.
Figure 4. Phenotypic model predicts a sharp transition in the dynamics. Beyond
the critical gradient \( B_c \sim \sqrt{2}A \) (dashed curve), genetic variance is too small to support
adaptation in the trait mean [7, 23], and the species’ range collapses abruptly. The columns of
dots show ten replicates for each sets of runs, with increasing slopes of the (linear)
environmental gradient \( b \). The deterministic prediction with evolvable variance [8], \( A = \sqrt{2}B \), is
given by the gray dotted line. As the effective environmental gradient \( B \equiv \frac{b \sigma^2}{r \sqrt{2V_s}} \) increases,
\( A \equiv \frac{V_G}{\sqrt{2}} \) increasingly deviates from the deterministic prediction due to the combined forces of
the genetic drift (see Figure S2) and the selection on small transient deviations of the trait mean
from the optimum (see Figure S9). When the threshold of \( B = 0.15N\sigma \sqrt{s} \) is reached (short
black line at about \( B \sim 0.4 \), assumes \( A = \sqrt{2}B \)), the range starts to contract (shown by dots
changing from blue to orange). With yet further increase in \( B \), the critical gradient \( B_c \) (dashed
curve), predicted for the phenotypic model [7, 23], is reached. The critical gradient \( B_c \) marks a
second transition in the range dynamics: the population collapses or fragments (open dots, see
Fig. S10) to a state with no or very little clinal variation maintained (\( A \sim 0 \)). Beyond the solid
line \( B_c = (2 + A)/\sqrt{2} \), the phenotypic model [7] predicts extinction, which is not observed for
the parameter range shown. Parameters: \( b \) increases from 0.025 to 1.25, \( \sigma^2 = 1/2 \), \( V_s = 1/2 \),
\( K = 50 \), \( \alpha = 1/10 \), \( r_m = 1.06 \), \( \mu \) decreases from \( 10^{-6} \) to \( 10^{-7} \) as the number of loci increases.
Time = 5000 generations. The scaled genetic variance \( A \) (averaged over central quarter of the
range) is not at equilibrium for collapsing populations. After the range collapses, the population
alternates between no and limited adaptation, where a small number of clines (often one)
establish transiently.
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<tr>
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<td>–</td>
<td>effective environmental gradient, $\sqrt{\text{dispersal load} / r^<em>}$: $B = b\sigma/(r^</em>\sqrt{2V_S})$</td>
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<td>$N\sigma\sqrt{r^*}$</td>
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**Table 1. Four scale-free parameters: $A$, $B$, $N\sigma\sqrt{s}$ and $N\sigma\sqrt{r^*}$ describe the model.**

Top section gives seven parameters of the model before rescaling, middle section gives important composite parameters. Denoting the dimensions, $T$ stands for time, $D$ for distance and $Z$ for trait. Note that with Poisson number of offspring, the effective population size $N_e$ (which measures rate of genetic drift / coalescence) is identical to the $N$ that regulates population growth due to crowding: hence both carrying capacity $K$ and population size $N$ have units of $T/D$. Mutation rate $\mu$ is set to be small, with minimal contribution to the dynamics, and hence is neglected in the rescaled parameterization (bottom); see Text S1 for more details.
Evolutionary and ecological dynamics: The continuous model

We model the joint evolution of allele frequencies and population size, including both genetic and demographic stochasticity. The fitness of an individual declines quadratically with the deviation of the trait $z$ from an optimum $\theta$ that changes linearly across space: $\theta = bx$, where $b$ is the gradient in the environment and $x$ is the distance in one dimension. The phenotypic trait $z$ is determined by many additive di-allelic loci, so that genetic variance can evolve. Population growth is given by the local mean (Malthusian) fitness $\tau$.

The Malthusian fitness of a phenotype $z$ is $r(z, N) = r_e(N) + r_g(z)$, where $r_e(N)$ is the growth rate of a perfectly adapted phenotype, and includes density dependence; $r_g(z) \leq 0$ is the reduction in growth rate due to deviation from the optimum. The ecological component of growth rate $r_e$ can take various forms: we assume that regulation is logistic and declines linearly with density $N$: $r_e = r_m(1 - N/K)$, where $r_m$ is the maximum per capita growth rate in the limit of the local population size $N \rightarrow 0$. The carrying capacity $K$ is assumed to be uniform across space. Stabilising selection on the optimum $\theta$ has strength $1/(2V_z)$. Hence, for any phenotype, the drop in fitness due to maladaptation is $r_g(z) = -\frac{1}{2} (z - \theta)^2$. The local mean fitness is then $\bar{r}_g(z) = -\frac{(z - \theta)^2}{2V_z} - V_P$, where $V_P = V_G + V_E$ is the phenotypic variance. The loss of fitness due to environmental variance $V_E$ can be included in $r_m = r_m - V_E/(2V_S)$. Hence, the $V_E$ parameter is redundant and we can set $V_G = V_P$. For any given additive genetic variance $V_G$ (assuming a Gaussian distribution of breeding values), the trait mean $\tau$ satisfies [32, Eq. 2]:

$$\frac{\partial \tau}{\partial t} = \frac{\sigma^2}{2} \frac{\partial^2 \tau}{\partial x^2} + \frac{\sigma^2}{2} \frac{\partial \log(N)}{\partial x} \frac{\partial \tau}{\partial x} + \frac{\partial V_G}{\partial \tau} + \zeta \tag{S1}$$

The first term gives the change in the trait mean due to migration with mean displacement of $\sigma$. The second term describes the effect of the asymmetric flow from areas of higher density. The third term gives the change due to selection. The last term $\zeta$ gives the fluctuations in the trait variance due to genetic drift, uniquely specified by their correlation $\langle \zeta(x,t)\zeta(x',t') \rangle = \delta(x-x') \frac{V^2}{8N} \delta(t-t')$, where $\delta$ is the Dirac delta; based on [19] and $V_G = \sum_i \alpha_i^2 p_i q_i$.

The trait mean is $\tau = \sum_i \alpha_i p_i$ for a haploid model, where $p_i$ is the $i$th allele frequency, $q_i = 1 - p_i$ and $\alpha_i$ is the allelic effect - the change of the trait $\tau$ as frequency of locus $i$ changes from 0 to 1. For both haploid and diploid models, all of the allele frequencies $p_i$ then change as:

$$\frac{\partial p_i}{\partial t} = \frac{\sigma^2}{2} \frac{\partial^2 p_i}{\partial x^2} + \frac{\sigma^2}{2} \frac{\partial \log(N)}{\partial x} \frac{\partial p_i}{\partial x} + \left( p_i q_i \frac{\partial \tau}{\partial p_i} - \mu (p_i - q_i) + \epsilon \right) \tag{S2}$$

The change of allele frequency due to a gradient in fitness and local heterozygosity is $p_i q_i \frac{\partial \tau}{\partial p_i} = s p_i q_i (p_i - q_i - 2\Delta_i)$, where selection at locus $i$ is $s_i = \frac{\sigma^2}{2V} \Delta_i = (\tau - bx)/\alpha_i$ (see (8), two-allele model, Appendix 3). Here the fourth term describes the change due to (symmetric) mutation at rate $\mu$. The last term $\epsilon$ describes genetic drift, $\epsilon = \sqrt{\frac{2N}{\alpha_i}} dW_s(x, t)$, where $dW_s(x, t)$ represents white noise in space and time, and has dimensions $1/\sqrt{\text{Time} \times \text{distance}}$. The covariance in the fluctuations of allele frequencies in a haploid population is $\langle \epsilon_i(x, t)\epsilon_i(x', t') \rangle = \delta(x-x') \frac{2\alpha_i}{2\alpha_i} \delta(t-t')$ [19].

Equation [S2] is only exact at linkage equilibrium: but we will see this is a good approximation for unlinked loci. Whereas migration across the habitat generates positive linkage disequilibrium between any pair of loci, stabilising selection drives negative disequilibrium, and these cancel unless selection per locus
is strong. The derivation, which generalizes an ingenious but little known argument by Felsenstein [22], is given in Text S2.

Population dynamics reflect diffusive migration, growth due to the mean Malthusian fitness $\bar{r}$, and stochastic fluctuations:

$$\frac{\partial N}{\partial t} = \frac{\sigma^2}{2} \frac{\partial^2 N}{\partial x^2} + \tau N + \xi$$

(S3)

The number of offspring follows a Poisson distribution with mean and variance of $N$. Fluctuations in population numbers are given by $\xi = \sqrt{N} dW_t(x,t)$. Covariance in population numbers introduced by demographic stochasticity [33, e.g.] is therefore $\langle \xi(x,t)\xi(x',t') \rangle = \delta(x-x') N \delta(t-t')$.

The parameters of the model are summarised in Table 1, followed by their rescaling that reduces the number of parameters to the minimal set: from six (without mutation) to four.

The equations above can be rescaled by taking time relative to the strength of density dependence $r^*$, distance relative to dispersal, trait relative to strength of stabilising selection and population density relative to equilibrium density with perfect adaptation $\bar{N} = Kr^*/r_m$; as in [7, 8].

$$T = r^* t, \quad X = x \sqrt{\frac{2r^*}{\sigma^2}}, \quad Z = \frac{z}{\sqrt{r^* v_s}}, \quad \bar{N} = N/\bar{N}$$

(S4)

The rescaled equations are:

$$\frac{\partial \bar{N}}{\partial T} = \frac{\partial^2 \bar{N}}{\partial X^2} + \bar{R} \bar{N} + \sqrt{\frac{\bar{N}}{\bar{N}v_s}} \frac{dW_t(x,T)}{\sqrt{r^*}}$$

and

$$\frac{\partial p_{i_1} \ldots p_{i_k}}{\partial T} = \frac{\partial^2 p_{i_1} \ldots p_{i_k}}{\partial X^2} + 2 \frac{\partial \log(\bar{N})}{\partial X} \frac{\partial p_{i_1} \ldots p_{i_k}}{\partial X} + \frac{s}{r^*} (p_{i_1} q_{i_1} - 2 \frac{Z - BX}{\alpha_i^*}) - \frac{\mu}{r^*} (p_{i_1} - q_{i_1}) + \sqrt{\frac{pq}{\bar{N}v_s}} \frac{dW_t(x,T)}{\sqrt{r^*}}$$

(S6)

Where the scaled allelic effect $\alpha_i^* = \alpha_i/(r^* v_s)$ gives $A = V_G/(r^* v_s)$ over all loci and $\bar{R} \equiv \bar{r} \equiv r^*$ as in [7, 8]. The effective environmental gradient is $B = \frac{B^* v_s}{r^* v_s}$ where $B^*$ is the strength of selection relative to density dependence is given by $s/r^*$ and $\mu/r^*$ is considered negligible. The last two parameters are $1/\sqrt{\bar{N}v_s}$ and $1/\sqrt{\bar{N}v_s}$: hence, $s/r^*$ can be omitted as redundant, as it arises as the ratio of the last two scale-free parameters.

The scaling of the fluctuations with $1/\sqrt{\bar{N}v_s}$ and $1/\sqrt{\bar{N}v_s}$ is based on dimensional arguments:

Because the dimension of $dW_t(x,t)$ is $1/\sqrt{\text{time} \cdot \text{distance}}$ and time is rescaled with $r^*$, scaled white noise $dW_t(x,T)$ needs to be multiplied by a factor that has units of $\sqrt{\text{time} / \text{distance}}$. It has been demonstrated previously that fluctuations in allele frequency grow with $\sim 1/\sqrt{\bar{N}v_s}$ [20, 21]: hence, the required factor is $\sqrt{\frac{1}{\bar{N}v_s}}$. Similarly, a good factor with the correct dimensions for $dW_t(x,T)$ is $\sqrt{\frac{r^*}{\sigma_m}}$, which gives the scaling of demographic fluctuations with $\sim 1/\sqrt{\bar{N}v_s}$.

Note that $A$ is commonly defined as $A = V_G/(r^* v_s)$, the extra factor of 2 in the definition of $A$ is dropped later in (7); the typographical error was first noted by [34].
Individual based simulation

The model is derived as a limit to continuous time, and so applies to a wide range of models that reduce to this limit. In this limit, the rate of spatial dispersal depends only on the variance of distance moved, and the effective population density (for both allele frequency and demographic fluctuations) depends only on the variance of offspring number.

**Life cycle:** Discrete-time individual based simulations are set to correspond to the model with continuous time and space. The life cycle is selection → mutation → recombination → migration.

**Dispersal:** The habitat is formed by a one-dimensional array of demes. With deme spacing $\delta x = 1$, the population size per deme corresponds to the population density. We assume diffusive migration with a Gaussian dispersal kernel. The tails of the dispersal kernel need to be truncated: we choose truncation at two standard deviations of the dispersal kernel throughout, and adjust the dispersal probabilities following [35, p. 1209] so that dispersal kernel sums to 1, and the variance of dispersal is adjusted correctly. For dispersal per generation at $\sigma = \sqrt{4/2}$, dispersal reduces to a nearest neighbour migration with a probability of migration left and right of $m = 1/4$.

**Selection:** Every generation, each individual produces a Poisson number of offspring with mean of the individual’s fitness $\text{Exp}(r)$; where $r = r_m(1 - N/K) - \frac{(z - \theta)^2}{2v_r}$, as defined earlier.

**Mutation:** Mutation rate is set to be small so that its contribution to genetic variance is negligible, but large enough to in principle enable expansion of a species range over the total time of 5000 generations. Specifically, it is set to one substitution per the whole population and generation. Genetic variance maintained in a population due to dispersal across environments can be substantially larger than genetic variance maintained by mutation-selection balance in uniform environments. In uniform environments, mutational variance $V_m = \sum \mu \alpha_i^2 = (\text{where } \alpha_i \text{ are the allelic effects, and } n_i \text{ is the number of loci}) \text{ is robustly estimated to be between about } 5 \cdot 10^{-5}V_E \text{ and } 5 \cdot 10^{-3}V_E$ [36]. Taking a heritability $h^2 = V_G/(V_G + V_E) = 1/3$ we get $V_m$ between $10^{-4}V_G$ and $10^{-2}V_G$. In our model, genetic variance is inflated due to dispersal across environments, and so $V_m/V_G$ must be smaller. The mutational variance $V_m$ is conserved in the simulation runs because the habitat is set to be 10 cline widths wide (without drift) and the number of genes is set so that the whole habitat would be filled if all genes evolved to the clinal form (at some part of the habitat). Taking the higher limit of $V_m = 10^{-2}V_G$, follows that $\mu$ should be smaller than about $4 \cdot 10^{-3}$. It turns out that the the effect of mutation cannot be fully included in the predictions, as the contribution of mutation-selection balance cannot be robustly separated from the clinal variation. A considerably higher genetic variance than $V_{G, \text{mut}} = 2\mu n_i V_s$ (up to the limit of $1/4\alpha^2 n_i$) can arise due inflation of the variance by mutation along existing clines. Therefore, we concentrate on a parameter range where the contribution of mutation to genetic variance is low, which is a biologically plausible range.

**Reproduction, recombination:** The mating partner is drawn from the same deme, with the probability proportional to its fitness. Selfing is allowed at no cost. The genome is haploid with unlinked loci (the probability of recombination between any two loci is $1/2$); the allelic effects $\alpha_i$ of the loci combine in an additive fashion.

**Simulation runs:** Evolution starts with a well adapted population at the centre of the habitat. The habitat is about 10 cline widths wide; the number of genes is chosen so that with all genes adapted, the population spans the whole habitat, and that there are enough genes to maintain the “optimal” variance $V_G = bV_s \sqrt{V_5}$ at the central part of the habitat. At the start of the simulation, half of the genes are adapted: their clines take the form and spacing as assumed for the deterministic model under linkage.
The population evolves for 5000 generations; in total, we recorded over a thousand runs where without genetic drift the local population density would be greater than 4 assuming uniform adaptation (such that trait mean matches the optimum). We a-priori eliminated very small local population sizes ($N < 4$) so that the population size within a generational dispersal is not excessively small. The parameters were first varied one at a time, and then we tested the threshold drawing the parameters from distributions consistent with our knowledge of the range expected in nature (see [24, Discussion]), and so that the range of the important scale-free parameters overlaps with the relevant range where range collapses. The later will be referred to as the “random” set, with 1000 runs.

The Mathematica code for the simulations, including the distributions used to draw the unscaled parameters for the “random” set is provided in File S1. Fig. S3 shows the realised distributions for both the unscaled parameters and compound parameters.

References: Text S1

Text S2

Clines are at linkage equilibrium:

Stabilising selection on a quantitative trait generates negative linkage disequilibrium, whereas dispersal generates positive linkage disequilibrium. Felsenstein’s [22] analysis of variance components showed that at equilibrium, the linkage disequilibrium generated by dispersal cancels out with the negative linkage disequilibrium generated by stabilising selection.

The argument extends to a quantitative trait underlain by many bi-allelic loci, here demonstrated for a haploid genome. The genetic variance is $V_G = \sum \alpha_i^2 (p_i(x)q_i(x)) + 2 \sum_{i \neq j} \alpha_i \alpha_j D_{ij}$. The increase of linkage disequilibrium at QLE [37] between dispersal with variance $\sigma^2$ and recombination $r_{ij}$ is given by $D_{ij,disp} = \frac{\sigma^2}{r_{ij}} \frac{dp_i}{dx} \frac{dp_j}{dx}$ [38]. With allele frequencies at equilibrium, the linkage disequilibrium generated by stabilising selection alone is $D_{ij,sel} = -r + \sqrt{r^2 + 4(1-r)\alpha_i \alpha_j / V_s} \sim -\alpha_i \alpha_j / (r_{ij} V_s) p_i q_i p_j q_j$ for $D_{ij}$ small.

In this first order approximation, the terms cancel for each pair of loci when the cline shape is the same as that under linkage equilibrium ([8, two-allele model]) – independently of the cline spacing across space: $D_{ij} = D_{ij,disp} + D_{ij,sel} = 0$. This is because $\frac{dp_i}{dx} = p_i q_i A / w_i$, and the cline width at linkage equilibrium is $w_i = \frac{4\sigma \alpha_i}{\sqrt{V_s}}$.

It may be that the cline width $w$ or the linkage disequilibrium is distorted by additional forces, and/or by strong selection. However, unless selection is strong, the first order approximation gives a simple prediction for the pairwise disequilibrium: $D_{ij} = \frac{\sigma^2}{r_{ij}} p_i q_i p_j q_j 16 / (w_i w_j) - \alpha_i \alpha_j / (r_{ij} V_s) p_i q_i p_j q_j$. Hence, negative LD should arise if the clines become wider than expected under linkage equilibrium, positive LD if clines get narrower – for example, due to genetic drift. Conversely, positive LD, generated for example by long-range dispersal, would drive steeper clines.

References: Text S2

Figure S1. Fluctuations in clines are substantially larger than demographic fluctuations.

(A) Fluctuations in clines $\langle F \rangle$ rise somewhat slower than predicted at $\langle F \rangle = 0.392/(N \sigma \sqrt{s})$ (dashed line), where $\langle F \rangle$ is variance in allele frequencies scaled by the expected allele frequency, averaged across space:

$$\langle F \rangle \equiv \frac{\int_{-\infty}^{\infty} \text{var}(p(x,t)) dx}{\int_{-\infty}^{\infty} (p(x,t)q(x,t)) dx}$$

[20]. High fluctuations (considerably above the dashed line) tend to be coupled with higher demographic fluctuations. For rare outliers $\langle F \rangle$ is yet higher than 2 (not shown). (B) The demographic fluctuations $\text{var}(\delta N)/N^2$ are substantially smaller, and rise roughly with $1/(N \sigma \sqrt{r})$. For both figures, variance in fluctuation is estimated from generations 4000 - 5000, using values 100 generations apart to avoid autocorrelation, and excluding ones where the genetic variance is less than half of the predicted value without genetic drift (which excludes collapsed populations).
Figure S2. Cline width decreases due to genetic drift as $w \sim w_s(1 - \exp[-\frac{N\sigma\sqrt{s}}{\sqrt{2}}])$. The reduction in the cline width $w$ due to genetic drift can be understood by interpolating between the deterministic cline width $w_s = 4\sigma/\sqrt{2s}$ (thick horizontal line) and the neutral limit $w_0$ (steep line on the left): $w = w_s(1 - \exp[-\frac{w_0(t)}{w_s}])$, black dashed line. (In the absence of selection but with genetic drift, the cline width $w$ stays finite and tends over time to $(w_0) \rightarrow 4\sigma^2N$ [41, p.2].) The dots give the observed cline widths with increasing $N$ and their mean deviations across 6 replicates. Using the method in (21), the reduction in cline width can be expressed as $w = w_s(1 - 0.392/(N\sigma\sqrt{s}))$ (dashed grey curve) - the fit would diverge though if $N\sigma\sqrt{s} \rightarrow 0$. Both dashed curves use the observed $w_s$ as the correct limit: the observed cline width $w$ stays somewhat lower (thin line) than predicted even as $N\sigma\sqrt{s} \rightarrow \infty$. This is because as $B$ increases, the selection on slight deviations of the trait mean from the optimum gets stronger and leads to steepening of the clines (see Fig. S1). Parameters: $b = 0.1$, $\sigma^2 = 1/2$, $V_s = 2$, $r_m = 1.025$, $\alpha = 1/\sqrt{20}$, $\mu = 10^{-4}$, carrying capacity $K$ increases from 4 to 260. Note that throughout, the formulae apply only to one-dimensional habitats: in two-dimensional habitats, the predictions will differ as the effect of genetic drift on a cline depends only weakly on selection [42].
Figure S3. Distribution of parameters used in the “random set”, as described in Table 1. Grey: all unscaled parameters. Light blue: Composite parameters. Dark green: Scale-free parameters.
Figure S4. Non-uniform carrying capacity generates a stable range margin. (A) The optimum changes across the environment with a constant gradient $b = 0.3$ - the population starts well adapted at the more central part of the habitat (lighter blue). (B) The population density declines away from the centre - red dots give the predicted failure of the adaptation, based on $B = 0.15N\sigma\sqrt{s}$. (C) Three representative clines are shown in black, other clines form the gray background (every tenth cline displayed). (D) Genetic variance is substantially lower than the deterministic prediction (black dashed line). The wiggly green line gives the predicted $V_{LE}^*$ including the effect of genetic drift: $V_{LE}^* = V_{LE}(1 - 0.392/(N\sigma\sqrt{s}))$ (see Fig. S2). Parameters: $b = 0.3$, $\sigma^2 = 1/2$, $V_S = 1$, $r_m = 1.1$, $\mu = 10^{-7}$. Population is shown after a stable range margin is reached at time = 60 000 generations (with the exception of the initial distribution of trait values, shown in light blue).
Figure S5. Sharp margin to a species’ range forms even when allelic effects $\alpha_i$ are non-uniform. With exponentially distributed allelic effects $\alpha_i$, the expansion slows down after 40 000 generations (see Fig. [S6], at the threshold predicted based on mean selection per locus $B^* = 0.15N\sigma\sqrt{\bar{s}} = 0.34$ (red dots). As in this example the allelic effects are not bounded, over very long times, as rare alleles with large effect are recruited (see Fig. [S7], the species’ range slowly stretches behind the threshold $B^*$. Parameters and subplot descriptions are the same as in Fig. [3]—with the exception that the allelic effects are exponentially distributed, with mean $\alpha = \sqrt{1/100}$. (C) Note that when allelic effects vary across loci, rarely a cline may establish in a reverse direction, correcting a substitution with a large effect on the trait mean. (D) As genetic variance $V_G$ increases towards the margins, it evolves to match the ever steeper environmental gradient: $V_G = b\sigma\sqrt{V_s}$, prediction shown in dashed line. Gradient in the central habitat is $b = 0.12; \sigma^2 = 1/2, V_S = 1/2, r_m = 1.06, K = 50, \mu = 2 \cdot 10^{-7}, \text{time} = 100 000$ generations (with the exception of the initial distribution of trait values, shown in light blue).
Figure S6. Range expansion slows down near the threshold based on mean selection coefficient even when the allelic effects $\alpha_i$ are non-uniform. Yet, over very long times, further alleles with large effects can be recruited as they are under stronger selection, and species’ range expands a little further. Example from Fig. S5. The extent of range expansion is only fully bounded by the substitution with the largest selection coefficient that can arise over a given time. For comparison, the rate of range expansion with equal allelic coefficients with $\alpha_i = \bar{\alpha}$ is given in gray (keeping all other parameters same, example from Fig. 3).

Figure S7. Over time, genetic drift degrades clines with small allelic effects $\alpha$. As more alleles with larger effect $\alpha$ contribute to adaptation, clines become narrower and under stronger selection. Parameters as in Fig. S5. Note that $\bar{s} = \frac{\bar{\alpha}^2}{2V_s}$ and in this example, $s = \alpha$. 

Figure S8. Alternatively, the threshold for range collapse can be expressed as 
\( \frac{b}{\alpha} \gtrsim 0.15 \ N r^* \). The threshold holds well unless spacing between the clines, \( \alpha/b \), is smaller than about 1/10 - this reflects the limits of our simulation system rather than a biological boundary – the deme-spacing is fixed to \( \Delta x \equiv 1 \). Data and their depiction are the same as in Fig. 2.
Figure S9. Selection on minute deviations from trait mean steepens the clines. Even for negligible genetic drift, clines get steeper than predicted for “perfect” adaptation as the effective environmental gradient $B$ increases. Predicted clines are given by dashed curves in (B, D). The blue and purple curves in (A, C) show the components of selection for a single central cline: $spq(p-q)$ (blue) vs. $-2spq\Delta$ (purple), where $\Delta = (\tau - bx)/\alpha$ gives a scaled deviation from the optimum. The “perfect” adaptation assumes the trait mean matches the optimum everywhere ($\Delta = 0$) - when the predicted and realised clines ought to match (for weak drift). As the effective environmental gradient $B$ steepens, the effects of small deviations of the trait mean from the optimum increasingly affect the clines. (A, B) Example with weak environmental gradient $B = 0.035$, where the cline widths are only 4% narrower than predicted because selection acts mainly (77%) via the diversifying term $spq(p-q)$. (C, D) Example with very steep effective environmental gradient $B = 1.2$, where the cline width is nearly twice as steep as predicted - correspondingly, the two components of selection are nearly the same: mean integrated selection components across all clines are 0.007 and 0.006, respectively. Both examples are chosen with very weak drift: $N\sigma\sqrt{s} = 23.7$ and $N\sigma\sqrt{s} = 22$ and very similar selection per locus $s = 1/80$ and $s = 1/60$. 


Figure S10. Species range can fragment when $B > 0.15N\sigma \sqrt{s}$ (Fig. 2), and additionally, $B > \sqrt{2A}$ (Fig. 4). Exact conditions that always lead to range fragmentation were not determined. Fragmented populations are shown in open circles in Figs. 3 and 4 (A) Typically, there is no adaptation in trait mean left within each sub-range. (B) Populations are disjunct, and across the habitat, the population size is considerably smaller than predicted for “perfect” adaptation. (C) Typically, there is no clinal variation although transiently, a few clines are maintained. (D) Correspondingly, genetic variance is mostly near zero. Parameters: $b = 1.18$; $\sigma^2 = 1$, $V_s = 0.44$, $r_m = 1.97$, $K = 29.2$, $\mu = 6 \cdot 10^{-8}$, $\alpha = 0.0093$, time = 5000 generations (shown at generation 4800). Note, that this fragmentation is not driven by edge effects that arise when population at the carrying capacity reaches the edge of the available habitat, where there is less maladaptive gene flow, which leads to local increase of density, followed by suppression of nearby populations towards the centre – and the effect propagates [43]; explained in [35, Text S1]. Here, the simulations are set up such that the population never reaches the margins of the available habitat.
Figure S11. Adaptation may suddenly fail if dispersal is too large. The threshold for collapse of adaptation (dashed line, $B \sim 0.15 N\sigma\sqrt{s}$) is weakly dependent on dispersal: to the first order, the effect cancels. Yet, for our model, the strength of density dependence $r^*$ decreases with genetic variance ($r^* = V_G/(2V_s)$), which in turn increases with $\sigma$; ($r^* \to b\sigma/(2\sqrt{V_s})$). When $r^{*2}/r_m$ becomes smaller than 1, further increase in dispersal is detrimental as it brings the population closer to the predicted threshold. This is because both $B = \frac{b\sigma}{\sqrt{2V_s}r^*}$, and $N\sigma\sqrt{s} = Kr^*/r_m\sigma\sqrt{s}$ are both dependent on $r^*$ — and the effects multiply. The scale for the colouring is adjusted (different to Fig. 2), so that the differences in the rate of range expansion (light to dark blue) and contraction (orange to red) are visible; gray dots again denote population that expanded less than one deme over 5000 generations. Parameters: $\sigma = [0.1, 4.24]$, $b = 0.45$, $V_s = 1$, $r^* \equiv 1$ hence $r_m = [1.02, 1.95]$; $K = 28$, $\mu = 4 \cdot 10^{-7}$, $\alpha = \sqrt{1/35}$, time = 5000 generations.
Figure S12. Effect of mutation rate on the rate of range expansion and local genetic variance. (A) Rate of range expansion increases about linearly with mutation rate $\mu$ per locus and generation ($5 \cdot 10^{-8} < \mu < 10^{-5}$). When mutation becomes too high, then the rate of expansion first decelerates, and for yet higher mutation rates ($\mu \gtrsim 10^{-2}$), the population starts to collapse. Note that in general, mutation rate per locus and generation is expected to be lower than about $10^{-4}$ (see Text S1). (B) Local genetic variance $V_G$ increases with mutation rate. For low to moderate mutation rates, genetic variance is maintained by mixing across the phenotypic gradient, $V_G = b \sigma V_s$. The dashed curve gives the prediction $b \sigma V_s + 2 \mu n_l V_s$, which assumes the components of genetic variance due to gene flow (first term) and mutation-selection balance (second term) combine additively, whilst all $n_l$ loci are at linkage equilibrium: the mismatch between the dashed curve and the realised genetic variance due to clinal variation, $\mu \ll s$ is not a sufficient condition for the contribution of mutation to be negligible. The top dashed line gives the maximum variance possible, $1/4 n_l \alpha^2$ (where $\alpha$ is a phenotypic effect of a single substitution). With increasing variance, population density drops steadily (not shown): eventually, species’ range starts to contract and population collapses. Parameters: $b = 0.4$, $\sigma^2 = 1/2$, $V_s = 1/2$, $r_m = 1.06$, $K = 50$, $\alpha = s = 0.01$, 800 genes. Initial population spans over 100 demes, population evolves over 5000 generations. For both plots, error bars give the standard deviations. Mutation rate for all simulations from the “random set” is set to one event per generation and the whole population (Text S1), which ensures that population can expand over 5000 generations, yet genetic variance is not significantly inflated by high mutation.
References: Supporting Figures