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LETTER

Why is adaptation prevented at ecological margins? New insights from individual-based simulations

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Abstract

All species are restricted in their distribution. Currently, ecological models can only explain such limits if patches vary in quality, leading to asymmetrical dispersal, or if genetic variation is too low at the margins for adaptation. However, population genetic models suggest that the increase in genetic variance resulting from dispersal should allow adaptation to almost any ecological gradient. Clearly therefore, these models miss something that prevents evolution in natural populations. We developed an individual-based simulation to explore stochastic effects in these models. At high carrying capacities, our simulations largely agree with deterministic predictions. However, when carrying capacity is low, the population fails to establish for a wide range of parameter values where adaptation was expected from previous models. Stochastic or transient effects appear critical around the boundaries in parameter space between simulation behaviours. Dispersal, gradient steepness, and population density emerge as key factors determining adaptation on an ecological gradient.

Keywords

Adaptation, dispersal load, ecological margin, habitat patch, individual-based simulation, population ecology, population genetics.

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INTRODUCTION

Geographical variation in fitness-related traits shows that populations can adapt to ecological change within a species' range (e.g. Hoffmann & Willi 2008). Nevertheless, many species' distributions end abruptly, either in space or in time, often without the presence of obvious ecological or physical barriers. Currently, ecological models can only explain such distributional limits if there is an increased chance of extinction at margins or if habitats become scarce and colonization more difficult (e.g. Holt & Keitt 2005), if there are large asymmetries in carrying capacities in the environment (Kawecki & Holt 2002), or if genetic variance is too low to allow perfect adaptation (see Kawecki & Ebert 2004; Kawecki 2008). Recently, models of ecological margins have made important advances by coupling population genetics with population ecology in that the match of a genetically variable trait to the optimum determines absolute fitness (see Lenormand 2002; Bridle & Vines 2007; Bridle *et al.* 2009a). These models typically pertain to ecological or patch margins, rather than species' margins, because they consider a continuous spatial area that is not many times greater than the dispersal distance, whereas species' ranges are larger and typically consist of discrete patches of habitat, especially near their margins (see Gaston 2003).

Dispersal along spatial ecological gradients generates a fitness cost (termed 'dispersal load'). When the trait mean matches the local optimum, this cost is generated by

dispersal to positions on the gradient where the optimum is different (the load equals the drop in fitness due to dispersal of one standard deviation). 'Standing load' arises due to the genetic variation around this optimum. Where the population fails to match the local optimum, there is an additional 'maladaptation load', which increases with the mismatch between the actual trait mean and its local optimum.

Limited adaptation (LA) occurs when genetic variance in the trait (which determines the response to selection) is low relative to the rate of change of the optimum. Three regimes therefore emerge from Kirkpatrick & Barton (1997): 'unlimited adaptation' (where the trait evolves to match the spatially changing selective optimum everywhere), 'LA' (where the population is well-adapted to the local optimum only in the centre of the species' range), and 'extinction' (where the population cannot be sustained at any point on the gradient). 'LA' behaviour is characterized by asymmetrical dispersal from the well-adapted central region to the poorly adapted margins.

In Kirkpatrick & Barton (1997), the genetic variance is fixed. Increasing the additive genetic variance allows better adaptation to the ecological gradient. However, additional genetic variation is supplied by dispersal between populations at different optima. Barton (2001) extended the Kirkpatrick & Barton (1997) model to include this effect. He showed that, for a range of quantitative genetic models, the increased evolutionary potential resulting from higher genetic variance allows adaptation across virtually any steepness of gradient. However, an absolute limit is reached where very high levels of variance (despite allowing the trait mean to closely match the local optimum everywhere) reduce mean fitness sufficiently (through standing load) to cause extinction of the entire population.

Models that allow dispersal to increase genetic variance therefore make it hard to explain why adaptation fails at ecological margins. What are these models missing that actually limits evolution at ecological margins? One limitation is that these models do not include stochastic effects such as genetic drift or increased extinction risk at the low population densities that may frequently occur in real habitat patches. Successful occupation of patches in nature also requires a colonization phase which is not included in these models. Alleaume-Benharira *et al.* (2005) incorporated the effects of drift in a stepping-stone model on an environmental gradient. Their results suggest that an intermediate migration rate provides the best compromise between dispersal load and genetic drift, increasing fitness in marginal populations as well as range-wide mean fitness. A similar result was also observed by Gomulkiewicz *et al.* (1999). In this study, an individual-based simulation model briefly introduced by Butlin *et al.* (2003) is developed further, and its behaviour compared to deterministic predictions. The basic assumptions of the model closely match Barton's (2001) analytical 'two-allele' model, which assumes that the quantitative trait is determined by n freely recombining bi-allelic loci with additive effects.

THE MODEL

The evolutionary dynamics for the simulated population take place within a continuous region of maximum extent 32 000 × 1000 units. There is an ecological gradient along the long (x) axis, which is uniform with slope b. The area is simulated as a cylinder; the edges of the second, short (y) axis are joined. Individuals occupy the vertices of a grid and more than one individual can occupy any given position. The model follows the fate of a starting population of 500 individuals that are initially distributed in the central 500 × 1000 units of the environment. Assuming an allelic effect size of α, their phenotypes ranged from $z_{opt} - 2\alpha$ to $z_{opt} + 2\alpha$ where z_{opt} is the optimum phenotype at the centre of the range.

The phenotype is determined by diploid unlinked bi-allelic loci with additive effects that mutate symmetrically at rate μ ($\mu = 0.0001$ per locus per generation unless otherwise stated). For most runs, 64 loci were used, with allelic effect $\alpha = 1$ (maximum phenotypic range = 0–128). In selected runs, the phenotype was controlled by 128 loci, each allele having effect 0.5. We also followed the fate of ten loci ($\mu = 0.001$) that were not subjected to selection. Population growth is logistic, dependent on the local density of individuals (N) and local carrying capacity (K). Females choose mates from the males available within a finite mating distance (MD), with a probability proportional to the fitness of each male at its position on the ecological gradient. This was fixed at MD = 150 (see Butlin *et al.* (2003) for a description of the effect of male dispersal on range expansion). Offspring then disperse and selection occurs after dispersal through the number of offspring produced by each female and the mating success of males. If no male is available within the mating area, the female leaves no offspring.

In order to minimize edge effects as the spatial extent of the population approaches the maximum x range, observations of simulation behaviour and calculations of genetic and demographic parameters were restricted to a central portion of the observed population, 8000 units wide.

The fitness of both sexes is determined by the same function: $W_F = W_M = 2 + r_F\ (1 - N/K) - s(bx - z)^2/2$ ($W \geq 0$). The number of offspring a female leaves is drawn from a Poisson distribution with mean W_F. Females choose males with probability proportional to W_M. In our model, there is no selective mortality or random effects on death rates. Generations are non-overlapping and the maximum rate of increase $r_m = r_F/2$; r_F is set to 1.6. K is the carrying capacity within a circle of radius 50 around the focal individual, N (density) is the number of individuals in such a circle (initially $N = 7.85$ individuals). $U_x = bx$ is the

phenotypic optimum at the point (x) on the gradient occupied by the female. The parameter s measures the rate of decline in fitness for phenotypes that depart from the optimum; the strength of stabilizing selection V_S is $1/(2s)$. Here, V_S is set to 4 and b (the spatial gradient in the optimum) is set to 0.004. Note that when drift and the effects of the margins are negligible, increasing dispersal with constant gradient is equivalent to increasing the gradient with constant dispersal (by dispersal of a distance σ, fitness decreases by $\frac{b^2\sigma^2}{2V_S}$).

The growth rate of a particular phenotype is

$$r[z,N] = r_m\left(1-\frac{N}{K}\right) - \frac{\left(z-U_x\right)^2}{2V_S},$$

the growth rate of the population is the average over all phenotypes, hence

$$r_N = \langle r[z,N]\rangle = r_m\left(1-\frac{N}{K}\right) - \frac{\left(\bar{z}-U_x\right)^2}{2V_S} - \frac{V_P}{2V_S}$$

Throughout, we assume addictive genetic variation, and no environmental variation in phenotype. (see Kirkpatrick & Barton 1997; Barton 2001; Polechová *et al.* 2009).

Total dispersal (TD) is determined by the combined effects of the two phases of dispersal, by males or their gametes (MD) and by offspring (D). The offspring of each female disperse to new positions in the habitat with a Gaussian distribution of dispersal distances, mean 0 and standard deviation D, in uniformly distributed random directions. As mating is a form of dispersal by males (or their gametes), the standard deviation of TD is given by $TD = \sqrt{D^2 + \frac{1}{2}SM^2}$ (see Crawford 1984), where SM is the expected distance between mating partners when a female chooses from a circle with radius MD, hence SM = ($1/\sqrt{2}$ MD). The expected distance σ along the x-axis is only in one dimension, hence $\sigma = TD/\sqrt{2}$.

For an infinite population, the population dynamics should approximately match the continuous time model described by eqn 7 in Kirkpatrick & Barton (1997) and if no linkage disequilibria (LD) are generated, the evolution of phenotype should follow the 2-allele n loci model of Barton (2001). In our model, population regulation occurs over discrete generations, and populations are finite in size, therefore allowing stochastic effects on demography and allele frequency, and the generation of lags. The program was written in C++, developed from that introduced by Kawata (2002), and is available on request from MK (kawata@mail.tains.tohoku.ac.jp). Output from the simulations for a given generation was analysed using a Genstat v10.1 (VSN International; http://www.vsni.co.uk) program (see Supplementary Information). This program is available on request from RKB (r.k.butlin@sheffield.ac.uk). Numerical predictions, based on analytical models, were calculated using Mathematica v7.0.0 (Wolfram Research, Champaign, Illinois, USA).

RESULTS

Simulations were typically run for 3000 generations, although by 1000 generations, one of three outcomes was usually achieved: (1) range expansion to fill the entire ecological gradient (UA); (2) extinction throughout the range (E); or (3) LA, where a cline in trait mean formed which was shallower than the optimum, generating a finite range (Fig. 1). At the boundary between UA and LA, a region of 'slow adaptation' (SA) was observed, where the population filled the available habitat, but took more than 3000 generations. Long runs (up to 10 000 generations) were used at the boundaries between these behaviours to explore the temporal stability of different outcomes, the rate at which allele-frequency clines formed (and a stable gradient in trait mean was reached), and how quickly different components of genetic variance (segregating variance and variance due to LD) became stable (Table S1).

Below we describe the behaviour of the model, and consider how it matches the predictions of the population genetic models of Kirkpatrick & Barton (1997) and Barton (2001).

Unlimited adaptation and deterministic extinction

For many parameter combinations (the dark blue region in Fig. 1, shown for a typical run in Fig. 2a), the populations spread to fill the available area, albeit with reduced density as standing load increased (Fig. 3a). This fits the predictions of the bi-allelic model of Barton (2001), where genetic variance is allowed to evolve but linkage equilibrium (LE) is assumed. Such behaviour was characteristic of runs with high carrying capacity unless the standing load due to dispersal was very high, leading to extinction over the whole range, also as predicted (see below).

Barton (2001) derived the equilibrium solution for an additive trait with n loci and two alleles. Assuming LE, the width of the cline for each locus should be $w = \frac{4\sigma\sqrt{V_S}}{\alpha}$. As each cline contributes to a shift of trait mean by 2α, there need to be $b/(2\alpha)$ clines per unit distance to match the gradient b. This implies variance $V_G = b\sigma\sqrt{V_S}$. This prediction was approached by our simulations when the population adapted over the whole range, unless selection due to dispersal across the spatial gradient was strong (Fig. 3b,c,d). In our simulations, the critical limit for extinction (independently of K) occurred at the expected steepness of gradient, though the genetic variance was higher than predicted (Fig. 1, and see

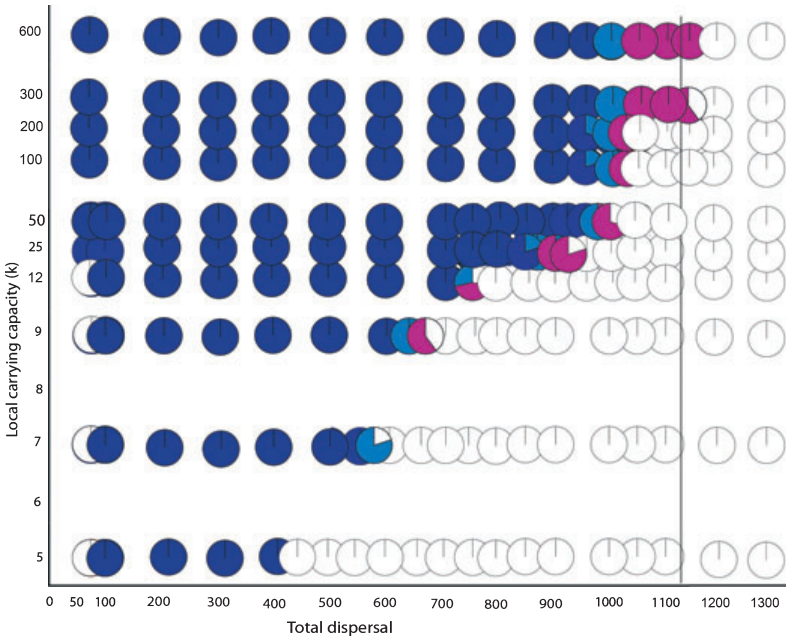

Figure 1 Effect of carrying capacity, *K*, on simulation outcomes after 3000 generations, or when expansion first reached the margins of the simulated area, for 10 runs per parameter combination as follows: blue (unlimited adaptation); light blue (slow spread); purple (limited adaptation, some clinal divergence); white (extinction). For parameters that apply to all simulations, see text. The solid line at TD = 1130, is the analytical prediction for the limit of population persistence assuming a Gaussian distribution of phenotypes.

below for derivation). This was probably because the assumption of a Gaussian distribution of phenotypes was violated at high values of dispersal, where variance due to LD was high (Table S1). Kurtosis in these simulations was above zero, which would reduce the fitness cost of genetic variance (e.g. UA: TD400, K50, median kurtosis = 0.35; UA: TD900 K50: median kurtosis = 0.55; LA: TD1000 K50; median kurtosis = −0.31).

Analysis of cline widths and positions for each locus (Table S1; Fig. 2a) showed the expected even spacing of cline centres. The trait mean therefore closely matched the optimum and population density remained uniform throughout the range (Fig. 2a). Phenotypic variance, density, and cline widths and spacing were close to those expected from analytical predictions (Table S1, Figs 2a, 3). The increase in total and segregating variance with dispersal was unaffected by carrying capacity (all the values for variance lie along the same line, regardless of *K*, provided the population adapted to the gradient), demonstrating that the effects of gene flow rather than neighbourhood population size dominated genetic variation throughout the range. As a result, neutral genetic variance was not reduced at the margins (Fig. 2).

As dispersal increased, segregating variance fell below analytical predictions, although the total genetic variance exceeded predictions due to a contribution from LD (Fig. 3b-d, Tables S1, S2). Note that total variance is simply the sum of segregating variance and variance due to LD (Fig. 3d). The LD can be predicted from a balance between recombination and the mixing effect of dispersal (Barton 1986, p. 418). The pair-wise LD depends on the gradients in allele frequencies at two different loci whose clines overlap, $D_{ij} = \frac{\sigma^2}{r_{ij}} \frac{\partial p_i}{\partial x} \frac{\partial p_j}{\partial x}$ (where r_{ij} is the recombination rate between loci i and j, set to ½ here). LD variance is then $2 \sum_{i \neq j} \alpha_i \alpha_j D_{ij}$ (for 64 loci, the allelic effects α are all set to 1; for 128 loci, α is set to 0.5). We did not estimate, however, what the cline shape is with LD. In the Fig. 3, we use the expected clines for uniform adaptation (UA) and LE, hence we necessarily overestimate the LD variance component. If LD variance is calculated from the observed clines, we get a significantly better fit (as expected, see Tables S1 and S2). Observed mean cline width increased with dispersal, as expected (Fig. 3c, Tables S1 and S2). However, observed mean width was consistently lower than predicted, because LD increases the effective selection on each locus. This effect increased as standing load increased, until populations entered the region of parameter space where adaptation was limited (LA). Clines were then present for only a few loci (see below), and these clines were typically very wide (Tables S1 and S2).

Even at very high values of *K*, unlimited adaptation was prevented with high TD, as predicted by deterministic models. At equilibrium, population density is $N = K(1 - \frac{V_G}{2r_m V_S}) = K(1 - \frac{b\sigma}{2r_m\sqrt{V_S}})$ (Barton 2001). The cost of dispersal across the gradient is $\frac{b^2\sigma^2}{2V_S}$. Extinction should therefore occur when the effect of phenotypic variance causes the population growth rate to become zero, regardless of population size, i.e. when $b > \frac{2r_m\sqrt{V_S}}{\sigma}$ (Barton 2001). As b is fixed to 0.004, $r_m = 0.8$ and $V_S = 4$, extinction should be observed for TD greater than TD = $\sigma/\sqrt{2}$ = 1130. In our simulations, the behaviour

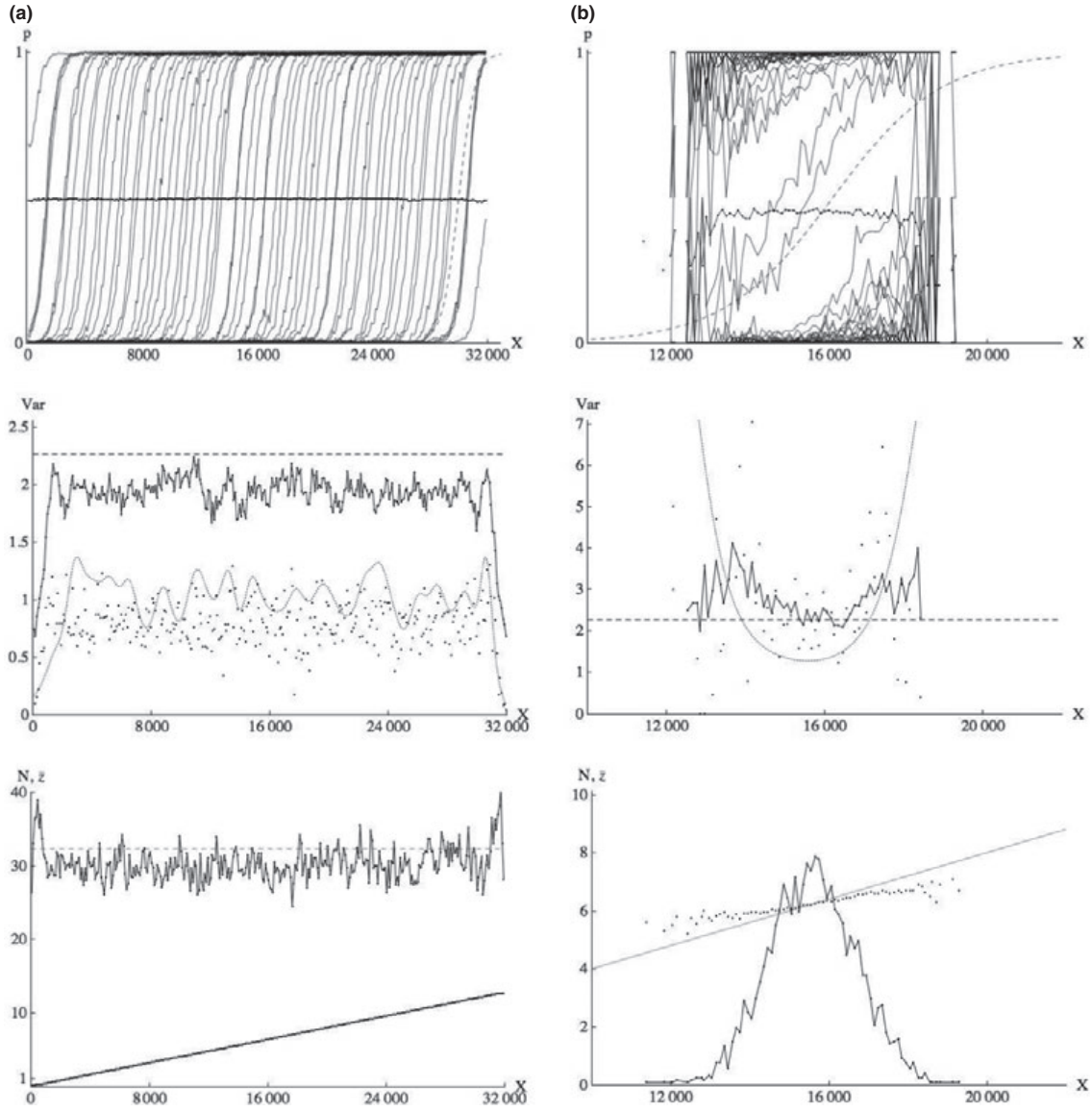

Figure 2 Behaviour of typical simulation runs leading to: (a) unlimited adaptation (K = 50 TD = 400); (b) limited adaptation (K = 50; TD = 975). Upper panels show individual clines in allele frequencies and the prediction (dashed line) for cline shape of a locus under selection assuming LE. The bold, solid line shows heterozygosity at 10 neutral loci. The central panels show segregating variance (continuous line), prediction for variance under LE (dashed line, from Barton 2001) and genetic variance due to LD (dots), with the prediction for LD variance (dotted line, from Barton 1986). Lower panels show density (continuous line), predicted density for uniform adaptation (a only, dashed line), actual trait mean (dots, value divided by 10) and gradient in the trait optimum (solid grey line).

changed to LA before this value, even when carrying capacity was high (Fig. 1). Behaviour varied among runs but only extinction occurred at a marginally higher TD than predicted. This value of TD and b therefore represents the absolute limit where the genetic variance required to track the optimum cannot be sustained demographically given this maximum reproductive rate.

Extinction at low carrying capacity

At the opposite end of parameter space, very low dispersal at low carrying capacities also leads to extinction (Fig. 1). This extinction behaviour was not observed when females were allowed to mate with the nearest available male. Extinction at these parameter combinations is therefore due

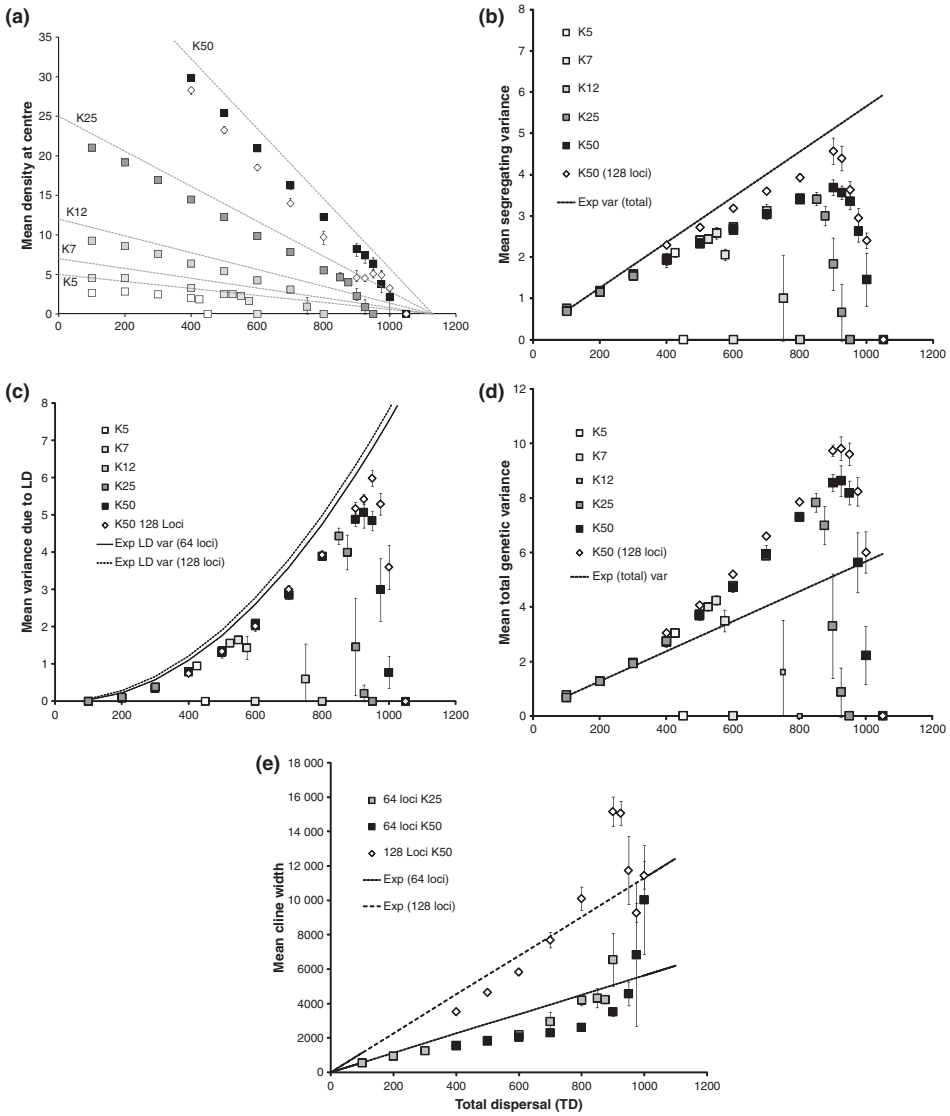

Figure 3 Effects of dispersal and carrying capacity on: (a) population density; (b) segregating variance; (c) variance due to LD; (d) total variance and (e) cline width. All runs used 64 loci except for the K50, 128 loci runs indicated by open diamonds. Error bars are standard deviations, based on five repeats, or total range for population density. Note that for high values of TD these values may be summed over runs with variable outcomes (see Fig. 1). Analytical predictions (dashed lines) are based on Barton (1986, 2001).

to Allee effects (the failure of females to find mates within their prescribed MD).

As dispersal load increased, mean density decreased, close to the rate predicted by Barton (2001), although actual densities were slightly lower than expected because total genetic variance was consistently higher than predicted (Tables S1 and S2 and Fig. 3a–d). However, with carrying capacity below $K = 300$, extinction occurred at dispersal values significantly lower than the absolute predicted limit of TD=1130 (Fig. 1). Typically, populations at low values of K became extinct very quickly (within 100 generations), before any cline was formed, or any population growth could occur. The proportion of runs that survived this early extinction increased with higher K. For runs that survived rapid extinction, transient 'LA' behaviour was observed, where a shallow trait mean gradient formed and some population growth occurred. However, extinction occurred within 3000 generations (and usually within 1000 generations), for parameter combinations for which the expected mean density fell below approximately 2 (Fig. 3a). A simple deterministic,

discrete-time series model (with population dynamics as in our simulation and observed neighbourhood size) revealed that the population growth rate is too small to cause simple demographic extinction due to delayed feedback.

Limited adaptation

As TD increased, a narrow region of LA was observed, with some parameter combinations generating either UA or LA behaviour in different runs. This region became wider as carrying capacity increased, and dispersal load drove populations deterministically to low densities (i.e. at high dispersal and high carrying capacity; Figs 1, 2b). Here, the trait mean only matched the optimum towards the centre of the range, and density declined as the mismatch increased further away from this point, resulting in a population that remained bounded in space. These LA populations persisted for at least 10000 generations (Table S1). However, variances and trait slopes varied during these runs, with resulting fluctuations in population size (and spatial extent) over short timescales.

In the LA region of parameter space, the gradient in trait mean was caused by clines in fewer loci and clines were wider on average than in the region of UA. Segregating variance was also lower than predicted for UA (Fig. 3b). Given the segregating variance, the gradient of the trait mean was close to that expected for LA under the phenotypic model with fixed variance of Kirkpatrick & Barton (1997) (Figure S3a, Table S1). If genetic variation rose above the threshold where LA exists for a given standing load vs. dispersal load, 'SA' rather than LA was observed (Figure S3b).

'Slow adaptation' behaviour, where the population took more than 3000 generations to fill the range (Table S1; Figure S1a), was observed at values of dispersal typically just below those producing LA behaviour. For some parameter combinations, either LA or SA was observed for different runs. During the establishment of these populations, a sigmoid trait mean gradient was observed, with the mean departing from the optimum at the range edges. These simulations also showed a gradual increase in the number of loci with clines that contributed to adaptation, whereas the LA runs fluctuated around a low number of clines (Table S1; Figure S1b).

Sensitivity analysis

The robustness of our results was tested by varying: (1) mutation rate from 0.0001 to 0.001; (2) the number of initially polymorphic loci from 2 to 20; (3) starting population size from 250 to 500; and (4) the number of loci from 64 loci (each with α of 1) to 128 loci (each with α of 0.5) (Tables S1b and S2b; Figure S2). In all cases, although there were small changes in where the transitions between behaviours occurred, the qualitative behaviour of the model was unchanged.

DISCUSSION

Our results show that the evolutionary fate of a population adapting to an ecological gradient depends on local carrying capacity (K) as well as dispersal relative to gradient steepness. In particular, we observe extinction at lower levels of dispersal/steepness of gradient than predicted by Kirkpatrick & Barton (1997) and Barton (2001), whose models do not include the stochastic effects associated with finite population sizes. In our model, these stochastic effects are variation in the numbers of offspring left by a females or fathered by males and dispersal directions and distances (and mutation). These lead to spatial and temporal variation in densities and allele frequencies. Excluding these effects is an important limitation because populations at the margins of ranges are expected to be small.

The Kirkpatrick & Barton (1997) and Barton (2001) models begin with complete adaptation to an ecological gradient, and explore the parameter combinations where this is stable. They ignore the effects of colonization and of stochasticity in the supply of mutations and the establishment of clines. By contrast, our simulations consider whether a small population adapted to the centre of a gradient can spread. This initial population has no phenotypic gradient. If dispersal is high, the population spreads widely across the environmental gradient, increasing initial load due to the mismatch between individuals' phenotypes and the local optimum. This causes very rapid extinction, within a wide region of parameter space where unlimited adaptation was predicted by Barton (2001). The lower the carrying capacity, the lower is the threshold dispersal for this rapid extinction. Occasionally, runs around this area of parameter space escape early extinction and form shallow clines that allow the population to expand slightly and briefly enter the LA behaviour described below, before stochastic effects cause extinction, usually within 1000 generations. Given that neighbourhood sizes are initially large, these stochastic effects are likely to be due to dispersal affecting local allele frequencies, so taking the phenotypic mean away from the local optimum, rather than simple demographic stochasticity. At higher carrying capacities, although population density is kept low due to dispersal load, LA behaviour becomes increasingly frequent (Fig. 1), and is observed over a wider region of parameter space. Increasing genetic variation in the initial population does not allow spread at higher dispersal because this variation also brings with it an increased standing load. Similarly, increasing initial population size does not aid adaptation in this region of parameter space because fitness is then affected by density-dependence, making selection for adaptation less effective.

Our results therefore suggest that a species' occupancy of a habitat patch is mainly limited by the cost of dispersal along the patch's ecological gradient, which prevents the establishment of a population soon after colonization.

At high carrying capacity, our simulations behave largely as predicted by analytical models which assume LE, an approach that is valid under weak selection. Stochastic effects, either on genotype frequencies or population sizes, have little effect in this region of parameter space where UA is achieved. However, linkage disequilibrium is generated by overlapping clines in allele frequency. This increases the standing load so that population density decreases slightly more than predicted under LE (Figs 1, 3a). The observed LD is predictable from the dispersal and the observed shapes of clines using the moderate selection approximations of Barton (1986). This fit is surprisingly good considering that the selection generated in these simulations is strong where dispersal is high (equivalent to a steep gradient in the environmental optimum).

Linkage disequilibrium is expected to be generated in natural populations wherever multiple loci contribute to local adaptation and selection is moderately strong. Evolutionary responses to ecological gradients are typically due to multiple loci, for example in heavy-metal tolerance in grasses (see Macnair 1993), and in insecticide resistance in *Culex* mosquitoes (Labbe *et al.* 2005, 2007). In *Anthoxanthum* grasses, LD appears elevated at population margins, with concomitant affects on local adaptation (Antonovics 1976). However, high levels of LD may not always be the case. For example, allele-frequency variation at only a few QTL loci (located within a single inversion) explains up to 60% of the divergence in body size along latitudinal clines in *Drosophila melanogaster* in Australia (Rako *et al.* 2006; Kennington *et al.* 2007).

In our simulations, the selection strength generated by dispersal along a fixed ecological gradient reaches its absolute limit at TD = 1130. How does this compare to likely levels of dispersal load in natural populations? Burt (1995, 2000) reviewed available data on TD load, giving a median of about 0.02, with most values being below 0.1. This latter value would correspond to TD = 316 (see Polechová *et al.* 2009). As variance is maintained by dispersal (and $V_G = V_P$), TD = 316 generates $V_P/V_S = b\ \mathrm{TD}/(2\ V_S)^{1/2} = 0.45$. This is moderately strong selection (estimates from Kingsolver *et al.* 2001 suggest that the median V_P/V_S in natural populations is about 0.2; but see also Hereford *et al.* 2004).

For a narrow range of parameter values, we observe LA behaviour, where the trait mean is shallower than the optimum. This generates a population with a spatial extent that is bounded by maladaptation, as may be observed in sticklebacks in North America, where dispersal from large lake populations to smaller stream populations may limit adaptation to their local ecological optimum (Moore & Hendry, 2009). However, because LA behaviour occurs only in a restricted portion of parameter space in our model we consider it unlikely to be a common explanation for limited ranges.

At high values of *K*, simulated populations persist in the LA state for thousands of generations; at lower values of K, this behaviour may only be a transient state *en route* to extinction. At the boundaries of this parameter range, the outcome varies between simulation runs and must have a chance element. What appears to happen in LA runs is that genetic variance initially increases due to the formation of clines in additional loci until the population reaches a locally stable state. The population is then subjected to temporal fluctuations in trait gradient and population density, but fails to achieve the optimum gradient (and hence unlimited adaptation). Where this occurs in our simulations, the trait means and variances fall within the region that also gives a finite range in the models by Kirkpatrick & Barton (1997) and Polechová *et al.* (2009) (Figure S3a), suggesting that the swamping effect of asymmetrical dispersal is stronger in these runs than its spreading effect on variance. However, more theoretical work is required to predict the conditions at which the population evolves into this state. If the genetic variance becomes higher than the Kirkpatrick & Barton (1997) threshold for UA, the population can move from the LA regime into the SA regime (Figure S3b). Again, such fluctuations in genetic variance near the margin are probably due to stochastic effects, so we cannot predict when the variance will cross this threshold, or when a specific parameter combination will generate a particular behaviour. Similar 'SA' is also observed in some source-sink models (e.g. Holt *et al.* 2003).

Empirical data for these variables are difficult to obtain, but do suggest that the amount of genetic variance determines rates of adaptation in real populations. For example, Willi & Hoffmann (2009) used data from experimental populations of *Drosophila birchii* subjected to heat-knockdown selection, and suggest that large populations persist due to reduced stochasticity in growth rate, higher reproductive output, and more additive genetic variation in heat resistance. Similarly, species' ranges in Australian *Drosophila* appear to be limited by low levels of additive genetic variation in stress resistant traits (Kellerman *et al.* 2006; Hoffmann & Willi 2008; Bridle *et al.* 2009b; Kellermann *et al.* 2009).

Relevance to population and species' margins in nature

The population genetic models explored here represent patches of habitat within species' ranges, within which persistence depends on evolving to match the ecological gradients observed along their length. Such patches may vary substantially in their quality (carrying capacity), even when populations within them match the local optima. Our results show that, where the carrying capacity of a patch is large,

a population can usually persist and evolve to match the optimum across the entire patch. Such patches might be typical of the core of the species' range. In more marginal patches, with lower K or steeper gradients, populations may persist but undergo only limited expansion, generating a cline in trait mean that is shallower than that demanded by selection. However, colonization of new patches within which the ecological gradient is steep (either inherently, or due to higher dispersal) will be difficult; many populations will fail to expand due to maladaptation load, even if initial population size is large and the population is adapted to some part of the patch. Such effects may be particularly important given that colonizers tend to show elevated levels of dispersal (Hughes *et al.* 2007; Duckworth 2008; Anderson *et al.* 2009).

Real habitat patches are likely to be characterized by central areas with shallow ecological gradients, with steeper gradients at the edges, rather than the linear gradients considered here. Patches beyond the current range margin may also have core environments that differ from occupied patches (see Holt *et al.* 2004). In this case, the genetic variance maintained along gradients within occupied patches may aid colonization, as is observed in Holt *et al.* (2005). Understanding ecological margins therefore requires integrating the effects of gradients within and between patches, and the effects of dispersal on colonization as well as on load. Limits to adaptation in natural populations also depend on other factors: how ecological gradients vary in time as well as space (Polechová *et al.* 2009), biotic as well as abiotic interactions (Bridle *et al.* 2009a), and the form of density-dependence (Filin *et al.* 2008). It is also unlikely that any ecological gradient will remain constant over even a few generations, let alone several thousand. Fluctuating selection could also elevate genetic variance substantially, without necessarily incurring high dispersal load. Given the key effect of dispersal on adaptation, it is important to understand when mean dispersal distances, or conditional or habitat-specific dispersal (Holt 2003) can evolve to aid expansion along otherwise impenetrable ecological gradients (Oliveri *et al.* 1995; Dytham 2009). This requires the development of more complex models as well as the gathering of empirical data on genetic variation in fitness, levels of dispersal, colonization rates and the local steepness of ecological gradients. In particular, future exploration of models where colonization between patches is combined with evolutionary responses to within-patch gradients is likely to prove fruitful. Understanding these models will involve studying the transient effects in early generations that our current model suggests are critical for successful colonization.

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SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of this article:

Figure S1 Plots of slow adaptation behaviour: TD950 K50, at generation 500, 1000 and 5000, compared to a limited adaptation run TD975 K50; the sigmoid shape of the trait slope indicates slow rather than persistently limited spread (up to 10 000 generations). Note also the steady increase in number of clines involved in the change in trait mean over time.

Figure S2 Plots of limited adaptation and full adaptation for 128 loci runs, for comparison with Figs 2a and b.

Figure S3 The relationship between gradients in trait means (β) and segregating variance (Vg) in runs with limited adaptation (a; top panel) or slow adaptation (b: lower panel).

Table S1 Temporal stability of runs at different parameter combinations for UA, SA and LA, showing rate of recruitment of clines, and match of observed clines widths, spacings, genetic variance, trait slope and density to expectations. These are shown for runs where the trait is controlled for (a) 64 loci and (b) 128 loci.

Table S2 Expected and observed cline widths, cline spacing, and segregating variance with increased TD, *K*, and numbers of loci. These are shown for runs where the trait is controlled for (a) 64 loci and (b) 128 loci.

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