Deleterious Mutations Can Surf to High Densities on the Wave Front of an Expanding Population

Justin M. J. Travis,† Tamara Münkemüller,‡§ Olivia J. Burton,* Alex Best,†‖ Calvin Dytham,¶# and Karin Johst†*

*Zoology Building, School of Biological Sciences, University of Aberdeen, Tillydrone Avenue, Aberdeen, AB24 2TZ Scotland, UK; †Natural Environment Research Council Centre for Ecology and Hydrology, Hill of Brathens, Banchory, Aberdeenshire, AB31 4BW, UK; ‡Department of Ecological Modelling, UFZ Helmholtz Centre for Environmental Research, Permoserstraβe, 15, 04318 Leipzig, Germany; §Forest Ecology, Department of Environmental Sciences, ETH Zurich, CH-8092 Zurich, Switzerland; ‖Department of Animal and Plant Sciences, University of Sheffield, Sheffield, S10 2TN, UK; ¶Vegetationsökologie & Naturschutz, Universität Potsdam, 14469 Potsdam, Germany; and #Department of Biology, University of York, York, YO10 5YW, UK

There is an increasing recognition that evolutionary processes play a key role in determining the dynamics of range expansion. Recent work demonstrates that neutral mutations arising near the edge of a range expansion sometimes surf on the expanding front leading them rather than that leads to reach much greater spatial distribution and frequency than expected in stationary populations. Here, we extend this work and examine the surfing behavior of nonneutral mutations. Using an individual-based coupled-map lattice model, we confirm that, regardless of its fitness effects, the probability of survival of a new mutation depends strongly upon where it arises in relation to the expanding wave front. We demonstrate that the surfing effect can lead to deleterious mutations reaching high densities at an expanding front, even when they have substantial negative effects on fitness. Additionally, we highlight that this surfing phenomenon can occur for mutations that impact reproductive rate (i.e., number of offspring produced) as well as mutations that modify juvenile competitive ability. We suggest that these effects are likely to have important consequences for rates of spread and the evolution of spatially expanding populations.

Introduction

The biology of range shifting has assumed considerable importance in recent years. Invasive species pose a major threat to native biodiversity and have widespread and substantial economic impacts (Sharma et al. 2005). Current rapid climate change is already responsible for the distributional shifts of many organisms (Parmesan and Yohe 2003), and recent predictions indicate that it may drive a high proportion of species to extinction over the next century (Thomas et al. 2004). Research geared toward predicting the future biogeographic ranges of species is booming, and there have been some major advances, not least in the development of increasingly sophisticated climate niche models that project future distributions (e.g., Araujo, Pearson, et al. 2005; Araujo, Whittaker, et al. 2005).

Somewhat belatedly, there is a growing recognition that evolutionary processes can play an important role in invasion biology (see reviews by Hänfling and Kollmann 2002; Lambrinos 2004; Hastings et al. 2005). Hybridization (e.g., Ellstrand and Schierenbeck 2000; Bossdorf et al. 2005), local adaptation (e.g., Quinn et al. 2001; Reznick and Ghalambor 2001; Parker et al. 2003; Butin et al. 2005; Hammershøj et al. 2006), and life history evolution (e.g., selfing rates—Daehler 1998; resistance to herbivores—Garcia-Rossi et al. 2003; dispersal behavior—Simmons and Thomas 2004; Phillips et al. 2006) have all now been implicated as determinants of either the probability that an introduction leads to an invasion or the spatial dynamics of the invasion.

Key words: evolution, invasion, range shifting, evolvability, epidemic, climate change.

E-mail: justin.travis@abdn.ac.uk.

Mol. Biol. Evol. 24(10):2334–2343. 2007
doi:10.1093/molbev/msm167
Advance Access publication August 16, 2007

Few theoretical studies have incorporated evolutionary processes into models of range expansion. However, some progress has been made in this direction: several studies have considered how genetic diversity should be structured in tree species that have undergone range changes, and these have demonstrated the important role that can be played by occasional long-distance dispersal (e.g., Le Corre et al. 1997; Austerlitz and Garnier-Géré 2003); García-Ramos and Rodríguez (2002) demonstrated the interacting roles played by local adaptation and habitat heterogeneity in determining the rate of spread of invasion; Travis and Dytham (2002) showed that range expansion may be accelerated by the evolution of increased rates of dispersal at the expanding front. Rather similar spatial models to those employed by Travis and Dytham (2002), Edmonds et al. (2004), and Klopfstein et al. (2006) demonstrate that neutral mutations arising on the edge of a range expansion sometimes "surf" on the wave of advance and can thus reach a larger spatial distribution and higher frequency than would be expected in stationary populations. Klopfstein et al. (2006) suggest that this surfing phenomenon may increase the rate of evolution of spatially expanding populations.

In this study, we examine whether the surfing behavior described for neutral mutations is likely to be equally important for the spatial dynamics of nonneutral mutations. Previous work on neutral mutations has highlighted the importance of the initial location of the mutation relative to the edge of the range expansion, and we assess whether this is equally important for mutations that affect fitness. Additionally, we look at allele frequency clines (AFCs) and establish characteristic spatial patterns for mutations with different fitness effects. In the light of our results, we reconsider whether mutation surfing is likely to accelerate evolutionary adaptation during range expansion. These results have potentially important implications for the study of range expansions in at least 3 contexts: invasion biology,

range shifting of biodiversity in response to current climate change, and the settlement of Europe by modern humans.

The Model

Our discrete time model is a direct extension of those used in recent, similar studies (Edmonds et al. 2004; Klopfstein et al. 2006) and has much in common with other cellular models used to study invasion dynamics (e.g., Travis and Dytham 2002). We simulate the range expansion of haploid individuals existing on a rectangular 2-dimensional stepping-stone lattice (25×100 demes) with absorbing boundaries. Initially the whole lattice is empty but suitable for colonization. Four individuals, carrying the same allele, are introduced into the middle of the left side of the grid (position [0,12]) to initiate the range expansion. Simulation of a generation consists of 2 steps; within-patch dynamics and dispersal between patches. The density of a deme is logistically regulated with carrying capacity K and intrinsic growth rate r. Dispersal can occur with individuals moving from their natal deme to one of the nearest 4 neighboring patches. In most simulations, we employ absorbing boundary conditions, where individuals moving into an unsuitable patch or dispersing off the edge of the lattice die. However, we also run some simulations employing reflective boundary conditions and compare the outcomes.

We allow for the occurrence of a new mutation exactly as specified by Klopfstein et al. (2006). The (x, y) coordinates of a deme are specified, where the new mutation should appear, along with the number of generations elapsed between the initial colonization of this deme and the occurrence of the mutation. Unless stated otherwise, in the simulations presented here, the mutation occurs at position (10,12) in the first generation following colonization of this deme. A single individual in this deme is selected at random to carry the new allele. Individuals carrying the mutant allele then experience the same series of events as the rest of the population, with demographic regulation preceding dispersal. In all the simulations reported in this study, the simulations continue either until the mutation has become extinct or until it has survived for 500 generations.

The previous studies by Edmonds et al. (2004) and Klopfstein et al. (2006) considered only neutral mutations, and they incorporated drift by using the binomial distribution. In this paper, we extend these previous models by allowing mutations to alter the relative fitness of individuals. We incorporate the processes of selection and drift as follows. In each generation, we assume that all the individuals give birth to a number of offspring that is drawn at random from the Poisson distribution with mean r. Offspring inherit their genotype from their parent. This produces a pool of juveniles within each deme that may consist of a mixture of mutants and nonmutants. If the number of juveniles is smaller than K, then all survive to become the adults of the next generations. Otherwise, the composition of mutants and nonmutants that survive juvenile competition to comprise the K adults in the next time step is drawn at random from Wallenius' noncentral hypergeometric distribution (Wallenius 1963). Using this distribution provides a computationally efficient way of simulating biased lottery competition. Four parameters are required to make each random draw from this distribution: the number of adults that will be present in the next generation (K), the number of mutants in the juvenile pool, the total number of juveniles, and the bias. If the bias parameter is set to 1.0, the model simulates the dynamics of a neutral mutation. Values of this parameter greater than 1.0 are used to simulate the dynamics of beneficial mutations, whereas values less than 1.0 are used to simulate the dynamics of deleterious mutations. For example, an individual with a bias of 1.1 is 10% more likely to survive to the next generation than an individual with a bias of 1.0.

Initially, we perform sets of simulations varying the fitness effect of the mutant but keeping all other parameters constant. We use biases of 1.050, 1.200, and 1.500 to simulate the dynamics of mildly, moderately, and highly advantageous mutations, respectively, and 0.952, 0.833, and 0.667 for mildly, moderately, and highly deleterious mutations, respectively. Note that the deleterious values are the reciprocals of the advantageous values to allow direct comparison. We also use a neutral mutation (bias = 1.000). In all these simulations, we set $K = 10$ and $r = 1.8$ and the probability that an individual disperses (m) is set to 0.1. We record descriptive statistics from as many simulations as it takes to obtain 500 "successful" simulations for each fitness effect. We define a successful simulation as one in which the mutant allele is still present in the gene pool 500 generations after its initial arrival in the population.

To establish how robust the results are to key assumptions of the model, we run 2 sets of additional simulations. In the first of these, we establish the role of key demographic parameters by running simulations in which we vary K, r, and m. In the second set, we seek to establish how important the nature of the mutation effect is for the dynamics. In all the other simulations conducted in this paper, mutations result in an individual having an altered competitive ability as a juvenile. Here, we modify the model such that individuals carrying the mutated gene have a different r (but have the same juvenile competitive ability; the bias in the Wallenius' noncentral-hypergeometric distribution is set to 1.0).

The descriptive statistics recorded include the frequency and spatial distribution of the mutation at different time points after colonization. We follow Klopfstein et al. (2006) in using the centroid of the mutant's spatial distribution to determine whether surfing has occurred, and we record the proportion of successful simulations in which the centroid of the mutant spatial distribution moved more than 30 demes in the direction of expansion. Additionally, we record 2 other statistics that we believe help to describe the spatial dynamics by identifying how frequently mutants are surfing anywhere along the leading edge of the range expansion and how often they are present within the most advanced deme of the expansion. First, we record the proportion of successful simulations in which the mutant is present within the deme (or demes) that is furthest right on the entire lattice (i.e., the occupied patch with the highest x coordinate). Second, we record the proportion of successful simulations in which the mutant is present within at least

Table 1
Summary Statistics for the Surfing Dynamics of Mutations with Different Fitness Effects

	Survival Probability	Probability of Surfing (Centroid Definition)	Probability of Mutation Being Somewhere along the Front	Probability of Mutation Being Right at the Front
Neutral (bias = 1.00)	0.207	0.084	0.068	0.057
		0.408	**0.330**	**0.276**
Deleterious (bias = 0.952)	0.098	0.084	0.066	0.059
		0.852	**0.672**	**0.598**
Deleterious (bias = 0.833)	0.066	0.066	0.060	0.054
		1.000	**0.910**	**0.818**
Deleterious (bias = 0.667)	0.051	0.051	0.049	0.043
		1.000	**0.968**	**0.850**
Beneficial (bias = 1.05)	0.362	0.081	0.074	0.067
		0.224	**0.204**	**0.186**
Beneficial (bias = 1.20)	0.527	0.100	0.085	0.075
		0.190	**0.162**	**0.142**
Beneficial (bias = 1.50)	0.639	0.133	0.106	0.100
		0.208	**0.166**	**0.156**

NOTE.—Two values are presented for each of the 3 surfing criteria. Above we report the proportion of all mutants to surf, whereas below (in bold) we show the proportion of surviving mutants to have surfed.

one of the rightmost demes. That is, for each y, we look at the occupied patch with the highest x coordinate and see if a mutant is present. We record the proportion of simulations in which a mutant is present within at least one of these patches. Additionally, we plot AFCs to investigate how the spatial distribution of a mutant is likely to differ depending upon its fitness effect.

Results

Surfing Behavior of Nonneutral Mutations

The likelihood that a new mutation survives to a specified time point will clearly depend upon its fitness effect (see table 1). The fitter the mutant relative to individuals with the initial allele, the greater its survival probability. For example, we find that beneficial mutations with a fitness of 1.5 times that of the initial allele survive with a probability of 0.639, whereas mutants with a fitness of only 0.667 that of the initial allele survive with a probability of just 0.051. As a proportion of all mutations (i.e., those that survive and those that perish), beneficial mutations are more likely to surf than deleterious mutations (table 1). However, if we consider the spatial distribution of only the surviving mutants (i.e., look only at the successful simulations), we find that the lower the fitness of the mutant the more likely it is to have surfed (table 1). In this paper, we are, like Edmonds et al. (2004) and Klopfstein et al. (2006), most interested in the spatial distribution of the mutants that survive a range expansion and accordingly we present most of our results in terms of the proportion of "surviving mutants" that have "surfed."

Examples of the spatial and frequency distributions of surviving beneficial, neutral, and deleterious mutations are shown in figure 1 for different times from the onset of colonization. These examples have been selected as they illustrate the typical results noted from observations of many runs of the simulations. These results highlight some important differences in the spatial distribution of surviving mutations depending upon their effect. Deleterious mutations that survive to the end of the simulation have distributions that are centered toward the expanding front, whereas most surviving beneficial mutations have spatial distributions centered close to their origin. This is as a result of each of the 3 types of mutations exhibiting unequal likelihoods of surfing on the expanding range (see table 1). For example, whereas 0.408 surviving neutral mutations had surfed (according to the centroid definition), only 0.190 of the moderately beneficial (bias = 1.20) mutations to survive had surfed. In contrast, all 500 of the surviving moderately deleterious mutations had surfed to some extent (according to the centroid definition), and of these, 0.818 were still surfing at the extreme front of the expanding population.

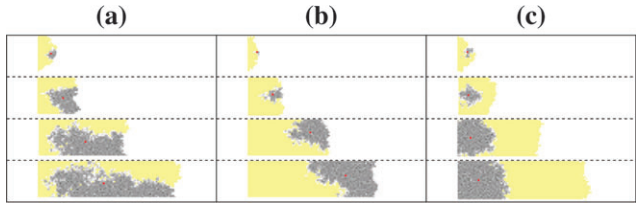

FIG. 1.—Typical spatial distributions of neutral (*a*), deleterious (*b*), and beneficial (*c*) mutations 75, 150, 300, and 450 time steps after the start of the invasion. The geographic origin of the expansion is at point (0, 12) on the left hand side of the grid, whereas the new mutations appear in the deme at position (10,12). Yellow shading shows regions occupied by only the initial genotype, whereas gray indicates regions where the mutant is present. The red point highlights the centroid location of the mutants. These results are gained using $K = 10$ and $r = 1.8$ and in (*a*) the fitness = 1.00, in (*b*) fitness = 0.833, and in (*c*) fitness = 1.200.

Deleterious mutations only survive for extended periods of time if they surf (fig. 2). Most deleterious mutations do not remain at the leading edge of the expansion for long and very rapidly become extinct. However, some mutants become established "surfers" and are present across a broad section of the leading edge. These mutants can continue to

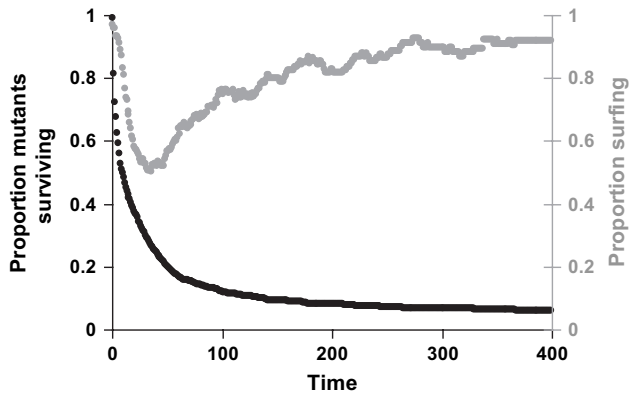

FIG. 2.—Mutation surfing leads to a decelerating rate of mutation extinction. The figure illustrates the proportion of deleterious mutations surviving through time (black line) and the proportion of these surviving mutations that are present somewhere along the leading edge of the range expansion (gray line). Here, fitness = 0.833 and other simulation conditions are as in figure 1.

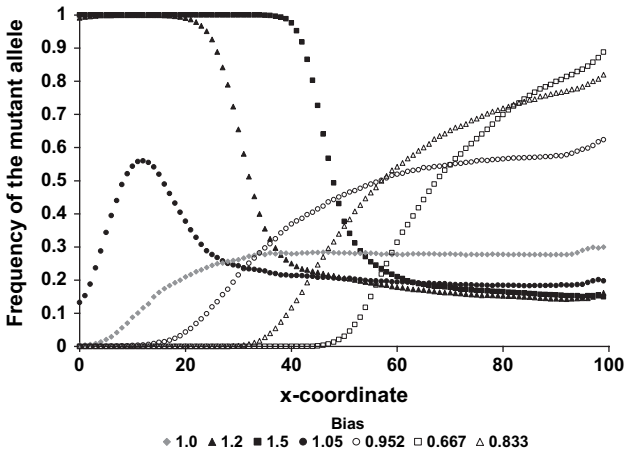

FIG. 3.—Profiles of mean mutant frequencies along the x axis. These results depict the mean frequencies for 500 successful simulations for each of the 7 types of mutations simulated. Both the direction and the gradient of the clines depend upon the nature of the mutation. Again, $K = 10$ and $r = 1.8$ and other simulation details are as in figure 1.

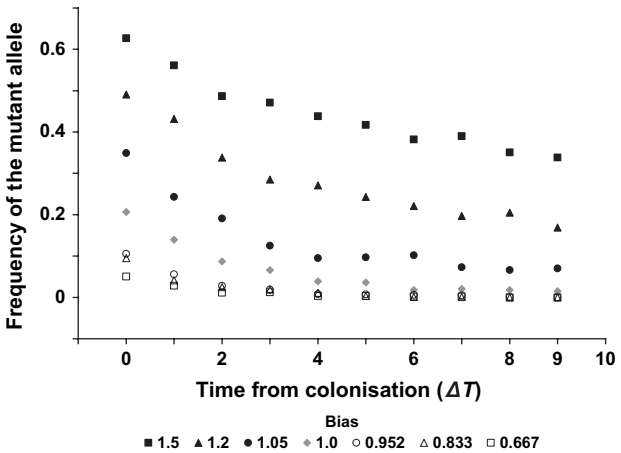

FIG. 4.—The age of the deme (ΔT) in which a new mutant first appears determines the probability that it survives for all types of mutation. We report the proportion of simulations for which the mutant is still observed after 500 generations as a function of the age at which the mutation occurred for the 7 mutation types. Simulation conditions are exactly as in figures 1 and 3.

surf and therefore survive for substantial periods of time. This process leads to a decelerating rate of mutant extinction through time (fig. 2).

Mutant AFCs

AFCs are observed for "surviving" neutral, beneficial, and deleterious mutations, although the nature of these clines is highly dependent upon the nature of the mutation (fig. 3). The mean frequency of a mildly beneficial mutation following range expansion is highest close to its origin (12 on the x axis), and there are relatively steep declines in frequency on either side of this peak. For neutral and deleterious mutations, there are positive clines in the direction of the range expansion, with steeper clines observed for the more deleterious mutations. For beneficial mutations, the clines are in the opposite direction, with the highest mutant frequencies found a considerable distance from the front. For biases 1.5 and 1.2, the mutant has reached fixation in all the simulations (mean mutant frequency = 1.0) for $x < 36$ and $x < 18$, respectively. It is interesting to note that the clines for beneficial mutations are much steeper than the clines for their equivalent strength deleterious mutations.

Proximity to the Wave Front

The number of generations elapsed between the initial colonization of a deme and the occurrence of the mutation (referred to as age of deme or ΔT in the following) is a key factor in determining the probability that the mutation survives (fig. 4). Klopfstein et al. (2006) illustrated that this was true for neutral mutations (see their fig. 4), and our results show it to be the case for both deleterious and beneficial mutations. However, steeper declines in survival probability are observed for deleterious mutations. For example, although a change in ΔT from 0 to 2 results in a 22% reduction in the proportion of surviving beneficial mutants (bias = 1.50), the same change in ΔT results in a 75% reduction in the proportion of surviving deleterious mutants (bias = 0.667). Thus, the age of the deme in which a mutant first appeared is especially important for deleterious mutations and in particular for those with strong disadvantages.

The Roles of r, K, and m

Qualitatively, we obtain very similar results over a range of r, K, and m. As r is increased, there is a greater probability that the mutant will survive and this is true regardless of the fitness effect of that mutant (fig. 5*a*). For deleterious mutants, higher r values result in a higher probability of surviving, whereas for neutral and beneficial mutants, the pattern is reversed with a lower frequency of surfing observed when r is higher (fig. 5*b*). The survival of mutants is greater when K is higher, and the shape of the relationship between fitness effect and survival probability is largely insensitive to varying K (fig. 5*c*). For surviving beneficial mutations, there is a greater surfing probability when K is small, whereas surviving deleterious mutations are more likely to have surfed when K is large. In a set of simulations varying the rate of dispersal and using in turn $m = 0.1$, $m = 0.2$, and $m = 0.3$, we find that the results are qualitatively very similar. The relationships between the fitness effect of a mutant and survival and surfing probabilities are consistent across dispersal rates. However, a closer look at the results does reveal some trends (see table 2). The probability of a deleterious mutation surviving is lower when m is higher; for example, for a mutation with fitness = 0.667, 0.050 of all mutants survive with $m = 0.1$, whereas 0.040 survive with $m = 0.3$. However, for beneficial mutations, there is a slightly elevated probability of survival for higher rates of dispersal. Surviving deleterious mutations are more likely to have surfed when dispersal rate is high, whereas surviving beneficial mutations are more likely to have surfed when m is low.

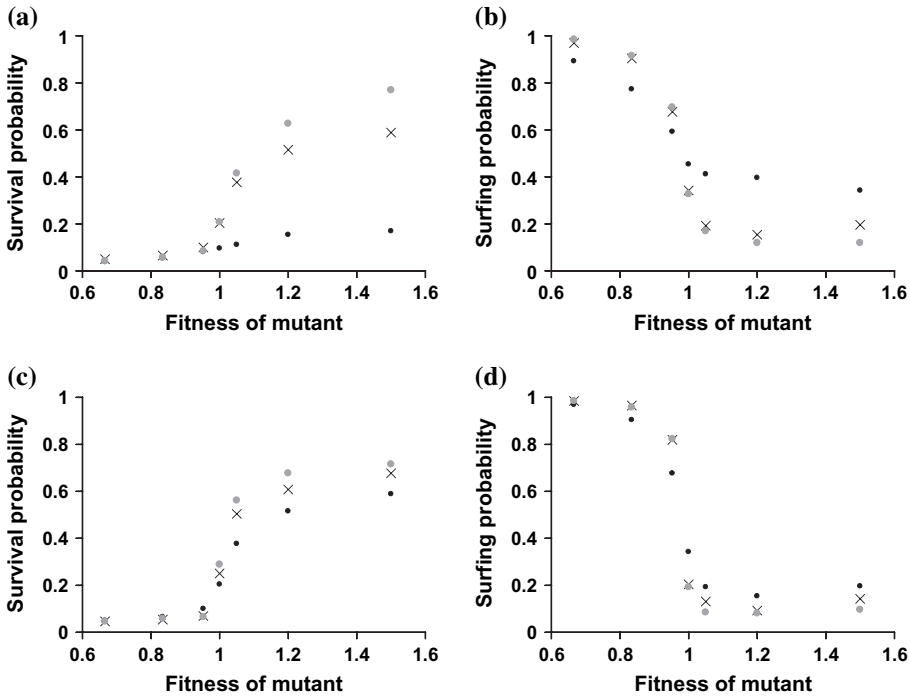

FIG. 5.—The effects of varying K and r. The probability that a mutant survives depends upon the fitness effect of the mutant and also on the values of r (*a*) and K (*c*). The probability that a "surviving mutant" surfs on the wave front also varies according to the fitness effect and the values of r (*b*) and K (*d*). In (*a*) and (*b*), the black points show results for $r = 1.2$, crosses $r = 1.8$ and gray points $r = 2.5$. In (*c*) and (*d*), the black points show results for $K = 10$, crosses $K = 50$ and gray points $K = 100$. In drawing (*b*) and (*d*), we use the third surfing definition (that the mutant has to be right on the front—see table 1). All other parameters are as in figure 1.

The Fate of Mutations that Alter r

When we consider the fate of mutants that have an altered r (mean number of offspring produced), we observe qualitatively similar results to those for mutations that impact on juvenile competitive ability (fig. 6). We should highlight here that whereas in other simulations, the rate of range expansion is constant regardless of the fitness of the individuals (fitness only altered relative competitive ability), in these simulations, the rate of expansion will increase should a mutant with higher r survive and surf. Similarly, it will decrease if a mutant with lower r surfs at the front. Unsurprisingly, mutants with lower r than the initial population are much less likely to survive than those with higher r (fig. 6*a*). However, those mutants that have mild deleterious impact on r have a nonnegligible probability of surviving. For example, when the initial population all has $r = 1.80$, mutants with $r = 1.75$ and $r = 1.70$ survive 8.0% and 3.8% of the time, respectively. When just the surviving mutations are considered, those that are deleterious are more likely to have surfed than those that are neutral or beneficial (fig. 6*b*). However, the relationship is not quite as straightforward as for the mutations impacting juvenile competitive ability; surviving beneficial mutants are considerably more likely to have surfed when they have increased r instead of increased juvenile competitive ability.

Landscape Structure and Surfing Dynamics

Most landscapes are heterogeneous, with some areas more suitable for colonization than others. This heterogeneity may have important implications for the surfing behavior and the spatial patterns that result. When an invading population has to pass through a narrower region of suitable habitat, the precise location at which the mutation first appears becomes extremely important (fig. 7). Mutations that appear very close to the geographical bottleneck and especially those that appear just beyond it are the most likely to survive. Additionally, this added landscape complexity means that mutations that arise in some locations have extremely reduced probabilities of surfing and/or survival. For beneficial mutations, the main effect is seen in the different probabilities of them surfing through the bottleneck to high densities toward the right hand side of the lattice. For deleterious mutations, the survival probability itself is highly dependent upon the location at which the mutation occurs. Qualitatively, very similar results are obtained when reflective, as opposed to absorbing, boundaries

Table 2
The Rate of Dispersal Influences the Likely Fate of a Mutation

	Dispersal		
Mutant fitness	0.1	0.2	0.3
0.667	0.050 (0.970)	0.044 (0.980)	0.040 (0.990)
1.000	0.206 (0.344)	0.183 (0.332)	0.172 (0.302)
1.500	0.587 (0.196)	0.617 (0.122)	0.603 (0.120)

NOTE.—Here, we report 1) the probabilities that mutations survive to $T = 500$ and in brackets; 2) the proportion of those surviving mutants to have surfed on the leading edge of the range expansion. All other simulation conditions are the same as used in figure 1.

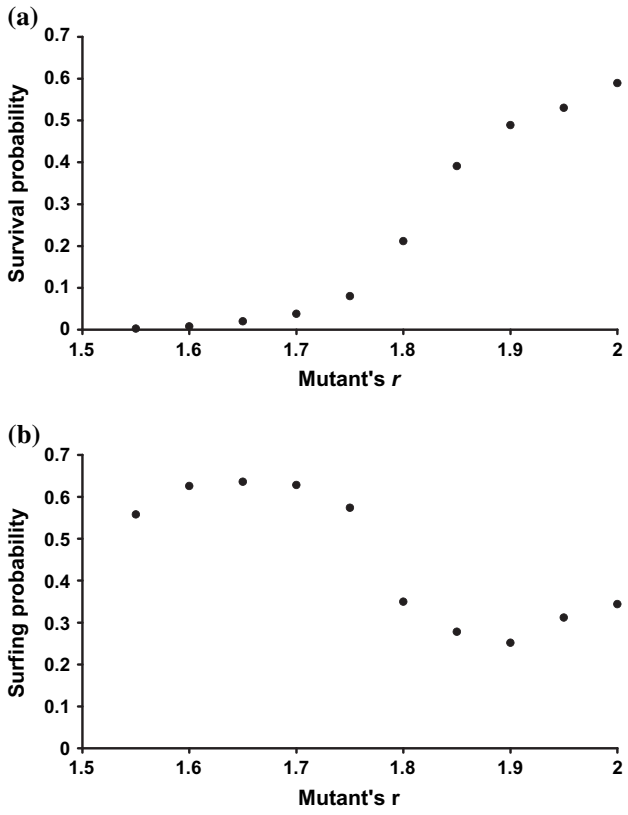

FIG. 6.—Mutations that reduce r can survive and surf. Qualitatively similar results are obtained when the mutation alters r rather than relative competitive ability. In (*a*), we report the probability that a single mutant survives to $T = 500$. For each value of r, we repeatedly ran the simulation until we had obtained 500 surviving mutants. In (*b*), we show the proportion of the surviving mutants that are surfing on the extreme edge of the wave (third surfing definition) at $T = 500$. In these simulations, mutations have no effect on relative competitive ability. The initial population, all have $r = 1.8$. All other simulation conditions are as described previously.

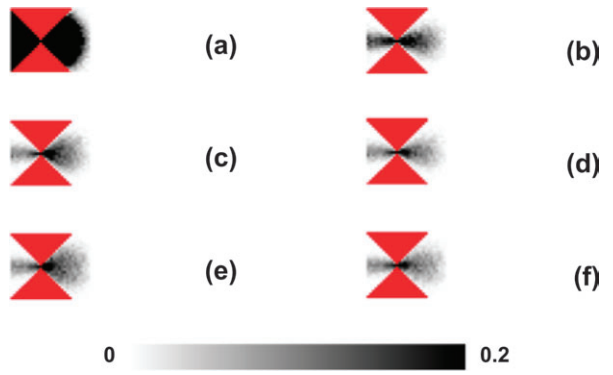

FIG. 7.—The impact of a bottleneck on the surfing behavior. The red cells indicate the region specified as unsuitable. Any individuals dispersing into this area die. These figures show how the bottleneck can introduce considerable spatial variability in the probabilities for survival (*a*), (*c*), and (*e*) and surfing (*b*), (*d*), and (*f*). (*a*) and (*b*) show results for a beneficial mutation with absorbing boundaries (fitness = 1.200), whereas (*c*) and (*d*) show equivalent results for a deleterious mutation (fitness = 0.833). (*e*) and (*f*) show the results for a deleterious mutation with reflective boundaries. Mutations that occur very close to the bottleneck and especially those that occur just beyond the bottleneck are the most likely to survive and surf. Darker gray shades indicate higher probabilities as shown by the legend—black indicates probabilities >0.20. For each coordinate (x, y) on the landscape between $9 < x < 40$, we ran the model 500 times introducing the mutant at (x, y) when $\Delta T = 0$.

are assumed. There are subtle quantitative differences (fig. 7) with both a greater survival probability and a greater probability of surfing for those mutants that occur in close proximity to the habitat edge.

Range Expansion from the South East of Europe

To illustrate the spread of mutant genes on a more spatially realistic landscape, we follow Klopfstein et al. (2006) by simulating the spread of a population into Europe from the near east. We run exactly the same model used for the regular grids but now on the European land surface, allowing us to examine how key features such as water bodies and uninhabitable mountain ranges influence the surfing phenomenon and the spatial genetic patterns that are likely to emerge. These effects are likely to be important both for patterns of genetic diversity observed in European humans today as a result of their past range expansion, but are equally likely to be important for invasive species spreading through Europe, and species undergoing range shifts as a result of climate change. For these simulations, we impose a grid over the European land surface with each grid cell roughly 7 km by 7 km. In most of the simulations, we assume that all land is available for colonization unless it is above 1,000 m (cf., Klopfstein et al. 2006 who assumed that all land was available). On average, surviving beneficial mutations are found most frequently toward their origin and least frequently toward the expanding range margin, whereas the converse is true for deleterious mutations (fig. 8). This pattern is the same as found on the regular lattices. The patterns observed for neutral and beneficial mutations are suggestive of a somewhat bimodal distribution with peaks in mean mutant frequency close to the origin of the mutations (higher peak) and also close to the edge of the range expansion (lower peak). This is particularly clear in figure 8*a* (neutral mutation) with a light gray area present just inside the boundary of the range margin. Because this is a stochastic model, different simulations result in range expansions that travel somewhat different distances, and this explains why in some of these figures there is a very distinct black or white band right at the expanding margin: a single invasion that happened to be either mutant/nonmutant that spreads slightly further than the rest will determine the mean mutant frequency in the extra areas that it reaches.

While observing many of these simulated range expansions across Europe, it rapidly becomes obvious that the mountains can play an important role, both in determining the final spatial patterns and in determining the likelihood that a mutation arising in a particular location survives. In figure 8*b* (bias = 0.833), the role of the Carpathian mountains is quite clear, with the wild-type mutations displacing the surfing deleterious mutations less rapidly in areas where the spread is impeded by the mountain range. In some cases, the presence of a mountain range can make a dramatic difference to the mean mutant frequencies that

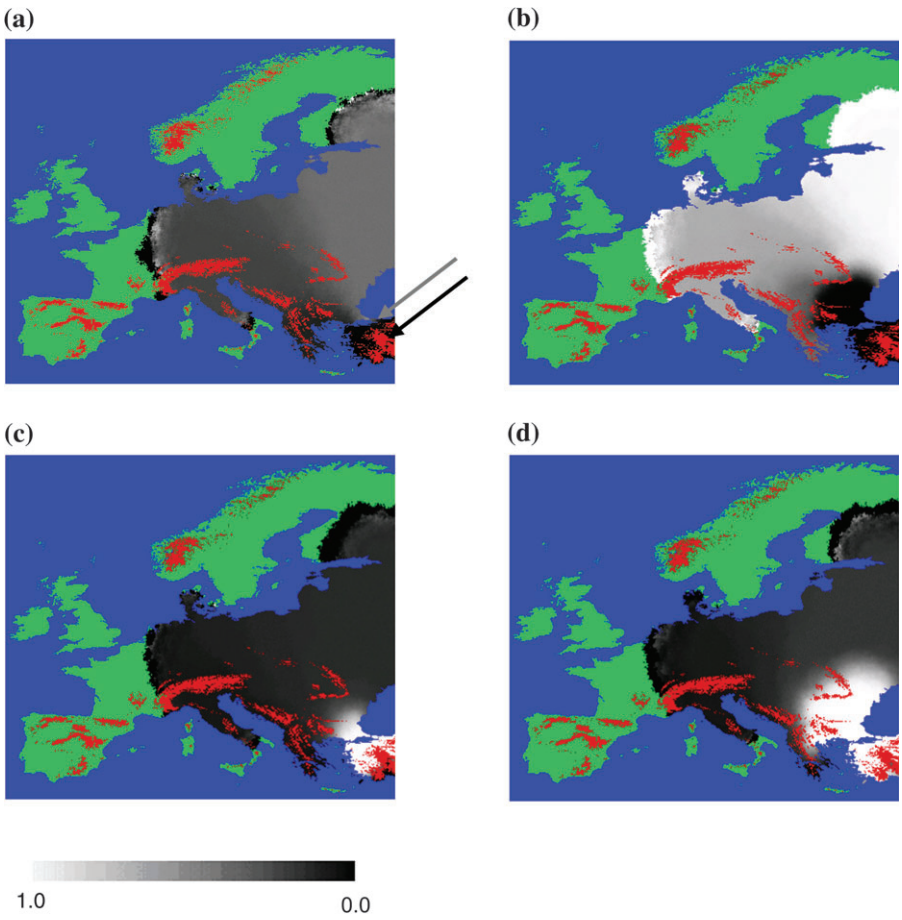

FIG. 8.—Mutation surfing in simulations of the colonization of Europe. These show the mean frequency of 100 surviving mutants (lighter is more frequent) in which colonization started in the far Southeast (shown by the black arrow in *a*) and a mutation occurred just to the east of Istanbul (shown by the gray arrow in *a*). In (*a*), the spatial distribution of a neutral mutation is shown (fitness = 1.00), in (*b*) a deleterious mutation (0.833), in (*c*) a moderate beneficial mutation (fitness = 1.200), and in (*d*) a strongly beneficial mutation (fitness = 1.500). They confirm that the same surfing effects happen on a real landscape and highlight the need to take landscape structure into account. These results were generated assuming that land over 1,000 m unavailable for colonization (shown in red). The dispersal function was also modified slightly from the regular lattice model: in these simulations, most dispersing individuals move to one of the nearest neighbor cells but 0.10 move up to 3 cells from the natal patch.

are observed: in figure 9, we present a comparison between 2 sets of simulations in which the only difference is the status of land above 1,000 m. Where the high land is unsuitable (fig. 9*a*), this deleterious mutation obtains much higher mean frequency over a wider spatial extent than where it is suitable (fig. 9*b*). This example is quite typical and certainly not extreme. Landscape features that promote bottlenecks can create even greater differences, but linear features, such as the Alps in this example, can also create quite substantial effects. When we run these simulations using reflective, rather than absorbing boundary conditions, we observe qualitatively similar differences between the dynamics of mutants with different effects. With reflective boundary conditions, the range expansion proceeds more rapidly as does the spread of the mutants, whatever their effect. We observe that the boundary conditions are most important when a mutation occurs close to a boundary in the proximity of a geographical bottleneck, as under these conditions the mutant is much more likely to survive and will have a much higher initial rate of spread when the boundary is reflective.

Discussion

Our results emphasize that the mutation surfing phenomenon can be important in determining the spatial distributions of beneficial and deleterious mutations in populations during and after range expansions. Previous work has described the surfing phenomenon for neutral mutations (Edmonds et al. 2004; Klopfstein et al. 2006), but this is the first study to consider the potential role of surfing dynamics for mutations with fitness effects. Our results indicate that deleterious mutations arising at an expanding front have a far higher chance of persisting than they would in a stationary population. Most deleterious mutations become extinct very rapidly following their introduction. However, even these competitively inferior mutants may begin to surf the expanding wave such that they reach local fixation in the population. When they occur right on the range margin, they may, by chance, become the colonists in the next available patch of habitat, and a succession of these founder effects can rapidly amplify the frequency of mutations even with quite considerable fitness

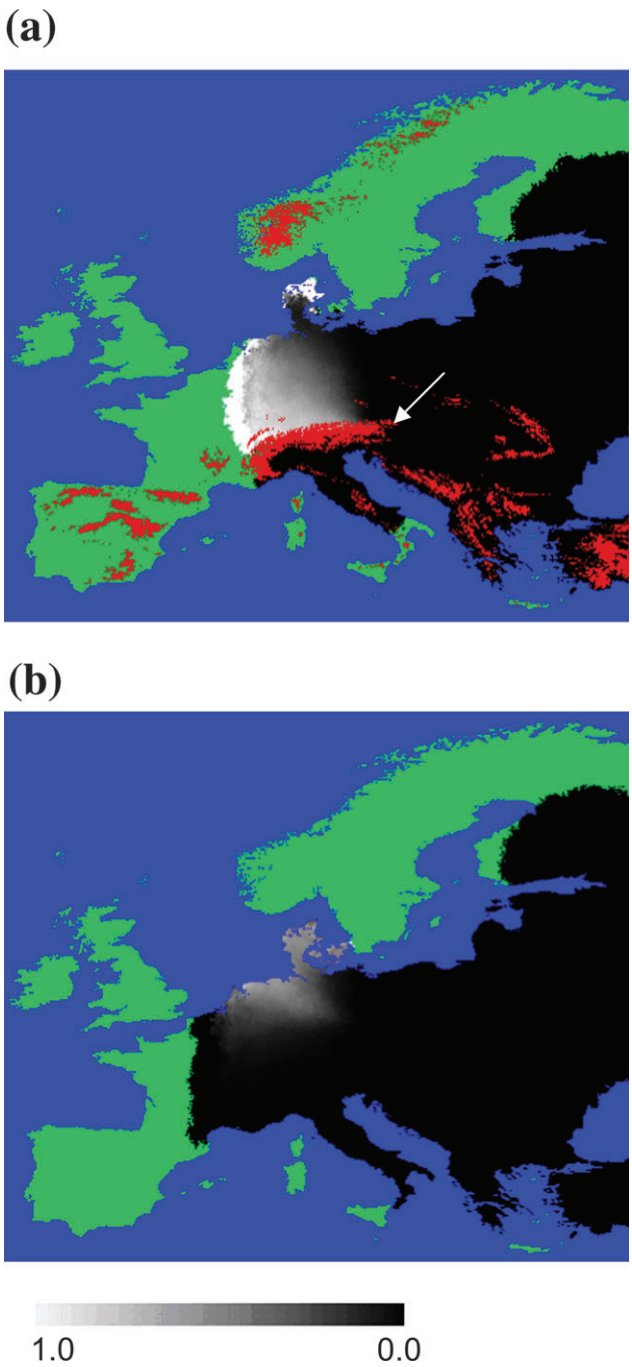

FIG. 9—Landscape structure can play an important role in determining spatial patterns in surviving mutation frequency. This figure depicts the mean frequencies for a deleterious mutation (fitness = 0.833) in simulations, where land above 1,000 m is assumed to be unsuitable (*a*) and suitable (*b*). In both cases, the mutation arises at the location indicated by the white arrow in (*a*). All other details are as described for figure 8.

disadvantages. In some simulations, deleterious mutations become "established surfers" occurring across a broad section of the expanding front, and in these circumstances the mutation can continue to surf and survive for long periods. The location at which a deleterious mutation occurs relative to the range front is even more important than Klopfstein et al. (2006) found it to be for neutral mutations. If time from colonization (ΔT) is very small, then the mutant is likely to arise in a patch with very few, or no, competitors. In this environment, it performs just as well as a nonmutant, and it will be as likely as a nonmutant to provide a colonist for the next empty cell. However, as ΔT increases, there will on average be more individuals in the patch and the patch is less likely to still be right on the wave front. This immediately makes it less likely that the mutant will be the individual that provides a colonist to the next vacant patch (and less likely that when it does provide a colonist, it is the only one). Additionally, competition becomes important as the local population reaches equilibrium density, and this means selection begins to act against the mutant. It is this second effect that makes ΔT even more important for deleterious mutants than for neutral mutants. If ΔT is higher than at their origin and in patches to which they initially expand, deleterious mutants are more likely to suffer from selection that favors the nonmutants. Conversely, for beneficial mutations, selection effects favoring the mutant over the established population tend to somewhat flatten the relationship between ΔT and the probability of a mutation either surviving or surfing.

Qualitatively similar results were obtained for a range of K, r, and m. However, all 3 parameters do have some effect on the observed results (fig. 5 and table 2). Increasing r and/or K inevitably lowers the probability that a new mutant goes extinct due to stochasticity and thus mutations have a higher probability of survival when K and r are higher. Surfing of deleterious mutations is somewhat more likely when r is higher, and this is a result of the increased rate at which the population at the range margin expands into empty space. A deleterious mutant that initially starts to spread at the expanding front has a higher chance of keeping ahead of the fitter individuals if r is higher. Dispersal probability has relatively little influence on the outcome. However, it does lead to subtle changes that differ according to whether the mutant is beneficial or deleterious. Deleterious mutations have a higher probability of survival when dispersal is low. On the contrary, beneficial mutations survive more frequently with higher rates of dispersal. This pattern can be explained as with a lower dispersal probability, the spatial population becomes more viscous and drift becomes more influential, favoring the persistence of deleterious mutations.

We compared the outcome of simulations using mutations that alter juvenile competitive ability with simulations where mutations altered r, the mean number of offspring produced. Even when the effect of a mutation is to reduce r, the mutation sometimes reaches high frequency and spatial extent when it occurs right at the expanding margin. However, this only occurs for mutations that result in a relatively small reduction in r (fig. 6). Whereas mutations impacting competitive ability never alter the rate of spread of a population into an empty region, mutations influencing r will modify the rate of spread if they become abundant at the front. A mutation with reduced r will only be able to persist at an expanding front through the surfing effect when its rate of spread into the empty region is greater than that at which the nonmutant population displaces the mutant from areas that it occupies. Our results

suggest that the region of parameter space within which this occurs may be relatively small. In the model presented here, we have considered the dynamics of just a single mutant. An interesting avenue for future work will be to explore whether the gradual accumulation of numerous deleterious mutations, each having a small effect on r, can potentially result in a decelerating rate of range expansion.

In this paper, we have focused on the survival and distribution of mutations 500 generations after their initial introduction at the front. Klopfstein et al. (2006) demonstrated that survival and surfing probabilities vary as time since arising increases. The survival probability of a neutral mutant can be much lower after 1,500 generations than after 500 (see table 1, Klopfstein et al. 2006), and this trend is most marked when K is high. The probability of a surviving neutral mutant having surfed increases the longer the simulation has run. We anticipate that deleterious mutations will experience a steeper decline in survival over time than advantageous mutations but that a greater frequency of deleterious mutations to have survived for longer will have surfed.

Klopfstein et al. (2006) suggested that range expansion might speed up evolutionary adaptation. Although it certainly seems to be the case that the surfing phenomenon can lead to considerable spatial differentiation in genotypes whether they are neutral or not, we do not find any evidence that beneficial mutations are concentrated by a range expansion. In fact, our model results indicate that when we observe recently expanded populations, we might expect to see the opposite pattern, with a concentration of the recent beneficial mutations toward the core of the range, and a higher than average frequency of deleterious mutations toward the recently expanded margins. Rather than an accelerated rate of evolution during a range expansion, we might instead expect an accumulation of mutational load.

Results from our European simulations and our simulations with the simple bottleneck landscape highlight the importance of landscape structure for this surfing phenomenon. Complex landscape structures result in great spatial heterogeneity in the probabilities that different locations will be the source of successful mutants. Although bottleneck features of a landscape perhaps provide the most potent effect, edge effects generated by linear features such as rivers or mountain ranges can also have substantial effects. A much fuller understanding of these effects is required before we are able to make proper inferences from existing genetic variation within populations. Additionally, incorporating more ecological details is likely to be important: for example, in the European simulations, we have assumed that all cells above 1,000 m altitude are unavailable, whereas those below 1,000 m are equally suitable. In reality, for most species, the pattern will be more complex with a broad and continuous range of habitat suitability and individuals making emigration and settlement decisions based upon a combination of habitat quality and local patch density. Incorporating these added layers of complexity would potentially increase the frequency of bottlenecks as an initial wave of range expansion is likely to be concentrated through often narrow strips of the best quality habitat (e.g., fertile river valley) before density dependence results in individuals gradually colonizing lower quality habitats (e.g., steeper slopes).

Many ecologists, presented with a pattern showing less competitive individuals toward an expanding range margin and more competitive individuals toward the saturated core population, would immediately think of the dispersal–competition trade-off (e.g., Higgins and Cain 2002; Kisdi and Geritz 2003). They would suggest that the pattern was consistent with selection favoring dispersal over competitive ability at the margins and the opposite at the core. However, the pattern observed in the model does not arise from this effect at all (there is no variation in dispersal ability between individuals in this model). This suggests that in expanding populations, care is required when invoking a competition–colonization trade-off as observing the pattern will not necessarily be confirmation of its existence.

As in Edmonds et al. (2004) and Klopfstein et al. (2006), we have investigated a model in which only a single mutation occurs during a simulation. This approach is ideal to illuminate the types of dynamics that the surfing effect can lead to. However, an important next step will be to develop models able to incorporate many mutations occurring stochastically throughout the simulation and having fitness effects drawn from realistic distributions. A further area for extension is the incorporation of more complex dispersal dynamics. We used a very simple stratified dispersal function in our model of Europe with the intention of increasing realism, but to date, there has been no exploration of the role that the shape of a dispersal kernel or form of density-dependent dispersal might play in driving the surfing dynamics. Previous work has highlighted the need to consider different shaped dispersal kernels in models investigating the genetics of colonization (e.g., Ibrahim et al. 1996), and there is considerable scope to incorporate some of these ideas in work looking at surfing dynamics, particularly for nonneutral mutants. We have run some simulations to compare the behavior of models assuming absorbing versus reflective boundary conditions. Qualitatively, we find that differences in the behavior of mutations with different effects are similar regardless of the boundary conditions. However, the absolute rate of spread of the entire range and the mutant cloud will be highly dependent upon the boundary conditions particularly when the density of boundaries on the landscape is high. How individuals move in relation to boundaries between different habitat types is an important issue (e.g., Schtickzelle and Baguette 2003). Future work could also usefully consider the behavior of individuals moving through habitats of different suitability and might move toward incorporating more sophisticated models of movement, for example, using correlated random walks (e.g., Bovet and Benhamou 1988) or foray search behavior (Conradt et al. 2003).

There has been no consideration of the potential for dispersal evolution to influence the probability of mutation surfing. Certainly, interindividual variation in dispersal rate will lead to different propensities for mutations to surf depending upon the individual within which they occur, and this might lead to interesting effects particularly when dispersal itself is likely to evolve during a range expansion. Previous work has already demonstrated that the

joint evolution of dispersal and other life history traits can result in different evolutionary outcomes compared with not allowing dispersal to coevolve (e.g., Johst et al. 1999; Dytham and Travis 2006), and a model developed to investigate the interplay between dispersal evolution and mutation surfing might provide considerable new insights. There is great scope for further work in this area, and developments offer considerable promise for a wide range of fields including human ecology, invasion biology, conserving species during climate change, and the evolutionary dynamics of an epidemic.

Acknowledgments

We thank Flemming Skov and Jens-Christian Flemming for providing the European land surface data. This work has been partly funded by both A Long-Term Biodiversity, Ecosystem and Awareness Research Network and NERC.

Literature Cited

Araujo MB, Pearson RG, Thuiller W, Erhard M. 2005. Validation of species-climate impact models under climate change. Glob Change Biol. 11:1504–1513.

Araujo MB, Whittaker RJ, Ladle RJ, Erhard M. 2005. Reducing uncertainty in projections of extinction risk from climate change. Glob Ecol Biogeogr. 14:529–538.

Austerlitz F, Garnier-Géré PH. 2003. Modelling the impact of colonisation on genetic diversity and differentiation of forest trees: interaction of life cycle, pollen flow and seed long-distance dispersal. Heredity. 90:282–290.

Bossdorf O, Auge H, Lafuma L, Rogers WE, Siemann E, Prati D. 2005. Phenotypic and genetic differentiation between native and introduced plant populations. Oecologia. 144:1–11.

Bovet P, Benhamou S. 1988. Spatial analysis of animals' movements using a correlated random walk model. J Theor Biol. 131:419–433.

Butin E, Porter AH, Elkinton J. 2005. Adaptation during biological invasions and the case of *Adelges tsugae*. Evol Ecol Res. 7:887–900.

Conradt L, Zollner PA, Roper TJ, Frank K, Thomas CD. 2003. Foray search: an effective systematic dispersal strategy in fragmented landscapes. Am Nat. 161:905–915.

Daehler CC. 1998. Variation in self-fertility and the reproductive advantage of self-fertility for an invading plant (*Spartina alterniflora*). Evol Ecol. 12:553–568.

Dytham C, Travis JMJ. 2006. Evolving dispersal and age of death. Oikos. 113:530–538.

Edmonds CA, Lillie AS, Cavalli-Sforza LL. 2004. Mutations arising in the wave front of an expanding population. Proc Natl Acad Sci USA. 101:975–979.

Ellstrand NC, Schierenbeck KA. 2000. Hybridization as a stimulus for the evolution of invasiveness in plants. Proc Natl Acad Sci USA. 97:7043–7050.

García-Ramos G, Rodríguez D. 2002. Evolutionary speed of species invasions. Evolution. 56:661–668.

Garcia-Rossi D, Rank N, Strong DR. 2003. Potential for self-defeating biological control? Variation in herbivore vulnerability among invasive *Spartina* genotypes. Ecol Appl. 13:1640–1649.

Hammershøj M, Travis JMJ, Stephenson CM. 2006. Incorporating evolutionary processes into a spatially-explicit model: exploring the consequences of mink-farm closures in Denmark. Ecography. 29:465–476.

Hänfling B, Kollmann J. 2002. An evolutionary perspective of biological invasions. Trends Ecol Evol. 17: 545–546.

Hastings A, Cuddington K, Davies KF, et al. (14 co-authors). 2005. The spatial spread of invasions: new developments in theory and evidence. Ecol Lett. 8:91–101.

Higgins SI, Cain ML. 2002. Spatially realistic plant metapopulation models and the colonization-competition trade-off. J Ecol. 90:616–626.

Ibrahim KM, Nichols RA, Hewitt GM. 1996. Spatial patterns of genetic variation generated by different forms of dispersal during range expansion. Heredity. 77:282–291.

Johst K, Doebeli M, Brandl R. 1999. Evolution of complex dynamics in spatially structured populations. Proc R Soc Lond B. 266:1147–1154.

Kisdi E, Geritz SAH. 2003. On the coexistence of perennial plants by the competition-colonization trade-off. Am Nat. 161:350–354.

Klopfstein S, Currat M, Excoffier L. 2006. The fate of mutations surfing on the wave of a range expansion. Mol Biol Evol. 23:482–490.

Lambrinos JG. 2004. How interactions between ecology and evolution influence contemporary invasion dynamics. Ecology. 85:2061–2070.

Le Corre V, Machon N, Petit RJ, Kremer A. 1997. Colonization with long-distance seed dispersal and genetic structure of maternally inherited genes in forest trees: a simulation study. Genet Res Camb. 69:117–125.

Parker IM, Rodriguez J, Loik ME. 2003. An evolutionary approach to understanding the biology of invasions: local adaptation and general-purpose genotypes in the weed *Verbascum thapsus*. Conserv Biol. 17:59–72.

Parmesan C, Yohe G. 2003. A globally coherent fingerprint of climate change impacts across natural systems. Nature. 421:37–42.

Phillips BL, Brown GP, Webb JK, Shine R. 2006. Invasion and the evolution of speed in toads. Nature. 439:803.

Quinn TP, Kinnison MT, Unwin MJ. 2001. Evolution of Chinook salmon (*Oncorhynchus tshawytscha*) populations in New Zealand: pattern, rate and process. Genetica. 112/113:493–513.

Reznick DN, Ghalambor CK. 2001. The population ecology of contemporary adaptations: what empirical studies reveal about conditions that promote adaptive evolution. Genetica. 112/113:183–198.

Schtickzelle N, Baguette M. 2003. Behavioural responses to habitat patch boundaries restrict dispersal and generate emigration-patch area relationships in fragmented landscapes. J Anim Ecol. 72:533–545.

Sharma GP, Singh JS, Raghubanshi AS. 2005. Plant invasions: emerging trends and future implications. Curr Sci. 88:726–734.

Simmons AD, Thomas CD. 2004. Changes in dispersal during species' range expansions. Am Nat. 164:378–395.

Thomas CD, Cameron A, Green RE, Bakkenes M, et al. (19 co-authors). 2004. Extinction risk from climate change. Nature. 427:145–148.

Travis JMJ, Dytham C. 2002. Dispersal evolution during invasions. Evol Ecol Res. 4:1119–1129.

Wallenius KT. 1963. Biased sampling: the non-central hypergeometric probability distribution [dissertation]. [Stanford (CA)]: Stanford University.

Lisa Matisoo-Smith, Associate Editor

Accepted July 31, 2007