

# Evolutionary rescue in novel environments: towards improving predictability

Michael Barfield and Robert D. Holt

*Department of Biology, University of Florida, Gainesville, Florida, USA*

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## ABSTRACT

**Background:** Populations are often subject to changes in their environments (either locally or due to movement of a population), which, if large enough, require them to adapt in order to persist. This is ‘evolutionary rescue’.

**Questions:** What factors affect the ability of a population to recover after a sudden change in its environment? What can be measured about an initial population, prior to the environmental change, that can improve the predictability of evolutionary rescue?

**Methods:** A deterministic model and simulations of an individual-based model (IBM).

**Results:** Heritability that decreases with decreasing population size could prevent evolutionary rescue in the deterministic model. For the IBM, the probability of rescue decreased with increasing magnitude of the environmental change and with decreasing initial population size. At times, heritability of a trait can increase as selection occurs. Most extinctions occurred shortly after the change. Rescue depended significantly on the genetics of the population at the time of the environmental change, and predictive power about which populations go extinct, or persist, is improved by knowing the mean genotypic value and genetic variance in the initial population. However, there remains considerable uncertainty in such predictions.

**Conclusions:** Persistence after a sudden environmental change was greater in populations with more individuals and more genetic variance at the time of the change, and depended on rapid adaptation soon after the change, without which extinction was likely. Understanding the amount and dynamics of genetic variation can improve predictability of persistence, but there is inescapable randomness in evolution and ecology that will always, we believe, preclude tight predictions.

*Keywords:* environmental change, extinction, heritability, individual-based model.

## INTRODUCTION

If a population persists in a habitat for a long period without immigration from outside, and so does not go extinct in the absence of recolonization, it must be reasonably well adapted to the habitat’s environment, so that at least its births roughly match its deaths (Royama, 1992). If the environment of that habitat changes, or the population (or part of it) moves to a new

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Correspondence: M. Barfield, Department of Biology, 111 Bartram Hall, University of Florida, Gainesville, FL 32611-8525, USA. email: mjb01@ufl.edu

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location with a sharply different environment, the population generally becomes less well adapted, and if this is reflected in sufficiently lower birth or higher death rates, then the population may be unable to sustain itself at any density. In this case, the population must evolve increased absolute fitness in order to persist (assuming again no immigration from outside). Gauging the probability of persistence is the challenge of gauging the potential for evolutionary rescue (Gomulkiewicz and Holt, 1995; Gonzalez *et al.*, 2013; Carlson *et al.*, 2014).

Following an abrupt change in the environment that leads to such maladaptation, it is expected that population size will start to decrease. The rate of decline should depend on the magnitude of difference between the original and novel environments (and at times on density dependence). Concordant with this demographic change, if the environmental change alters selection on phenotypes, and genetic variation is available, selection on the population can cause it to become better adapted to the new conditions. There therefore can be a race between the evolution of greater fitness and the decline in population size. If the population equilibrates at an evolutionary state at which its absolute fitness (mean number of offspring that survive to adulthood per adult) is less than one [or if it simply has no genetic variation relevant to adaptation in the novel environment, the ‘genostasis’ of Bradshaw (1991)], then the race is lost and the population is doomed. But even if the population can potentially evolve to a state at which it has an absolute fitness permitting persistence in the new environment, it is not guaranteed to persist. The reason is that if the population size reaches a low enough level, the population is at risk of extinction because of demographic stochasticity. If the population evolves fast enough to survive, surmounting this risk, it is said to have achieved ‘evolutionary rescue’. Whether evolutionary rescue occurs or not depends upon both demographic and genetic factors (Gomulkiewicz and Houle, 2009).

Of course, not all selective processes enhance overall population fitness; if there is strong frequency dependence, populations can even evolve to drive themselves extinct (Ferriere and Legendre, 2013). And non-selective processes such as genetic drift can further hamper adaptation and persistence. Determining the conditions that allow evolutionary rescue versus extinction is further complicated by the fact that, in the region of interest where either outcome is possible, population sizes reach low enough levels that many sources of stochasticity are present (demographic and genetic, for example), and mathematical models that encompass such stochasticity are challenging to analyse. One expects the rate of evolution by natural selection to be governed by the genetic variation in fitness, but such variation itself has a dynamic, boosted by mutation and depleted by selection and drift. This raises challenging questions of how predictable evolutionary rescue might be.

Models of evolutionary rescue have to date examined a number of genetic architectures that can underlie adaptation, from asexual or clonal variation (Gandon *et al.*, 2013; Martin *et al.*, 2013; Orr and Unckless, 2014), through mixtures of major and minor genes (Gomulkiewicz *et al.*, 2010), to polygenic models where many loci contribute to the trait undergoing selection (Gomulkiewicz and Holt, 1995; Holt and Gomulkiewicz, 2004; Holt *et al.*, 2005; Knight *et al.*, 2008; Gienapp *et al.*, 2013). Here, we return to two models of evolutionary rescue under the assumption of polygenic inheritance of a trait undergoing selection. In both models, an individual’s fitness depends on the difference between its phenotype (a single trait) and the phenotypic optimum for its environment (‘fitness’ is the mean number of offspring in one generation that survive to adulthood per adult in the previous generation). The first of these is a deterministic model of coupled demography and evolutionary dynamics based on one examined by Gomulkiewicz and Holt (1995), where they assumed that the heritability of a trait was

constant. We will briefly examine a heuristic extension of this model in which trait heritability varies as a deterministic function of population size. In general, small populations are expected to lose genetic variation (Lande and Barrowclough, 1987). The model suggests that a kind of bi-stability can exist, where populations that become too small are doomed to extinction, because of a downward spiral of increasing evolutionary impotence as their numbers decline following environmental change.

However, it is an implausibly strong assumption that genetic variation will follow population size so tightly. Thus in the remainder of this paper, we return to an individual-based model (IBM) we have explored previously (Holt *et al.*, 2003, 2005; Holt and Gomulkiewicz, 2004; Knight *et al.*, 2008), and examine in particular how initial measurements of a population can improve our ability to predict whether or not it will show evolutionary rescue. In both cases, we examine a simple kind of environmental change, where there is an abrupt change from one phenotypic optimum to another, and where in the initial environment the population has reached its evolutionary equilibrium in a stable environment. Then, over a single generation, there is an abrupt environmental change, after which there is no further change in the environment. This environmental shift leads to a transient phase of maladaptation in persisting populations, as well as a risk of extinction. Adaptive plasticity can help populations persist in a novel environment (Chevin *et al.*, 2010, 2013); we assume that such plastic responses may indeed have already happened in response to the environmental change, but that such a plastic response does not suffice to permit persistence; moreover, plasticity, if present, is implicit, not explicit, in our treatment.

### EVOLUTIONARY RESCUE WITH A DETERMINISTIC MODEL

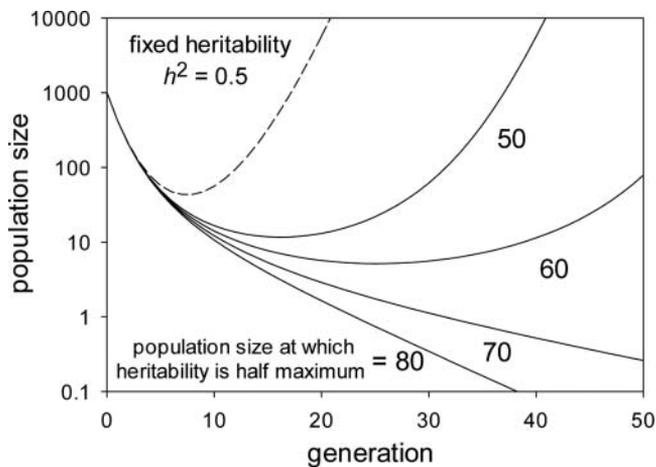
Deterministic models of evolution in an abruptly changed environment are useful because they often allow analytical results to be obtained, and can shed light on processes at play in more complex, stochastic models. Gomulkiewicz and Holt (1995), for example, used a deterministic model of the evolution of a quantitative trait in a discrete-generation population to determine the conditions that led a population subject to a sudden environmental change to usually persist and the conditions that led to its likely extinction. For this deterministic model, as long as heritability was positive, thus permitting evolution towards the new optimum, population size never became (or asymptotically approached) zero. Therefore, to determine whether extinction was biologically likely, they defined a heuristic critical population size below which extinction was expected, and determined the conditions for the population size to dip below this level (the initial population size was scaled against this critical population size). Their model tracked the population size  $N_t$  over time as well as the population's degree of maladaptation  $d_t$  (the difference between the population's average phenotype and the phenotypic optimum in the new environment, the latter of which is assumed to be 0). The recursions for these variables are  $N_t = \bar{W}_{t-1} N_{t-1}$  and  $d_t = k d_{t-1}$ , where

$$\bar{W}_t = W_{\max} \sqrt{\frac{w}{w+P}} \exp\left\{\frac{-d_t^2}{2(w+P)}\right\}$$

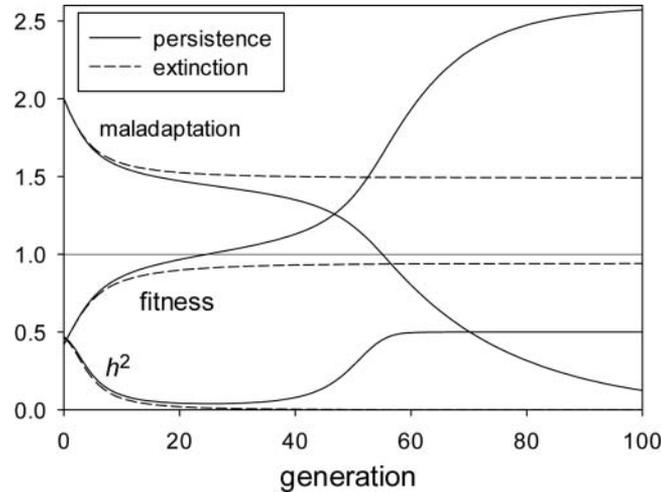
is the mean fitness at generation  $t$ , and  $k = [w + (1 - h^2)P]/(w + P)$ , where  $h^2$  is the trait's heritability. Individual  $i$  was assumed to have fitness  $W_{i,t} = W_{\max} \exp\{-d_{i,t}^2/(2w)\}$ , where  $d_{i,t}$  is the individual's maladaptation,  $w$  determines the width of the fitness function, and

$W_{\max}$  is the fitness with perfect adaptation; phenotypes are assumed to have a Gaussian distribution with variance  $P$ , and  $\bar{W}_t$  is the average fitness over all individuals. It was assumed that the maladaptation immediately after the environmental change ( $d_0$ ) was large enough for the initial fitness to be less than 1, so the population size initially declined, but that  $W_{\max} > 1$  (so that persistence was evolutionarily feasible). Since  $k < 1$  if heritability is non-zero, the maladaptation  $d_t$  declined each generation and fitness therefore increased. The boundary in parameter space between likely survival and likely extinction depended on the degree of initial population maladaptation (scaled by the sum of the phenotypic variance and the width of the fitness function), the initial population size, and the trait's heritability  $h^2$ . Higher initial population size and heritability or lower initial maladaptation made rescue more likely. The parameters of this model were all assumed to be constant.

However, as population size falls, the amount of genetic variability can change. In general, small population sizes are expected to lose genetic variation due to drift and inbreeding (Ellstrand and Elam, 1993). Thus one might conjecture that populations that are declining in abundance will also lose genetic variation, as estimated, for example, by reduced heritability for traits governing fitness. This could hamper the response to selection, and thus prolong the period during which a population is declining. As a first heuristic step towards elucidating the consequences of this feedback between demography and genetics, we modified the Gomulkiewicz-Holt model by making the heritability a saturating function of population size, so that,  $h^2 = h_{\max}^2 N / (N + N_{0.5})$ , where  $h_{\max}^2$  is the maximum heritability (which is approached for a large population),  $N$  is population size, and  $N_{0.5}$  is the population size at which the heritability is half its maximum. For low population sizes, heritability is approximately proportional to  $N$ . Population size trajectories for various values of  $N_{0.5}$  are shown in Fig. 1 (for  $h_{\max}^2 = 0.5$  and  $N_0 = 1000$ ). For a constant heritability, of course, populations ultimately recover (dashed line; recall this model is deterministic).



**Fig. 1.** Population size trajectories for the deterministic quantitative genetic model of Gomulkiewicz and Holt (1995) modified by making heritability dependent on population size. The initial population size is 1000, the maximum heritability is 0.5, and the population size at which the heritability is half its maximum is labelled next to each curve (higher numbers imply a greater drop in heritability for a given drop in  $N$ ). For the dashed line, heritability is constant at 0.5.

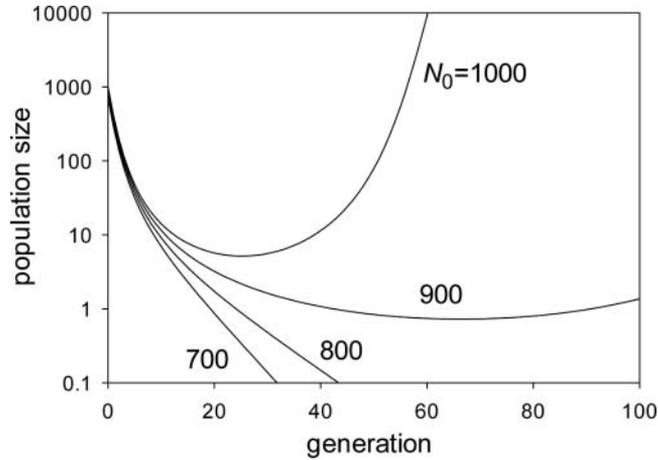


**Fig. 2.** Maladaptation, fitness, and heritability trajectories corresponding to the  $N_{0.5} = 60$  (persistence) and 70 (extinction) curves of Fig. 1. Selection causes maladaptation to drop and therefore fitness to increase. However, the initial drop in population size causes the heritability to decrease, slowing evolution. If fitness reaches 1, the population size and heritability increase and evolution accelerates (solid lines). If the initial slowdown in evolution is rapid enough, the fitness never reaches 1 and the population goes extinct (dashed lines).

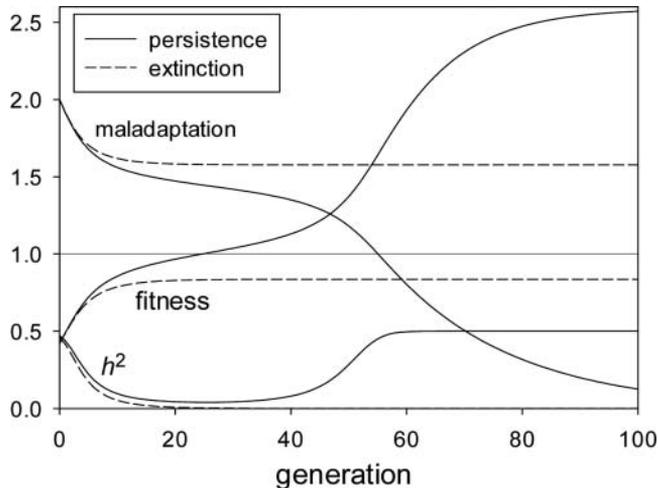
For  $N_{0.5} = 50$  or 60, the populations also recover, although they reach much lower population sizes, but for higher values (70 or 80), the populations asymptotically approach 0. Higher  $N_{0.5}$  implies that heritability is a stronger function of  $N$ .

Trajectories for heritability, maladaptation, and population mean fitness for  $N_{0.5} = 60$  (persistence) and 70 (extinction) are shown in Fig. 2. The populations begin with absolute fitness less than 1 just after the environmental change, so the population sizes initially drop as shown in Fig. 1. However, selection then causes evolution, so the degree of maladaptation starts to drop and as a result fitness increases. However, since heritability is a function of the decreasing population size, heritability decreases along with  $N$ , causing evolution to slow down. As long as fitness remains below 1, both population size and heritability continue to drop, and the rate of increase of fitness decreases. If fitness can reach 1, however, the population size starts to increase, as does the heritability, which causes evolution to accelerate (as shown by the solid lines in Fig. 2). However, even though fitness always increases, it is possible for it to be unable to reach 1 in the limit, if the slowdown in evolution is sufficiently rapid (as shown by the dashed lines). In this case, the population size asymptotically approaches 0. This model illustrates one possible cause of the ‘extinction vortex’ identified by Gilpin and Soulé (1986).

Figures 3 and 4 are similar to 1 and 2, except it is the initial population size that is varied rather than  $N_{0.5}$ . A relatively small decrease in initial population size can cause a large decrease of the population size trajectory, since the smaller population size means that evolution is slower, so the initial population size difference grows with time. This model thus reveals a kind of bi-stability – initially small populations decline inexorably towards extinction, whereas larger populations with the same population parameters eventually rebound in numbers and persist.



**Fig. 3.** Population size trajectories for the model of Gomulkiewicz and Holt (1995) modified by making heritability dependent on population size, for different initial population sizes. The maximum heritability is 0.5, the population size at which the heritability is half its maximum is 60, and the initial population size is given next to each curve.



**Fig. 4.** Maladaptation, fitness, and heritability trajectories corresponding to the  $N_0 = 1000$  (persistence, solid lines) and 700 (extinction, dashed lines) curves of Fig. 3.

This model should be viewed as a heuristic exercise, since the relationship between population size and genetic variation is unlikely to be as tightly bound as we have assumed. Our next model makes no such assumption (and indeed shows how the above model oversimplifies the dynamics of genetic variation in some crucial respects), but genetic variation still changes as population size varies, and this has important consequences for evolutionary rescue.

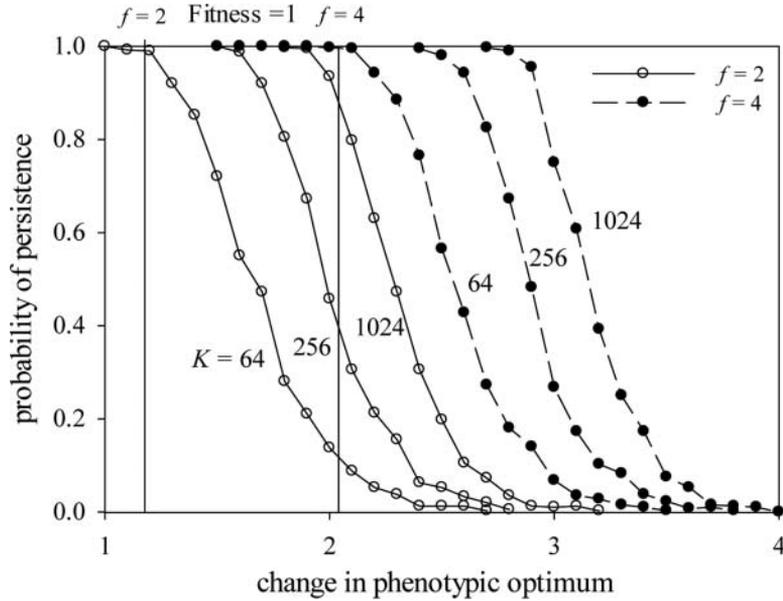
### INDIVIDUAL-BASED MODEL SIMULATIONS OF EVOLUTIONARY RESCUE

Deterministic models sometimes allow for analytical solutions, from which general conclusions can be drawn. However, they leave out many details, such as the many sources of stochasticity present in real populations. These can be included, however, by using simulations of individual-based models, which do not require as many restrictive assumptions as deterministic models, but give results only for the parameter sets simulated. We have used individual-based models based on that of Burger and Lynch (1995) to study the probability of adaptation in source–sink systems (e.g., Holt *et al.*, 2003) and the probability of success of invasive populations (Holt *et al.*, 2005). In these models, individuals are hermaphroditic and die after reproduction, with non-overlapping generations. Each individual has a trait (phenotype  $z$ ) that determines its survival from juvenile to adult (when selection takes place), which is given by  $\exp[-(z - z_{\text{opt}})^2/(2\omega^2)]$ , where  $z_{\text{opt}}$  is the optimum phenotype and  $\omega$  determines the strength of selection (a lower value representing stronger selection). The optimum phenotype is initially 0 but changes suddenly to a positive value  $\theta$ . An individual's phenotype is the sum of allelic values at  $n$  diploid loci and a random component that has a zero-mean Gaussian distribution with variance  $V_E$ . There is free recombination with no dominance or epistasis. Allelic values can take on any real value. During reproduction, each offspring haplotype mutates with probability  $n\mu$ ; if a mutation occurs, a random locus is selected and a zero-mean Gaussian with variance  $\alpha^2$  is added to the previous allelic value. Density can be regulated by limiting the number of mating sites to a value  $K$ . If there are fewer than  $K$  adults, then all adults mate as a female, while if there are more than  $K$ ,  $K$  are chosen at random to act as female. Each mating female mates with a randomly selected (with replacement) adult to act as male, which can be the same individual as the female (so selfing is allowed). Each mating pair then produces  $f$  offspring.

For all simulations described below, a population was initialized with  $K$  adults with random alleles that gave the expected steady-state average genetic variance and 1000 generations were simulated with the phenotypic optimum held constant at 0, to allow the population to reach mutation–selection–drift equilibrium. The change in the phenotypic optimum then occurred, and each simulation was continued for another  $T$  generations. Many simulations were done for each set of parameters, and the probability of persistence was the fraction of such populations that did not go extinct after this  $T$  generation period. When parameter values are not given, they are:  $K = 256$ ,  $\omega^2 = 1$ ,  $V_E = 1$ ,  $n\mu = 0.01$ ,  $\alpha^2 = 0.05$ , and  $T = 1000$ .

With a sudden change in the phenotypic optimum, the probability of persistence decreased for larger magnitudes of the imposed change, and the probability of persistence increased as either the population size (ceiling  $K$ , which is also the initial reproductive population) or fecundity increased (Fig. 5). The probability of persistence does not begin to drop until the change in phenotypic optimum is marked enough to reduce the fitness of a population at the initial phenotypic optimum to less than 1 (vertical lines). The effect of population size is approximately logarithmic (i.e. quadrupling  $K$  from 64 to 256 or from 256 to 1024 had about the same effect in shifting the persistence curves to the right).

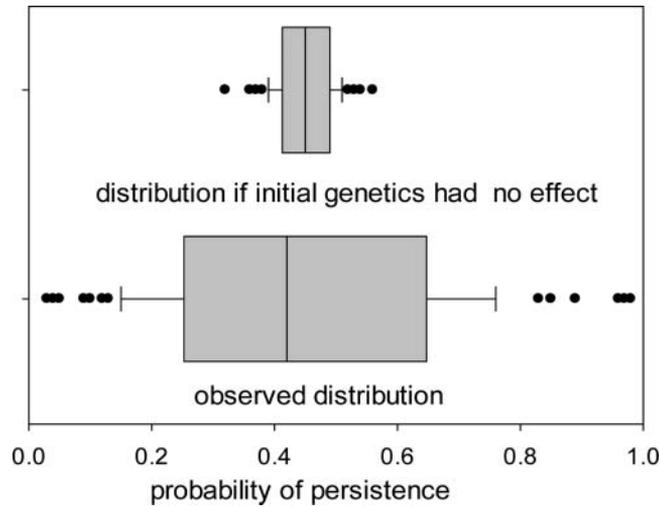
For Fig. 5, each point was determined by 400 populations simulated with the same parameters. Each population was initialized independently and simulated for 1000 generations before the change in phenotypic optimum to allow it to reach selection–mutation–drift equilibrium. In order to determine whether the genetics of the population at the time of the environmental change was important to population persistence, we



**Fig. 5.** Probability of persistence for 1000 generations of populations whose environment undergoes a sudden change in its phenotypic optimum of magnitude indicated on the abscissa. The ceiling on the number of reproductive adults,  $K$ , is indicated next to each curve (to the left for left three curves, to the right for right three curves), while the fecundity,  $f$  (number of offspring per reproductive adult), is 2 for the curves with open circles and solid lines and 4 for curves with solid circles and dashed lines. Vertical lines indicate where expected mean fitness of a population at the initial phenotypic optimum is 1. Altogether, 400 simulations were performed for each set of parameters.

performed additional simulations in which we initialized 100 populations, simulated each for 1000 generations, and then stored the values of the genetics of each population at the end of those 1000 generations (at the parental stage). We then used each of these parental sets to start 100 different populations undergoing immediate environmental change, and we simulated each for 1000 generations and recorded whether it persisted or not. We calculated the probability of persistence for each starting population (by which we mean the population at the time of environmental change), and the overall probability of persistence. If the genetics of the populations at the time of the environmental change did not cause differences in persistence probability, then the distribution of the persistence probabilities for the different starting populations should be binomial with parameters  $N=100$  (the number of runs with each starting population) and  $p$  approximately equal to the probability of persistence over all 10,000 populations (which was 0.444). Figure 6 shows box plots of 100 points from this binomial distribution (top) and the observed distribution of probabilities of persistence for the 100 starting populations (bottom). There is a very large difference between the two, indicating that the genetics of the population at the time of the environmental change is important in determining persistence. For one starting population, the probability of persistence was 98%, while for another it was 3%; both these would have an extremely low probability if starting population genetics did not matter.

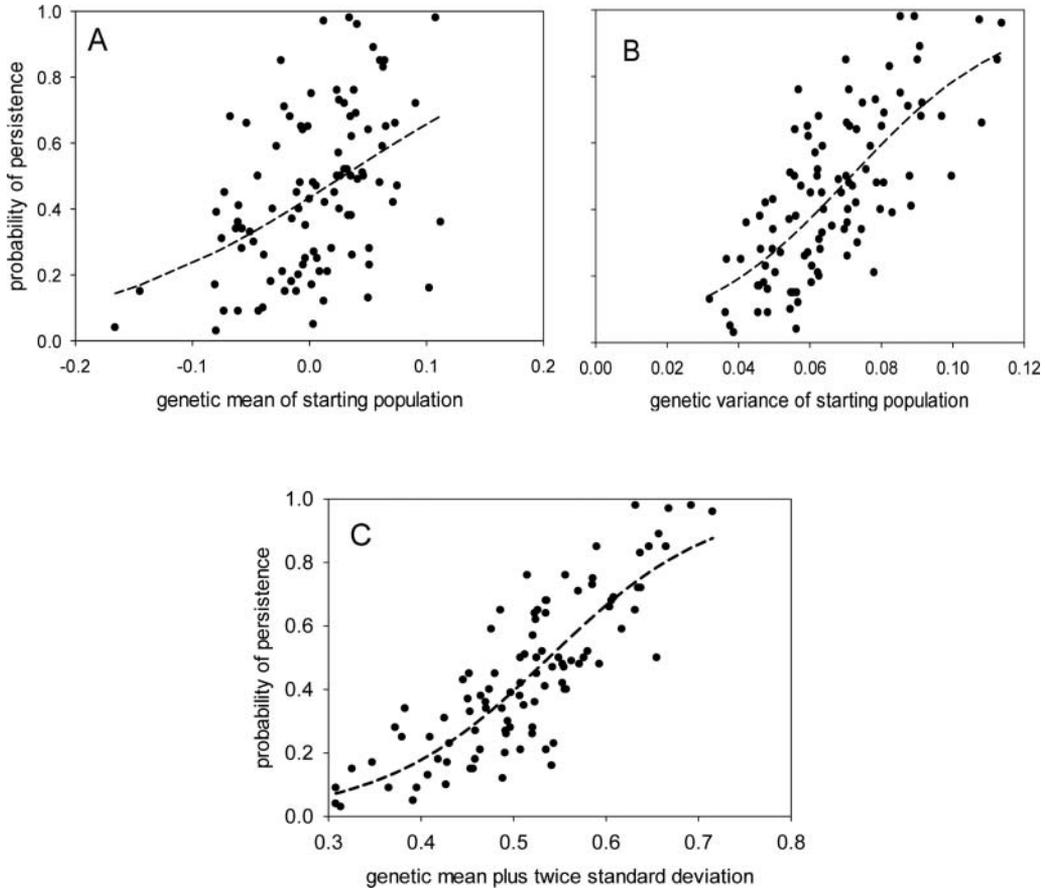
To characterize the important characteristics of the starting population genetics, we plotted the probability of persistence as a function of the genetic mean (Fig. 7A) and of the



**Fig. 6.** Box plots of the distributions of the probabilities of persistence for a sudden change in phenotypic optimum ( $\theta$ ) of 2.9 units. At the top is the distribution if the genetics of the population at the time of environmental change did not affect the probability of persistence, while the bottom is the observed distribution. The data for the upper box plot was 100 random deviates generated from a binomial distribution with parameters  $N = 100$  and  $p = 0.444$ , the average persistence probability over all 10,000 runs. The observed distribution had a standard deviation of 0.24 while the expected value with no effect of initial genetics was 0.05. Only 24% of observed values were within the 95% confidence interval assuming no effect of the starting genetics. The vertical line in each grey box is the median, the boxes extend to the first and third quartiles, and the whiskers extend 1.5 times the interquartile range from the boxes. Beyond the whiskers, each outlier is plotted.

genetic variance (Fig. 7B) of the starting population. In both cases, there was a highly significant effect, but there was considerable variation, which was lower with genetic variance (for linear regression,  $p < 0.001$  in both cases and  $R^2$  was 20% for genetic mean and 53% for genetic variance). The genetic mean might have been less important because it did not vary much; the change in phenotypic optimum was 2.9, while the genetic mean rarely was more than 0.1 units from the initial optimum. Populations with higher variance, in contrast, would likely have many more individuals significantly above the genetic mean (the highest genetic standard deviation is about 0.35). Since it is likely that persistence depends more on the upper tail of the genetic distribution (which contains individuals with the highest fitness after the environmental change), we then plotted persistence probability as a function of the genetic mean plus twice the genetic standard deviation. This resulted in a very significant relationship ( $p < 0.001$ ) with a moderate amount of spread (Fig. 7C), with  $R^2 = 68\%$ . Since the dependent variable is a probability, logistic regression was also used. For the genetic mean plus twice the genetic standard deviation predictor, logistic regression was again highly significant ( $p < 0.001$ ), with a 70% concordance rate (dashed lines in Fig. 7 are logistic fit; linear regression lines were quite close to these).

For the results above, we used persistence determined 1000 generations after the change in the environment. To investigate the timing of extinctions, we performed additional simulations in which we recorded, for each parameter set, the fraction of populations that had not gone extinct for each generation after the change in phenotypic optimum (up to

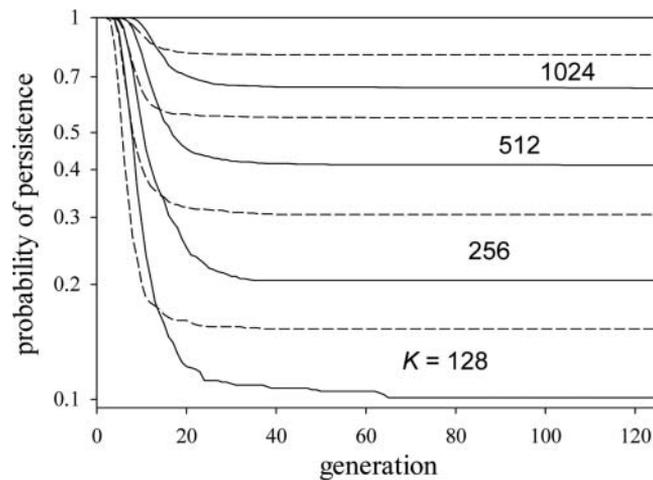


**Fig. 7.** Relationship between the probability of persistence and (A) the average genotype (genetic mean), (B) the genetic variance, and (C) the genetic mean plus twice the genetic standard deviation of the population at the time of environmental change for populations displayed in Fig. 6. The dashed lines are logistic regression lines.

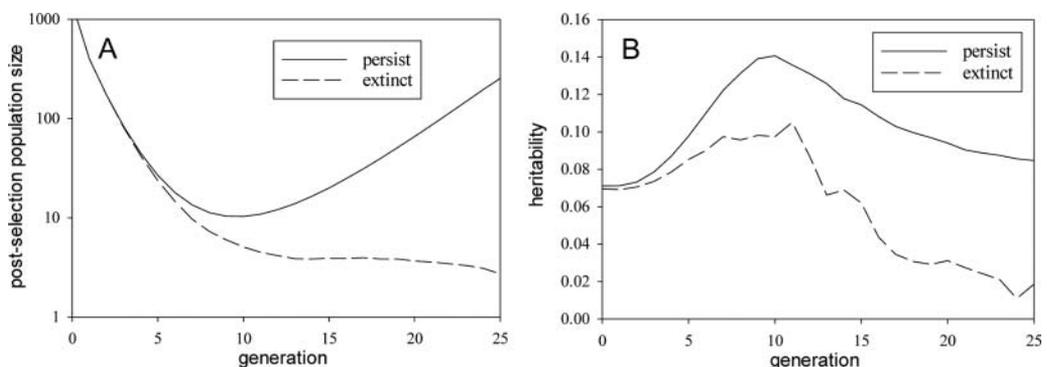
1000 generations). All populations persisted for the first few generations (longer for larger initial populations; Fig. 8), reflecting the time it took for even quite maladapted populations to drop low enough so that they were in danger of extinction due to demographic stochasticity. After this short initial period, there was a sharp drop in the probability of persistence (indicating many extinctions), followed by a levelling off as extinction frequency drops (eventually to 0; Fig. 8). For each parameter set, more than half the extinctions occurred by generation 15 and more than 90% by generation 23 (earlier for smaller populations); no extinctions occurred after generation 120. Therefore, there was a brief period after the environmental change when each population was vulnerable to extinction. If a population survived that short period, it usually persisted for a long time. This is very different from what we observed with a continual linear increase in the phenotypic optimum (unpublished results), for which populations continued to go extinct throughout each simulation (even out to generation 10,000), and in some cases after an initial period

there appeared to be an almost-constant per-generation extinction rate (Fig. 8 is plotted with a logarithmic ordinate scale, on which a constant extinction rate would be indicated by a straight line).

We also calculated the population size and heritability after the sudden change in phenotypic optimum. For the parameters used in Fig. 5 with  $f=2$ ,  $K=1024$  and a change in phenotypic optimum of 2.3, about 45% of the populations persisted. Immediately after the change in phenotypic optimum, the populations are maladapted and so their sizes decreased quickly (Fig. 9A). We surmised above that a decrease in population size could



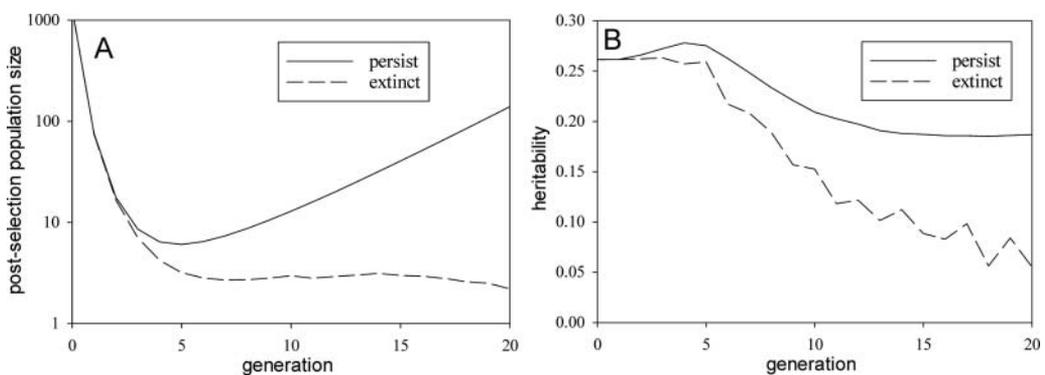
**Fig. 8.** Probability of persistence as a function of time, population size, and fecundity for a sudden change in the phenotypic optimum. Solid lines are for fecundity  $f=2$  and change in phenotypic optimum of 2.2; dashed lines are for  $f=4$  and change in phenotypic optimum of 3. Simulations were continued for 1000 generations, but no extinctions beyond generation 120 occurred. Altogether, 1000 simulations were done for each parameter set.



**Fig. 9.** Average trajectories of (A) population size and (B) heritability for populations that persisted for 100 generations (solid lines; 4575 populations) and populations that went extinct after generation 25 but before generation 100 (dashed lines; 313 populations) after a sudden change of phenotypic optimum of 2.3 units. The initial population size ( $K$ ) was 1024 and mutation rate per haplotype ( $n\mu$ ) was 0.01.

lead to a decrease in genetic variance and therefore heritability. However, in this case the heritability was initially fairly low (about 0.07; Fig. 9B) and actually increased as the population size decreased. For populations that persisted (solid lines in Fig. 9), the heritability started dropping near the time that the population size reached its lowest point, eventually returning to near its initial value. Populations that went extinct (dashed lines in Fig. 9) stayed at low numbers longer, and the average heritability did drop to below its initial value. The dynamics of heritability is clearly more complex than captured by our simple deterministic model.

It is interesting to consider the issue of when heritability will decline along with population size. For instance, in the results just presented, there was a low initial heritability. To increase heritability, we increased the mutation rate by a factor of 10. We also increased the change in phenotypic optimum to 3.8, which gave about 64% persistence; the higher mutation rates allow populations to persist with larger changes in the phenotypic optimum, probably mostly because of increased initial genetic variance, which should be approximately proportional to  $\mu$  (Burger and Lynch, 1995). The initial heritability was over 0.26 (Fig. 10B), and for populations that persisted it rose only slightly (to less than 0.28) before falling to less than the initial value. Heritability decreased while the population was still low, although the peak heritability did occur at the time of the lowest average population size. For populations that went extinct, the heritability was almost constant for the first five generations and then dropped (Fig. 10B). Therefore, in this case the heritability does tend to drop after the change in the phenotypic optimum, although there appears to be a lag between the drop in population and the drop in heritability. This is not surprising, as it takes time for selection and drift to purge genetic variation. Therefore, failure of evolutionary rescue could occur in part because sometimes heritability does drop at low population size.



**Fig. 10.** Average trajectories of (A) population size and (B) heritability for populations that persisted for 100 generations (solid lines; 12,836 populations) and populations that went extinct after generation 100 but before generation 200 (dashed lines; 123 populations) after a sudden change of phenotypic optimum of 3.8 units. The initial population size ( $K$ ) was 1024 and the mutation rate per haplotype ( $n\mu$ ) was 0.1.

## DISCUSSION

Understanding the factors that can permit species to persist in changed environments is of fundamental importance in our rapidly changing world (Hoffmann and Sgro, 2010). A population experiencing a change in environmental conditions (either *in situ* or due to population movement) might be subject to extinction if the change is large enough for the mean fitness of the population to be reduced sufficiently so the population cannot reproduce itself. The environment can change in many ways. We have examined here a large, sudden change in the environment. Since fitness is assumed to suddenly drop below 1, population size drops and the population has a limited time to adapt or it goes extinct. We presented a deterministic model in which heritability declines along with population size. This can lead to a kind of evolutionary trap for a population because once its numbers are low, selection is ineffective at increasing mean fitness. To predict extinction in this model, one has to know (i) the magnitude of the environmental change, (ii) initial population size, and (iii) how genetic variation changes with shifts in population size.

The individual-based model suggests that most extinctions occur soon after the change (see Fig. 8). Because the environment is assumed to be constant after the change, if the population succeeds in adapting, it has little risk of extinction thereafter. It is likely that the evolutionary contribution to population persistence draws on the initial genetic variation in the population, rather than subsequent genetic mutational input of variation. Larger initial population size makes adaptation and persistence easier (Fig. 1) by giving the population more time to adapt before its size drops to the point at which demographic stochasticity becomes significant. A smaller change in the environment also improves persistence, for two reasons. It leads to a smaller rate of initial population decline, also providing more time to adapt, but it also requires less adaptation before the population starts increasing. Because the generation of new genetic variation through low mutation rates generally takes a long time, adaptation to a sudden change depends largely on genetic variance that is present at the time of the change. This is illustrated in Fig. 7B, which shows that the probability of persistence can be strongly influenced by the initial genetic variance of the starting population. However, note the scatter around this regression line. There is considerable remaining uncertainty, even if one has a precise measure of initial genetic variance. In addition to variance, the mean genotypic value contributes to population persistence, but weakly so (Fig. 7A). More powerful than either is combining information on the initial mean and a metric of genetic variability (Fig. 7C). Again, there is considerable variability around the fitted logistic regression lines. Given the limited time for adaptation, it is probably very important that alleles be present in the population at the time of the environmental change that can be combined to yield genotypes with an absolute fitness near or greater than 1. A higher genetic mean and variance make this more likely, but do not guarantee it. What is important for persistence is not the total pool of genetic variation, but the tail of the distribution for which individuals have a higher absolute fitness than 1.

The bottom-line of the individual-based model is that evolutionary rescue becomes more predictable if one includes information about several aspects of the entire distribution of genotypic values, not just the mean or the variance. However, there is still a substantial amount of unpredictability, even with perfect information about the initial state of the system, because of inherent stochasticity in both extinction and the process of evolution. This is exemplified in the results summarized in Fig. 6. For each of the 100 starting populations, some of the replicates went extinct, and some persisted. In this example, even if one

had complete knowledge of the initial genetic and demographic conditions, some populations are likely to go extinct, and others to persist. Indeed, there are only a few initial populations where one can say with near certainty that the population will go extinct or persist. However, in other cases (Fig. 5), if there is only a modest environmental change, persistence is reasonably predictable. Similarly, an extreme change is likely to cause extinction.

The other general point from the IBMs is that initial population size can have a huge impact on the likelihood of persistence for intermediate magnitudes of changes in selective optima, but little impact if those changes are severe [Fig. 5; also a conclusion from Gomulkiewicz and Holt's (1995) model]. Bell and Gonzalez (2009) present experimental evidence that initial population size can definitely facilitate evolutionary rescue, and Bell (2013) points to some examples where very large initial population sizes were no buffer against extinction. Low-Decarie *et al.* (2015) exposed microbial communities to sudden severe stress and found that rescued communities were often dominated by initially common taxa, but in addition had some initially rare taxa that had greatly increased in frequency.

Another pattern in the individual-based model results that is reminiscent of the deterministic model is that populations that do go extinct overall tend to have lower heritabilities prior to extinction. Initially, however, in contrast to what was assumed in the deterministic model, heritability actually rises after the environmental change, even though population size is declining. A likely reason for this is that there were alleles segregating at low frequency in the pre-disturbance population that now become advantageous in the new environment. As their frequency rises, there can be a transient increase in genetic variance, since it takes a while for previously favoured alleles to be selected out of the population. But then genetic variance declines again as the newly favoured alleles increase towards fixation (without getting there) in the new environment.

It is useful to end by outlining directions for future work. We note that the heritability did start to drop, after an initial period, especially in those populations tending towards extinction (see Fig. 10). So a possible alternative to the deterministic model we presented above would be to craft a delay-differential equation model, where heritability changes with a lag after the population starts to decline (we thank J rgen Ripa for this suggestion). However, this approach would not capture the more complex phenomena revealed by the IBM, among which is that heritability can actually exhibit a transient phase of increase. Our approach of examining an abrupt step change in the environment, after which there is no further change, is just the first step towards understanding evolutionary rescue in temporally varying environments. Environmental change can happen in many other ways. For instance, a gradual but persistent change in the environment in one direction requires that a population continually adapt (Burger and Lynch, 1995). The continually changing environment means that there may always be a degree of maladaptation. The limit to the rate of change to which a population can adapt in this scenario is determined not so much by the initial standing genetic variation, but instead by how quickly it can generate new variation through mutation. Initial variation is less important, since it will eventually be insufficient to rescue the population if the directional change is persistent and becomes large. In this case, extinctions can continue to occur long after the change has first begun, since the population always has some chance of having a period during which it fails to generate sufficient variation. Population size is then likely to fall and reduce the rate at which new variation is generated. The resulting positive feedback can lead to sudden, rapid population decline even for populations that have adapted to the change for a long period (R.D. Holt and M. Barfield,

unpublished observations). In this case also, a large population size, or a less vigorous rate of environmental change, makes persistence more likely.

Other possible forms of change include mixtures of ‘steps’ and ‘ramps’, or autocorrelated stochastic changes, and more complex models would incorporate spatial heterogeneity and dispersal as well. A particularly significant limitation in the models explored here is that we have focused on evolutionary rescue in a single species coping with a changed environment. But species exist in webs of interacting species. This has both demographic and genetic consequences for evolutionary rescue. If the environmental change, for instance, knocks down the abundance of competing species, this could increase the initial growth rate of the focal species and thereby indirectly make the focal species more likely to adapt to the environmental change. Or, if there is selection on traits governing interspecific interactions, genetic correlations between these traits and the trait directly experiencing selection can hamper selective responses in the latter. All of these issues are ripe for further theoretical exploration and, critically, empirical testing with experimental evolution studies.

### ACKNOWLEDGEMENTS

We thank the University of Florida Foundation and NSF grant DEB-1354754 for support, Doug Morris and Per Lundberg for their efforts on behalf of this special issue and comments on our manuscript, and Jörgen Ripa for a helpful and insightful review.

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