Negative environmental perturbations may improve species persistence

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Among the factors proximally involved in the extinction of small isolated populations, genetic deterioration and temporal variation in environmental quality have been the subjects of intensive research in ecological and evolutionary sciences. However, previous theoretical studies and population viability assessments generally assumed a strict dichotomy between these two types of threat. Yet a number of empirical studies have recently suggested that the effects of genetic deterioration and environmental variation should not be considered independently, by demonstrating that the main effect of inbreeding depression lies with its tendency to exacerbate the deleterious consequences of environmental stress. Capitalizing on these results, I developed a stochastic model to examine the impact of random environmental perturbations on the persistence time of small isolated populations subject to inbreeding depression and mutation accumulation. The model assumes that spontaneous deleterious mutations have more severe effects when perturbations occur, which results in more efficient purging of the mutation load. Under this assumption, I find that negative perturbations may paradoxically improve middle- and long-term species persistence for realistic frequency of occurrence and severity distribution.

Keywords: population viability analysis; extinction; inbreeding; deleterious mutations; environmental perturbations

1. INTRODUCTION

As a consequence of habitat destruction and fragmentation, most of the plant and animal species at risk of extinction occur in several small isolated populations. Among the factors proximally involved in the extinction of such populations, genetic deterioration and temporal variation in environmental quality have been the subjects of intensive research in ecological and evolutionary sciences (Shaffer 1987; Lande 1993; Lynch *et al.* 1995). Within the last two decades, the strict opposition between genetic and environmental causes of extinction has led to a controversy over their respective weights in limiting population viability (Lande 1988; Caughley 1994; Spielman *et al.* 2004). The claim that the environment is of greater importance than genetic aspects in reducing persistence is partly due to the broader array of conditions under which environmental variations may be a threat to populations. It is well established that environmental stochasticity (i.e. temporal fluctuations in vital rates of all individuals in a similar fashion, due to variation in the quality of the environment) and catastrophes (i.e. rare, severe environmental events negatively affecting vital rates) have a major influence on the viability of both small and large populations (Shaffer 1987; Lande 1993). In contrast, genetic problems are more inherently associated with small and isolated populations (Lande 1988; Whitlock 2000). In the short run, the major genetic consequence of the reduction of population size is

the rapid increase of the frequency of individuals homozygous for deleterious alleles identical by descent, resulting in the reduction of fitness termed inbreeding depression (Hedrick & Kalinowski 2000). At longer time-scales, gradual processes may lead to an accumulation of deleterious mutations in the population and subsequent fitness decrease (Lynch *et al.* 1995). Inbreeding is associated with elevated extinction risk in experimental, captive and natural populations (Saccheri *et al.* 1998; Bijlsma *et al.* 2000; Reed & Bryant 2000).

The debate over the respective importance of environmental and genetic threats has been partially resolved in recent years, owing to strong evidence of the effect of inbreeding on extinction risk (Spielman *et al.* 2004). Ecological as well as genetic threats to population persistence are now considered jointly in books and collective books on conservation biology (Young & Clarke 2000; Frankham *et al.* 2002) and in some population modelling studies (Mills & Smouse 1994; Robert *et al.* 2005; Volis *et al.* 2005). However, in these studies, genetic and environmental factors are assumed to reduce vital rates independently, without any real integration between these processes.

My focus is on single populations potentially subject to both environmental and genetic threats. However, rather than examining the respective weights of these two types of threats, I focus on their interaction. In demographic terms, environmental harshness can be modelled as a chronic or punctual constraint reducing the mean of one or several demographic rates in a given population. What is generally neglected in population models is the effect of such constraint on the inter individual variance of these rates and its subsequent effect on selection. Recent

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The electronic supplementary material is available at <http://dx.doi.org/10.1098/rspb.2006.3601> or via <http://www.journals.royalsoc.ac.uk>.

empirical literature strongly suggests that increasing environmental harshness results in more efficient selection. In humans, it has been proposed for decades that the improvement of medicine leads to relaxed selection and accumulation of deleterious mutations (Crow 1997). The genetic–environment interplay is also expected to affect mutation detectability in mutation accumulation experiments in laboratory species. This is a possible explanation for the discrepancy between mutation rate estimates obtained under competitive conditions in *Drosophila melanogaster* (Mukai *et al.* 1972) and under non-competitive conditions in *Caenorhabditis elegans* (Keightley & Caballero 1997). The evidence of this type of interaction has been strengthened by recent work on inbreeding depression in various taxa. Inbreeding depression has been shown to be more severe in natural than in artificial environments (Crnokrak & Roff 1999) and more severe in stressful than in benign environments (Bijlsma *et al.* 2000; Cheptou *et al.* 2000; Meagher *et al.* 2000; Keller *et al.* 2002). It is therefore likely that it occurs mostly following biotic or abiotic environmental perturbations (Keller *et al.* 1994; Coltman *et al.* 1999).

Here, I use individual-based models that include stochastic demographic, environmental and genetic mechanisms. Genetic deterioration is considered by assuming a mixed model system in which inbreeding depression is due to partially recessive deleterious alleles of both large and small effect initially at the mutation–selection balance frequency (Charlesworth & Charlesworth 1999). Punctual environmental perturbations occur stochastically with a probability P_c at each time-step to reduce population size in a proportion C . During these events, the removal of individuals from the population occurs with respect for their genotype, with the individuals of low fitness being preferentially removed. Under these assumptions, I find that punctual negative environmental events result in healthier populations and may eventually improve species viability.

2. MATERIAL AND METHODS

(a) *Species life cycles*

I use two different stochastic life cycles. First, I consider a dioecious semelparous species (non-overlapping generations) in which males and females pair randomly according to their social mating system. Both monogamous and polygynous mating systems have been used. For the polygynous system, I assumed no restriction in harem size (Poisson distributed). Individuals reproduce in each time-step (generation) and all adults die after reproduction. The individual fecundity is F (Poisson process) and each newborn individual has a probability s_0 of surviving until reproduction (Bernoulli trial). The sex of each individual is randomly determined according to a 1 : 1 sex ratio.

The second life cycle is a dioecious iteroparous life cycle (overlapping generations) in which individuals pair in each time-step (year) only if they have reached the age of first reproduction. As for the semelparous life cycle, the mating system is either monogamous or polygynous. The theoretical set of demographic parameters used in this model corresponds to typical long-lived birds or mammals (e.g. carnivore, raptor; parameters are presented in table 1).

Table 1. Theoretical demographic parameters used in the iteroparous demographic model. ($s_{(x)}$ is the annual survival rate between age x and $x+1$. The deterministic growth rate and generation time obtained with this set of parameters are, respectively, 1.1 and 13.8.)

parameter	value
juvenile survival $s_{(0)}$	0.8
immature survival $s_{(1)}$	0.8
immature survival $s_{(2)}$; $s_{(3)}$	0.9
adult survival $s_{(\geq 4)}$	0.94
age at maturity	4
annual individual fecundity	0.4

(b) *Population regulation*

In the main simulation, population size is truncated to the carrying capacity K in each year, independently of the genotypes and ages of individuals. I examined an alternative model for regulation, by assuming a linear decrease of fecundity with increasing population size N , where the mean individual fecundity at time t is given by $F_{(t)} = 1 + ((F_{\max} - 1) (1 - N_{(t)}/K))$, $F_{\max}$ being the fecundity in the absence of density dependence. As no qualitative effect of the system of density dependence was detected, results are only presented for the truncation method.

(c) *Genetic mechanisms*

Genetic factors are parameterized using values from a broad array of empirical studies (Mukai *et al.* 1972; Drake *et al.* 1998; Lynch *et al.* 1999). The genome of each individual is explicitly represented as two series of $L=1000$ different diploid loci. Each of these series can carry two types of allele at each locus: a wild-type and a deleterious allele. The first and second series are used to model, respectively, mildly deleterious and lethal mutations. The coefficients of selection, coefficients of dominance and average numbers of genomic mutations per generation are, respectively, $s_d=0.02$, $h_d=0.35$, $U_d=1$ for mildly deleterious mutations and $s_l=1.0$, $h_l=0.02$, $U_l=0.05$ for lethal mutations. Initial frequencies of mildly deleterious and lethal alleles q_{0d} and q_{0l} are given by the mutation–selection balance. In the main simulation, selective coefficients used to compute these initial frequencies are, respectively, s_d and s_l (mutation effects in the absence of perturbation). Using these mean frequencies, the initial number of each type of deleterious alleles present in each founder is stochastically determined from a Binomial distribution.

Additionally, I tested different scenarios regarding the initial selection–mutation equilibrium frequencies of deleterious mutations q_{0d} and q_{0l} to consider the effect of the environment on selection in the source population from which individuals are sampled. In all situations, q_{0d} and q_{0l} are given by the mutation–selection balance frequency, assuming that the effective selective coefficients of mildly deleterious and lethal mutations in the large source population are s'_d and s'_l , with $s'_d = (1 + \alpha)s_d$ and $s'_l = (1 + \alpha)s_l$. α is a coefficient describing the rate of additional selection due to the presence of perturbations in the source population.

During fertilization, the probability of transmission of each allele at each locus is given by the Mendelian rules. New mutations stochastically occur in each zygote (Poisson distributed, with means U_d and U_l). I assume multiplicative interactions for fitness (no epistasis) and free recombination of all loci (no linkage).

(d) Effects of genetics and environmental variation on vital rates

I assume that, deleterious alleles act by reducing juvenile survivorship only. In the absence of environmental perturbation, the survival rate of the individual i is given by:

$$s_{0i} = s_0 v_{di} v_{li},$$

with the relative reductions in survival due to mildly deleterious and lethal alleles in individual i being given by:

$$w_{di} = (1 - h_d s_d)^{nd1i} (1 - s_d)^{nd2i} w_{d0}^{-1} \quad \text{and} \quad w_{li} = (1 - h_l s_l)^{nl1i} (1 - s_l)^{nl2i} w_{l0}^{-1},$$

s_0 is the expected survival rate of mutation free individuals; $nd1i$ and $nd2i$ are, respectively, the numbers of heterozygous and homozygous mildly deleterious mutations carried by the individual i ; $nl1i$ and $nl2i$ are, respectively, the numbers of heterozygous and homozygous lethal mutations carried by the individual i ; and w_{d0} and w_{l0} are the expected initial reductions in survival due to mildly deleterious and lethal alleles present at time zero, given by:

$$w_{d0} = (1 - h_d \cdot s_d)^{2L \cdot q0d} \quad \text{and} \quad w_{l0} = (1 - h_l \cdot s_l)^{2L \cdot q0l}.$$

Punctual perturbations occur stochastically with a probability P_c at each time-step t to reduce population size in a proportion C . An individual i is randomly drawn from the pool of $N_{(t)}$ living individuals and is removed from the population if a uniform random number is higher than $w_{di} w_{li}$. The process is repeated until N_r individuals are removed, with N_r being drawn from a Binomial distribution ($N_{(t)}, C$).

(e) Model with constant population size

I also use a non-overlapping generations one sex model with constant population size N , in which individuals pair and reproduce randomly (with possibility of self-fertilization at the random rate) until the number of newborn individuals reaches $N C^{-1}$. The expected reproductive output of an individual i is $(w_{di} w_{li} F)$ (Poisson distributed). Perturbations occur randomly. In case a catastrophe occurs, population size is truncated to N , with each individual i having a probability to die proportional to $w_{di} w_{li}$. If no perturbation occurs, population size is simply randomly truncated to N .

Initially, $N_{(0)}$ individuals are present in the population, with $N_{(0)} = K$. Extinction occurs when population size is equal to zero. Population viability and fitness evolution are investigated by using Monte Carlo simulations in which 1000 population trajectories are drawn. Simulations are run either over a fixed time horizon (100–500 generations) or until the extinction of all trajectories.

3. RESULTS AND DISCUSSION

(a) Effect of perturbations on fitness

In order to avoid biases due to extinction and variation in population size in fitness assessments, I first use a non-overlapping generations one sex model with constant population size N . As a result of selection improvement, perturbations have a positive effect on W_d and W_l (the mean relative contributions to fitness of loci with mildly deleterious and lethal mutations), which increase with both their severity and frequency (figure 1a). Results however indicate that this increase is mostly due to mildly deleterious mutations, perturbations having almost no

Figure 1. Relative indexes of population growth as a function of the frequency of perturbations. Model with constant population size ($N_0=K=100$). (a) Reduction in relative fitness in the presence of lethal (circles), mildly deleterious (triangles) or both types of mutations (squares; after 100 generations). Constant perturbation severity with $C=0.5$ (solid lines) and $C=0.75$ (dashed lines). (b) Reduction in relative fitness in the presence both types of mutations (triangles), mean demographic effect of perturbations on the growth rate (circles) and cumulative effect of mutations and perturbations on the growth rate (parameter r_l , squares; after 500 generations). Constant perturbation severity with $C=0.75$.

effect on selection against recessive lethals. This positive effect on population growth is obviously counter-balanced by a direct negative demographic impact of perturbations on the long-term growth rate of the population. This impact is proportional to $1 - P_c C$. When considering both genetic and demographic effects using the parameter r_l (proportional to the long-term growth rate and defined as $r_l = W_d W_l (1 - P_c C)$), the most beneficial situation (i.e. the situation that maximizes r_l) is obtained for an intermediate frequency of perturbations (figure 1b).

(b) Effect of perturbations on persistence time for different demographic scenarios

In a second step, the impact of perturbations on viability is assessed by using a fully stochastic demo-genetic model, in which the overall impact of perturbations on population persistence results from a balance between their effects on selection and demography. This balance depends on the frequency of perturbations and strongly interacts with the reproductive potential of the species. Species with high-reproductive potential show a non-monotonous variation in persistence time when increasing perturbation frequency (figure 2). Very rare perturbations have a negligible effect on selection and are eventually disadvantageous. Frequent perturbations have a beneficial effect on selection, but they ultimately induce a reduction in population persistence owing to their strong negative impact on long-term population growth (Lande 1993).

Figure 2. Mean (solid circles, solid lines) and median (open circles, dashed lines) times to extinction (in generations) as a function of the frequency of environmental perturbations with constant severity ($C=0.5$). Stochastic semelparous life cycle, with different deterministic growth rates λ depending on the individual fecundity F . Juvenile survival rate s_0 is 0.5 in all cases. ($N_0=K=100$). Both types of deleterious mutation are considered.

However, perturbations of intermediate frequency have an overall beneficial effect on persistence, the optimal frequency being an increasing function of the reproductive potential of the species considered. The maximum growth rate of the species (expressed on a per generation basis in figure 2) strongly affects this result because it determines the time-scale over which extinction occurs. Slow-growing species show a monotonous decrease of persistence time with increasing perturbation rate, owing to their intrinsically short-persistence time, and the subsequent low contribution of mutation accumulation to extinction. However, most existing populations are intrinsically growing and the factors limiting the viability of many endangered populations are related to extrinsic constraints on their carrying capacity. Most vertebrate species, even those of conservation concern, have per generation growth rates higher than 50% (see Niel & Lebreton 2005 for birds), which makes the results presented here relevant for many endangered species. These trends observed for semelparous species are qualitatively similar when examining iteroparous overlapping generations species (see §3d).

(c) Importance of genetic assumptions

Classical mutation accumulation experiments on *Drosophila* have suggested that the per generation diploid genomic mutation rate (U) was high (on the order of 1), with a weak average effect on fitness of each mutation (a few percent; Mukai *et al.* 1972). In nematodes, other experiments (see Keightley & Caballero 1997) have uncovered lower mutation rates and larger average effects. Recent evidence indicates that there is substantial variation in genome-wide properties both within and between species (Baer *et al.* 2005). A sensitivity analysis of the results to the mutation parameters (U , s and h) has been conducted, indicating that the validity of the qualitative conclusion presented here (i.e. a maximal persistence obtained for non-zero perturbation rates) primarily depends on the average mutational effect s . When s is large, mutations are easily counter selected, even in the absence of perturbation, so increasing perturbation rate becomes always deleterious to population persistence.

Figure 3. Influence of initial frequency of deleterious mutations of populations on their viability. α represents the rate of additional selection due to perturbations in the source population. Stochastic iteroparous life cycle ($N_0=K=50$), with both types of deleterious mutations. Empirical severity distribution of perturbations of Erb & Boyce (1999). The dashed line indicates the mean time to extinction of the reference population ($\alpha=0$; no perturbation).

The dominance coefficient h and mutation rate U have no qualitative impact on results. Changes in mean persistence time according to the frequency of perturbations are presented for different values of s , U and h in fig. 1 of electronic supplementary material.

If perturbations have an effect on selection in small populations, they may have an effect on allelic frequencies in large populations as well. Thus, I tested additional scenarios regarding the initial selection–mutation equilibrium frequencies of deleterious mutations in order to consider the effect of environmental perturbations on selection in the source population from which individuals are sampled. The coefficient α was used to describe the rate of additional selection in the source population (see §2). Times to extinction with increasing α and different frequencies of perturbations P_c were compared with a reference population with no perturbation nor additional previous selection (i.e. $\alpha=0$ and $P_c=0$; dotted line on figure 3). In all cases, results are only weakly sensitive to α indicating that previous selection in large source populations has little impact on persistence, compared with the impact of current selection in small isolated populations.

(d) Use of variable perturbation severity

Although the trends described above still occur when the severity of perturbations is variable, the balance between their positive and negative effects greatly depends upon the shape and variance of the severity distribution. Results for constant, normal and uniform distributions are presented in figure 4a. Other theoretical distributions (e.g. negative exponential, beta) have been used to model perturbation severity. In all cases, a non-zero optimal frequency of perturbation is obtained when the occurrence of very severe perturbations ($C>0.9$) is small relative to the occurrence of perturbations of intermediate severity ($0.2>C>0.8$). For a given average effect, an increase in the variance of severity distribution has no impact on

Figure 4. Mean times to extinction (in years) as a function of the annual frequency of environmental perturbations. Stochastic iteroparous life cycle ($N_0 = K = 50$). Both types of deleterious mutation are considered. (a) Use of different theoretical severity distributions of perturbations for a given average effect (50% decrease in survival). Solid squares, constant distribution ($C = 0.5$); open triangles, Normal distribution (mean = 0.5; variance = 0.0225); open circles, uniform distribution with parameters 0 and 1. (b) Empirical severity distributions of Young (1994) (solid triangles) and Erb & Boyce (1999) (open squares).

selection in the long run, but it increases the probability of stochastic extinction following a severe perturbation. Perturbations have therefore an overall positive effect on persistence when the variance in severity distribution is low (see figure 4a). Recently, using extensive data on mammals, Young (1994) concluded to an over abundance of catastrophic die-offs in the 70–90% range and an underabundance of die-offs greater than 90% (relatively to a uniform distribution of die-off severity). Young's data reanalysed by Erb & Boyce (1999) were closer to a negative exponential distribution, with a virtual absence of die-offs greater than 90%. These empirical distributions were used to model catastrophe severity in our context. As for the theoretical ones, they lead to improved persistence times for perturbations of intermediary frequencies (figure 4b). Although the frequency of environmental perturbations in nature is difficult to assess due to the requirement of long-term records, some authors have reported frequencies on the order of few percents per year within taxa (Gerber & Hilborn 2001). A recent study encompassing 88 species of vertebrates indicates that the probability of a severe die-off for a particular population is approximately 14% per generation (Reed *et al.* 2003). This estimate corresponds to a rate of 1% per year with the set of demographic parameters used in figure 4. However, this value may be underestimated in our case because most of the studies cited above considered only reductions of population size of 50% or greater.

Figure 5. Mean time to extinction (in generations) as a function of the carrying capacity, in the presence (open triangles) or absence (solid triangles) of environmental perturbations (with constant severity $C = 0.7$ and per generation frequency $P_c = 0.1$). Stochastic semelparous life cycle, with individual fecundity $F = 2.75$ and juvenile survival $s_0 = 0.5$.

Environmental perturbations may obviously have an unconditionally negative and important influence on viability for most populations (Shaffer 1987; Lande 1993). In agreement with this statement, sensitivity analyses suggest that the improvement of viability by environmental perturbations occurs for a restricted range of population size. As exemplified in figure 5, the extinction of extremely small populations occurs on average at very short-time-scale (i.e. before genetic factors substantially impact them). The stochastic extinction of such populations is therefore accelerated in the presence of perturbations further reducing population size. At the opposite, perturbations have also an overall negative effect on the persistence of large populations. Environmental perturbations, and in particular, catastrophes, are indeed the major source of extinction for populations with effective sizes larger than few 1000s, for which inbreeding depression and mutation accumulation play minor roles in extinction (Lande 1988). However, the persistence of populations maintained at intermediate sizes is primarily reduced by the progressive fitness erosion caused by the fixations and accumulation of mildly deleterious mutations (Lynch *et al.* 1995; Whitlock 2000; see figure 1). The long-term persistence of such populations may therefore be improved by the presence of perturbations for realistic values of perturbation severity and frequency. This emphasizes the complex role of environmental perturbations on species dynamics, which, besides their demographic deleterious effects, may act as evolutionary buffers against extinction. This conclusion is however qualitative and the application of these findings into management plans would require precise data on the effects and magnitude of environmental factors on selection, which are generally not available.

Further work is required to assess the more general effect of extrinsic and intrinsic constraints on selection and on the persistence of small populations under regulation, especially when the persistence of these populations is limited by anthropogenic perturbations. Although different kinds of environmental pressures (physical/climatic event, pollution, predation, exploitation, parasitism, competition,

reduction of resources or habitat) may have equivalent effects on survival/reproduction in a given species (and are therefore merely accounted as punctual or chronic constraints on population dynamics in population models), they may engender very different levels of discrimination between individuals of distinct genotypes. This suggests that genetic and demo-genetic models neglecting the environment–genetics interaction may strongly misestimate the extent of selection, yielding biased short-/long-term estimates of fitness and extinction rates.

I thank Denis Couvet and two anonymous referees for comments on the manuscript.

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