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Fitness heterogeneity and viability of restored populations

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Abstract
We develop a stochastic model of population viability which explicitly links demography and genetics in order to examine the consequences for extinction dynamics of different levels of heritable fitness variance within a population. We particularly focus on situations in which a local small population is artificially built with individuals that were taken from several large source populations. Our results suggest that different levels of fitness variability within a population (due to partially recessive deleterious alleles rather than local adaptation) have a large influence on its viability. Moreover, the optimal level of fitness variance for maximizing population persistence is a function of the species life-cycle. Two mechanisms with opposite effects are mainly responsible for the different patterns of extinction obtained depending on the life-cycle, (1) purging of deleterious alleles, (2) demographic stochasticity. For high growth rate or long-lived species, a high fitness variance decreases short-term viability and increases long-term viability. In contrast, for other cases, a high fitness variance increases both short- and long-term viability.

INTRODUCTION

To increase the success of reintroduction of a population, in relation to its genetic composition, two strategies have been proposed. The first consists of releasing individuals from the populations most likely to have local adaptations to the release site, such as indigenous populations (May, 1991; Montalvo & Ellstrand, 2000). Such an approach is not always possible, owing to a lack of appropriate individuals, and it involves the risk of producing a population with a low genetic variation precluding future adaptations. The alternative strategy is to release individuals from a large diversity of populations in order to maximize the genetic variability on which selection will act (Tordoff & Redig, 2001). Our topic is to study the consequences, in terms of population viability, of different levels of variance of the genetic load, by using a genetic and demographic model of population viability. We will focus in the present paper on the case of small restored populations which are subject to a decrease in fitness owing to the accumulation of deleterious alleles which can lead to extinction (Lynch *et al*., 1999). When considering deleterious mutations, gathering several individuals from assorted environments to release them in the same site can result in producing a very heterogeneous population for two main reasons. First, the mean frequency of unconditionally deleterious mutations present in a given population depends on its past and present size, so that frequency may vary according to the size of the source population. Second, disparity among populations in past selection against deleterious mutations may result in a disparity in the current allelic frequencies, particularly in the cases where some of the released individuals originate from captive populations where selection is relaxed (Frankham *et al*., 1986; May, 1991).

In this paper, we use a simulation model in order to investigate the effects of the heterogeneity of fitness within a population on population viability. In our models this heterogeneity is expressed as the variance in the frequency of deleterious mutations, i.e., mutations that are detrimental in the release site of the restored population. Although the genetic load (for *Drosophila melanogaster*) has been characterized as being comprised of both a lethal component and a mildly deleterious component (Simmons & Crow, 1977; Charlesworth & Charlesworth, 1987; Lande, 1988; Crow, 1993) we focus on mutations of small effect and do not consider lethal recessive mutations since we know from earlier

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simulations that their influence on extinction rate is small compared with mildly deleterious mutations (Hedrick, 1994).

From a high heterogeneity of fitness among the founders of a reintroduced population, we can expect an unequal representation of the genome of the released individuals, which may influence the mean fitness of the overall population in two ways. On one hand it increases the potential for purging of deleterious alleles by increasing the variability on which selection acts (Couvet & Ronfort, 1994). On the other hand, an unequal representation decreases effective size and leads to lower levels of genetic variation in the population, relative to a population derived from the same number of founders in which all have equal representation (Lacy, 1989). One of our goals is to compare the relative effects and interactions of these two genetic processes and their impact on population viability. However, the amount of fitness heterogeneity also has a very strong impact on population demography, and particularly on the risk of extinction by demographic stochasticity. Since the different genetic and demographic processes involved in fitness variability can have opposite effects in terms of viability, a better understanding of their potential consequences on released populations' persistence requires the study of their interplay for different life-history strategies.

METHODS

Life-cycle

We use a two-sex individual-based model. The individual-based approach in which each individual is characterized by a list of parameters allows us to consider explicitly the variability within the population in terms of genetic characteristics, sex and age.

As a first step, we use non-overlapping generation models, in which males and females in each generation pair according to their social mating system and all adults die after reproduction. Fecundity is then the only parameter of fitness (see Appendix). In order to apply our model to species with overlapping generations presenting different realistic life-history traits, we also simulate the impact of fitness variability on extinction for a long-lived species and for a short-lived one. The demographic parameters used for these models are presented in Table 1. These demographic parameters were computed to obtain different generation lengths with a same asymptotic growth rate, by using a deterministic matrix model (computer program ULM; Legendre & Clobert, 1995; Ferrière *et al*., 1996). For overlapping generation models, each time step, males and females pair, and reproduction is followed by differential survival according to the interaction between genotype and age for each individual (see Appendix).

In order to test the robustness of our results to different mating systems, we simulate some reintroductions using a monogamous mating system, in which males and females are paired one to one, and using a polygynous mating system, in which a single male can mate with several females, without any restriction in the number of females per male.

Table 1. Demographic parameters used for two types of species with overlapping generations, computed for an equivalent deterministic growth rate. The generation time is computed as the mean age of the parents of the offspring produced by a population at the stable age distribution (Caswell, 2001).

	Short-lived species	Long-lived species
Juvenile survival (s_0)	0.4	0.551
Immature survival (s_1)	–	0.735
Adult survival (v)	0.535	0.89
Age at maturity	1	3
Productivity (f)	4.77	0.82
Generation time	3.08	9.38
Life expectancy at birth (ls)	1.3602	4.7326
Deterministic growth rate	1.03	1.03

Demographic stochasticity for reproduction results first from the drawing of the number of offspring of each reproducing female from a Poisson distribution and second from the random determination of the sex of each individual. Further, each survival event is drawn from a Bernouilli function.

All reintroductions take place on empty sites. Because all individuals are released in generation zero, the initial number of individuals in each simulation is equal to the number of released individuals.

Because our study focuses on processes acting when population size is small, density dependence has no impact on our results. Population size is truncated to the carrying capacity in each generation. In all the cases discussed below, the carrying capacity is 1000 except where we explicitly examine the effect of the carrying capacity (non-genetic model, Fig. 1).

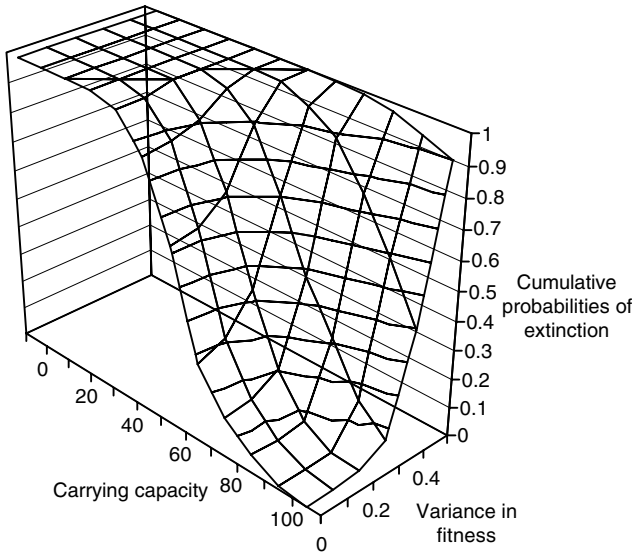

Fig. 1. Cumulative probabilities of extinction of a restored population (non-genetic model) as a function of the carrying capacity and the variance in fitness. (Probabilities were obtained after 100 generations.)

Genetic characteristics

The genome of each individual is described as a series of integer values, each representing the state of a given locus among 100 different diploid loci. The initialization of the genomes of the released individuals depends on the hypotheses we make concerning their origins and the heterogeneity among them, but in all cases we assume that they descend from large populations. Thus, the mutation–selection balance gives the mean initial frequency of mutations present in the genome. The extent of fitness variability within the released population is given by a Gaussian distribution on the number of deleterious mutations initially present in each individual. It allows the simulation of numerous cases of population releases with the same mean initial fitness in which we can modify the variance. The probability of transmission of the parental alleles at each locus during the fertilization is given by Mendelian rules. In the case of a very small population, fixation of old mildly deleterious mutations present in the founders can contribute substantially to the risk of extinction (Lynch *et al*., 1993, 1999; Lynch, Conery & Bürger, 1995 *a*, *b*). In our model we used a constant coefficient of selection s of 0.05 for the life-time disadvantage for non-overlapping and overlapping generations simulations, corresponding to values commonly assumed (Simmons & Crow, 1977; Lynch *et al*., 1999). Moreover, we assume a coefficient of dominance h of 0.3, which is supposed to be the harmonic mean of the values of h across loci, weighted by mutation rate (Lynch *et al*., 1999). As a result of the genetic characteristics and hypotheses outlined above, the magnitude of the average genetic load present in the population initially released is 1.65 lethal equivalents per individual. New mutations occur stochastically in every generation, with a mean of one mutation per genome, corresponding to the value most commonly assumed (Lynch *et al*., 1999). We assume multiplicative interactions for fitness and free recombination of all loci. Thus the presence and the accumulation of deleterious alleles is characterized in terms of the relative fitness of the individual, by using a genetic factor w_i to decrease the performances of the individual i, in a proportion $1 - w_i$ (see Appendix).

Extinction probabilities are investigated in several reintroduction scenarios, by using Monte Carlo simulations in which 1000 population trajectories are drawn over 30 generations (or 100 years) for the model including genetics and 100 generations for the demographic model.

RESULTS

In the following section, we compare populations with a low variance in fitness ('low-variability populations') with populations with a high variance in fitness ('high-variability populations').

Non-genetic model

Figure 1 shows the comparison of the effect of different levels of fitness variability on extinction with a simple model, in which the differences of fecundity (or survival) have no genetic basis. In this model, the generations do not overlap and fertility of each pair is drawn from a Normal distribution without any consideration for the genetic characteristics of the parents. The deterministic growth rate is higher than one ($\lambda = 1.2$), so demographic stochasticity caused by random fluctuations in sex ratio and in fertility is the only factor influencing extinction. It appears that the negative impact of demographic stochasticity is strongly enhanced by a high variability of fitness within the population.

Genetic model

In the case of non-overlapping generations, after a sufficient number of generations, the probability of persistence of released populations almost always increases with an increasing level of fitness heterogeneity, for different asymptotic deterministic growth rates and different numbers of released individuals (Fig. 2). The only exceptions concern very high growth rates ($\lambda_{det} > 1.2$) for which extinction probabilities are extremely low. The influences of two different levels of fitness variability are compared for a 'fast-growing' population and for a

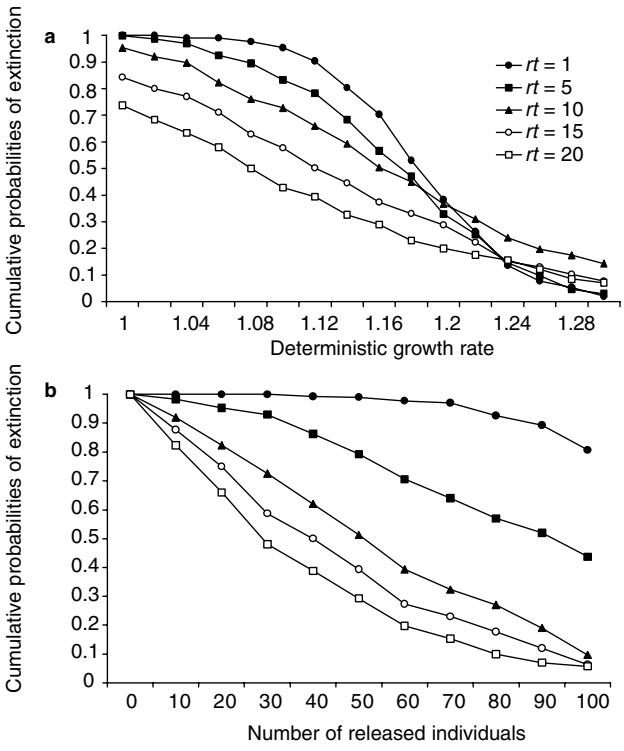

Fig. 2. Cumulative probabilities of extinction as a function of the deterministic asymptotic growth rate (a) and the number of released individuals (b). The number of released individuals in (a) is 20. The deterministic growth rate in (b) is 1.05. *rt* is the ratio (variance/mean) for the number of deleterious mutations per released individual. (Probabilities were obtained after 30 generations.)

'slow-growing' population (Fig. 3(a)). Whatever the growth rate, beyond 20 generations after release, extinction is higher for 'low-variability' populations (although extinction rates are high in all cases). In contrast, during the very first generations, for the 'fast-growing' population, extinction is higher when fitness variability is high. In order to allow a better understanding of the mechanisms that cause extinction, the changes of the mean relative fitness (b) are shown. It appears that the higher short-term extinction of 'high-variability' populations does not result from a differential value of mean fitness between 'high-' and 'low-variability' populations (Fig. 3(b)). After more than 20 generations, the higher fitness of the 'high-variability' population is due to a lower frequency of deleterious alleles and a higher rate of heterozygosity (not shown).

Comparison between species with overlapping and non-overlapping generations (Fig. 4) shows that the rate of decrease of fitness variability is similar in terms of generations: equilibrium is reached after eight generations for non-overlapping generations and after 80 years for overlapping generations (which correspond roughly to eight generations). But for a same number of generations the mean frequency of deleterious alleles decreases more in the overlapping case (Fig. 4(b)).

For populations with overlapping generations, when considering an equivalent annual growth rate (Fig. 5), the patterns of extinction differ between a long-lived species (generation time = 9.38 years) and a short-lived species (generation time = 3.08 years). In both cases, 'high-variability' populations exhibit a higher long-term persistence and a lower short-term persistence than 'low-variability' ones. But this short-term cost of increasing the variation in fitness is more substantial in the case of the long-lived species.

DISCUSSION

Our results suggest that the viability of a reintroduced population depends on the initial fitness variability. Moreover, the effect of fitness variance for maximizing population persistence is a function of the species' life-cycle. Long-term viability is always increased when fitness heterogeneity is high, but heterogeneity has a negative impact on short-term persistence, when the growth rate is high or when the generation time is large.

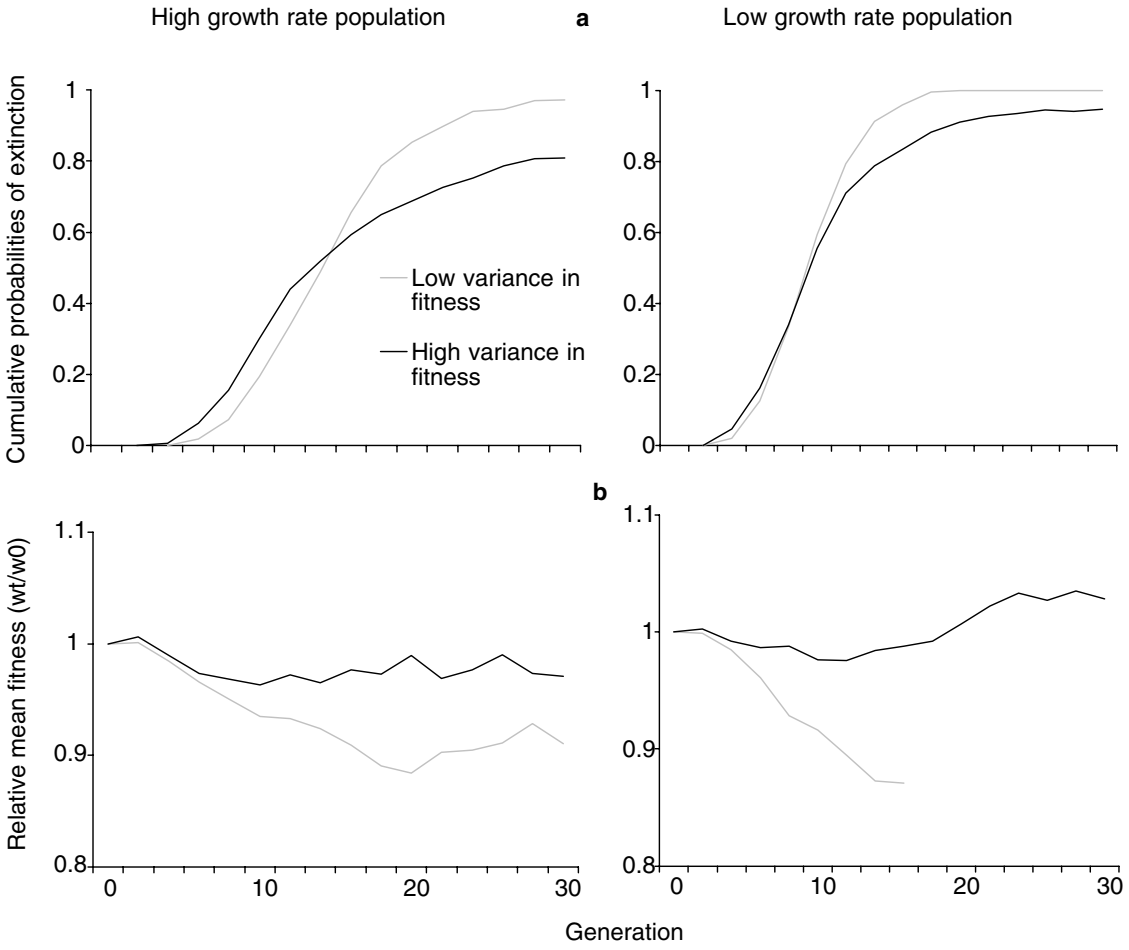

Fig. 3. Influence of the level of fitness variance of a released population of 20 individuals in terms of viability (a) and relative mean fitness (b). (*wt/w0*) represents the mean fitness at generation *t* divided by the mean initial fitness. The ratio (variance/mean) is 1 for the 'low-variability' population and 10 for the 'high-variability' population. The deterministic growth rate is 1.12 for the 'fast-growing' population, and 1.02 for the 'slow-growing' population.

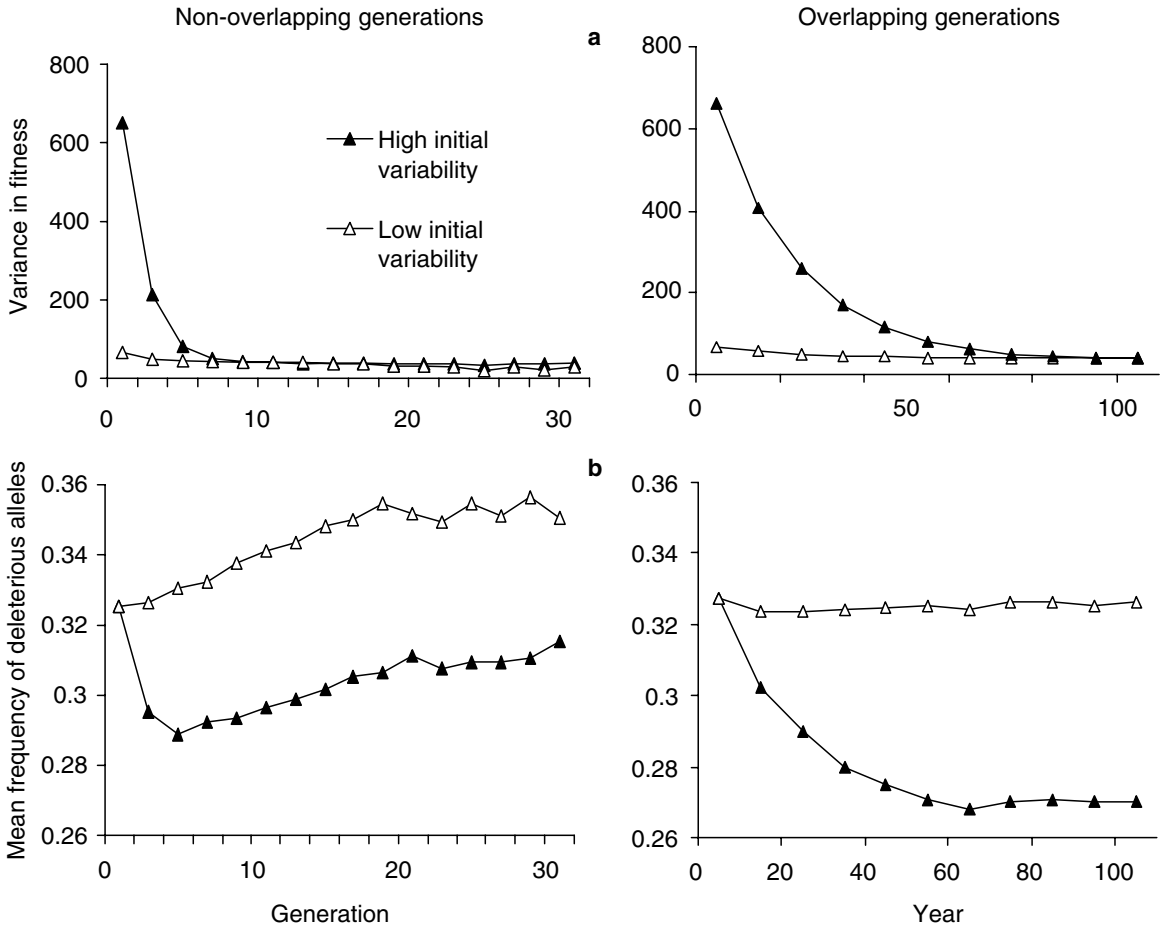

Fig. 4. Evolution of the mean and variance of the frequency of deleterious alleles per individual for species with overlapping and non-overlapping generations. For the population with overlapping generations, the demographic parameters used are those presented in the right side of Table 1.

Two mechanisms with opposite effects are mainly responsible for the different patterns of extinction obtained: purging of deleterious alleles and demographic stochasticity.

Purging of deleterious alleles

As emphasised by Lynch *et al*. (1995*b*), a precipitous reduction in population size does not necessarily reduce the frequency of deleterious recessive alleles causing inbreeding depression. Further, the genetic load due to the increase of homozygosity as well as the fixation and accumulation of mildly detrimental mutations and their interplay with demographic stochastic processes can contribute to rapid extinction. Our results suggest that the high fitness heterogeneity expected in a population built from several sources can accelerate the decrease of inbreeding depression through a faster purging process. Obviously, the efficiency of purging varies with many parameters, and particularly with those influencing the balance between the intensity of natural selection and genetic drift. This balance depends respectively on the selective effect of the deleterious mutations and on the effective size of the population (Kimura, 1983). As an example, Hedrick (1994) found that populations enduring continuous full-sib mating were unable effectively to purge deleterious recessive alleles when those alleles had a selective effect of $s = 0.0625$, a value close to that used in our study. However, in our case, where more than 20 individuals are released, and with a growing population, purging can be sufficiently efficient to slow substantially the accumulation of deleterious alleles of weak effect. Because demographic parameters are held constant in our models for each genotype, the change of the mean individual fitness within the reintroduced population can only be due to the variation of the mean frequency of deleterious alleles present in the genome and to the proportion of these alleles expressed as homozygotes. Whichever the life-cycle, 'high-variability' populations show a decrease in their mean frequency of deleterious alleles, while this frequency increases or remains stable in low-variable ones. These results for 'low-variability' populations are consistent with those from previous studies, which predicted that, for very small populations, an increase in the relative importance of random genetic drift should increase the likelihood of fixing future deleterious mutations (Lynch & Gabriel, 1990; Lynch *et al*., 1995*a*,*b*, 1999). However, rapid and

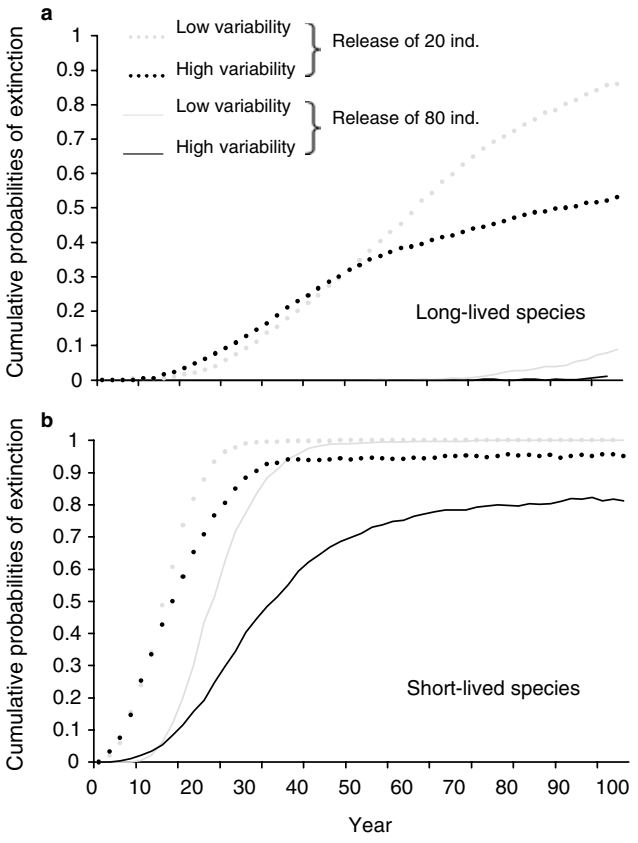

Fig. 5. Cumulative extinction probabilities of a restored population with overlapping generations. The ratio (variance/mean) is 1 for the 'low-variability' population and 10 for the 'high-variability' population; (a) long-lived species, (b) short-lived species (values of the demographic parameters for both life-history strategies are presented in Table 1).

efficient purging during the very first generations following release is a way to slow the course towards extinction (Figs 3(a) and 5). Particularly, in Figure 5(b), the release of 20 highly variable individuals is more advantageous on a long-term scale than the release of 80 low-variable individuals. For small reintroduced populations, it would help to increase the probability of sufficiently increasing the mean fitness and growth rate until the population size is large enough to reach an equilibrium mutation load.

Demographic stochasticity

Another consequence of a high variability of fitness is to increase demographic stochasticity. Figure 1 shows the long-term probabilities of extinction obtained with a non-genetic model, in which the only cause of extinction is demographic stochasticity (Lande, 1988; Boyce, 1992; Legendre *et al.*, 1999). Because the performance of a given individual is 'non-heritable' in such a model, the average rates of survival and fecundity do not change with time, but in the case of the 'high-variability' population the number of individuals that really contribute to the reproduction of each generation is lowered, so the uncertainty in the number of offspring is enhanced and the risk of extinction increases greatly.

Deterministic growth rate

The modelling of two populations that show different deterministic growth rates leads to different results in terms of the temporal pattern of extinction. The growth of the population can be split into two phases: in the first phase the mean population size is low, and demographic stochasticity plays the main role in the process of extinction. Populations that exhibit the higher fitness variability tend to go extinct more frequently than low-variable ones during this phase (see Fig. 3(a) for example). After 15 to 20 generations, the annual probability of extinction of initially highly variable populations begins to decrease while the annual extinction rate of 'low-variability' populations remains high. During this second phase, the non-extinct populations reach larger sizes (so demographic stochasticity has a lower impact), and the higher mean fitness of the 'high-variability' populations (resulting from a stronger purging process) allows them to persist longer than the 'low-variability' ones. As a result, the cumulative extinction probabilities of 'high-variability' populations become lower than those of the 'low-variability' populations after roughly 20 generations.

From a demographic view-point, when the mean fitness is low, increasing the variance in fitness is a smaller disadvantage for the population. As a consequence, the difference in extinction rates due to demographic stochasticity during the first phase between 'high-' and 'low-variability' populations is reduced with a low deterministic growth rate (Fig. 3). The pattern of extinction resulting from these interactions is summarized in Figure 6.

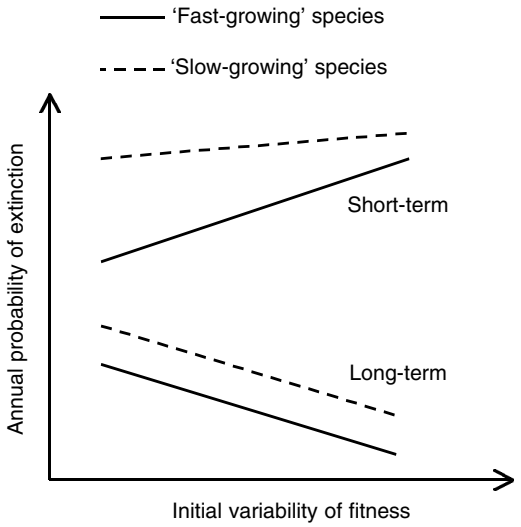

Fig. 6. Schematic pattern of the annual probability of extinction as a function of the initial fitness variability. These results are also dependent on the generation time: the 'short-time' relationship presented here for 'fast-growing' species is true only for relatively long-lived species.

Overlapping generations and generation time

Genetic processes do not always act with the same intensity according to whether the generations overlap or not (see Rogers & Prügel-Bennett, 2000 for genetic drift). And genetic results may be highly dependent on the structure of the models of populations with overlapping and non-overlapping generations. The results presented in Figs 2 and 3 and in the left part of Fig. 4 were obtained with a non-overlapping generation model in which fecundity is defined as the only parameter of fitness (as described in the Appendix). However, similar simulations were run with a non-overlapping generation model in which selection acts on survival only (as in Lynch *et al*., 1995*a*,*b*, not shown). The comparison between these two models suggests that increasing the variance of fitness has a larger effect on viability when selection acts on survival, relative to situations where selection acts on fecundity. Indeed, one given survival event depends on the genome of one individual, whereas fecundity depends on a pair of individuals, and is then averaged by fertilization. For overlapping generation models, fitness depends on both fecundity and survival, and the benefit of increasing fitness variation is intermediate between the two non-overlapping generation models described above. These results emphasize the importance of the type of model used and the sensitivity of the results to the processes which link the genetic part with the demographic part.

Because the global influence of a high variance in fitness on viability is dependent upon the balance between a short-term demographic mechanism and a long-term genetic mechanism, the generation time also has an impact on the pattern of extinction. Genetic processes are slower in the case of a species with a long generation time, while the events of survival and reproduction on which demographic stochasticity acts arise every time-step (year). Although these demographic processes scale with the generation time (for instance, the reproductive output per year is negatively correlated with the generation time in most real organisms as in our study), the balance between genetic and demographic forces is influenced by the generation time. In Fig. 5, the negative demographic effect of a high variance in fitness remains stronger than its positive genetic effect for a longer period in the case of the long-lived species (50 years ~ 5-6 generations, Fig. 5(a)), relative to the short-lived species (about 3 generations, Fig. 5(b)). The two types of life-history categories compared in Figure 5 are representative of many species encountered in birds (for example, within the Passeriformes and Charadriiformes orders for short- and long-lived species, respectively) and mammals (for example within the Rodentia and Artiodactyla orders for short- and long-lived species, respectively).

Mating system

Polygamy leads to the same results as monogamy in the case of species with overlapping generations, as well as in the case of those with non-overlapping generations. Extinction risk depends mostly on the numbers of reproducing females and males, which is more or less dependent on the fluctuations in sex ratio. Although the impact of such fluctuations is substantially reduced in the case of polygyny because the number of males has less importance (Lynch *et al*., 1995*a*; Legendre *et al*., 1999), this lower susceptibility does not change our results because a high fitness variance only increases the uncertainty in the number of offspring and does not directly affect the sex ratio. However some characteristics of the mating system, such as mate fidelity for monogamous species, should be taken into account when considering a realistic model (Bustamante 1996, 1998).

Release strategy

Another factor that interacts with the level of fitness heterogeneity is the age of the released individuals for long-lived species. When considering a species, in which the age of first breeding is n, the release of juveniles (individuals of age 0) implies no reproduction for n years. During this period, the process of purging consists in a differential mortality between the individuals of different fitness, but the heterogeneity of fitness decreases less

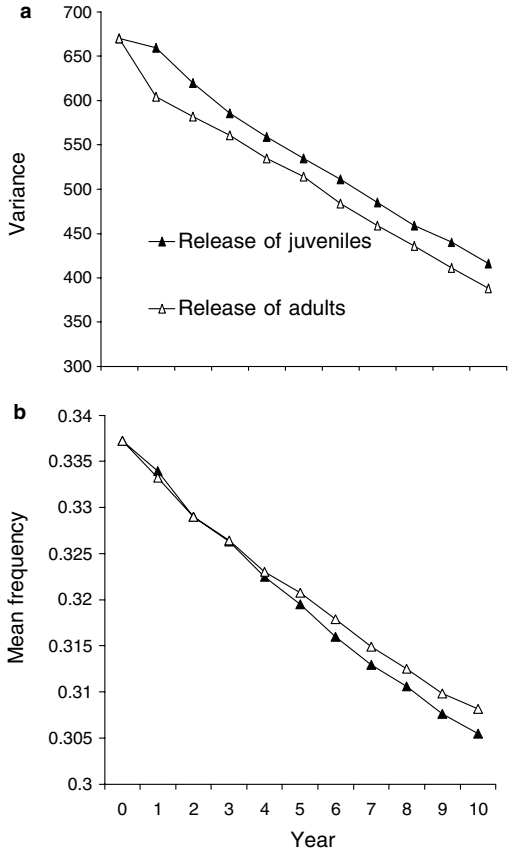

Fig. 7. Impact of the age of the released individuals on the evolution of the variance (a) and mean (b) of the frequency of deleterious alleles within a restored population (long-lived species, parameters on Table 1).

than for the adult release because there is no fertilisation (Fig. 7). The positive genetic effect of a high fitness heterogeneity is then enhanced when releasing young individuals relative to the release of adults. Such effect can modify the patterns of extinction when comparing release strategies, particularly for species which suffer from translocation cost: while, in the absence of genetic effects, releasing adults is advantageous in some species (Sarrazin & Legendre, 2000), releasing juveniles might become advantageous when taking into account genetic effects. This assumption is true only in the cases where selection is different before and after the release, such as in the case of release of captive-bred animals, or when considering local adaptation to the release site. Additional work is necessary on this aspect.

In the situation where a stock of animals available for restoration is made from several sources, such as different natural or captive populations, the fitness of each of these individuals in the release environment is a priori unknown. Although there has been some suggestion that releasing individuals from the populations most likely to have local adaptations to the release site may increase the chance of success (Griffith *et al*., 1989; May, 1991; Wolf *et al*., 1996; Montalvo & Ellstrand, 2000), an alternative approach consists of releasing individuals issued from several differentiated populations (Tordoff & Redig, 2001). Our results suggest that the release of individuals issued from multiple sources, which might imply a larger variance in the genetic load, is a way to enhance the process of purging of deleterious alleles present within the founded population. Moreover, releasing individuals from several sources is also a way to maximize the genetic diversity and to decrease the rate of inbreeding (May, 1991). Consequently, our results show qualitatively that, for monogamous and polygamous species, a strategy which consists of releasing a very heterogeneous population relative to a homogeneous one is efficient for populations that have a low growth rate. For populations with a high growth rate (especially if the generation time is long), such a strategy is more hazardous and can lead to higher extinction probabilities during the very first generations. These recommendations can be taken into account whatever the number of released individuals (Fig. 2(b)). Obviously, from a genetic view-point, the long-term persistence of a population is not only linked to the fixation of mildly deleterious genes but is also dependent on its ability to adapt to a changing environment. Such an aptitude is mostly determined by the genetic variability within the population (Gilpin & Soulé, 1986; Lande, 1988, 1995) and not by its fitness variability. However, efficient purging of deleterious mutations in a 'high-variability' population might contribute to maximizing the growth and population size and then indirectly minimizing the loss of genetic diversity.

Admittedly, a number of genetic processes, which may interfere with those described above, have not been considered in this study. For instance, although it has been considered sometimes as being less of a problem than inbreeding depression (Hedrick & Miller, 1992), the phenomenon of outbreeding depression may also have an influence on the viability of restored populations, owing to the loss of local adaptation (Roff, 1998) and/or the loss of intrinsic co-adaptation (Templeton, 1986; Lynch, 1991; Roff, 1998). All these genetic considerations may interfere with the method of release (low versus high initial heterogeneity), and their relative importance depends on particular details of the ecology and life-history of the species considered. Consequently, the more accurate strategy for re-establishing a population should always be assessed by a specific quantitative work.

Molecular techniques can provide some useful information on the genetic variation present within a heterogeneous population. However, such information is insufficient to predict the extent of variability of fitness expected for the population in a given environment. Since measurements of fitness are rarely available in natural conditions, it is difficult to make any quantitative link between the genetic variation present in a population and the heterogeneity of selective value expected. It is worthwhile to develop and use techniques that might help to assess the genetic qualities of the reintroduced individuals, in particular standard methods in quantitative genetics, using pedigree information, to disentangle phenotypic from genetic effects.

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APPENDIX

The genetic factor w_i that characterizes the relative fitness of the individual i is calculated as follows:

For species with non-overlapping generations: $w_i = (1 - h.s/2)^{n1} \cdot (1 - s/2)^{n2}$
with:
n1: number of heterozygotic deleterious mutations
n2: number of homozygotic deleterious mutations
s: magnitude of the deleterious effect on fitness ($s = 0.05$)
h: dominance of the deleterious alleles ($h = 0.3$).
The number of offspring from the pair (i, j) is then given by $w_i.w_j.f.$; where f is the basic fecundity ($f = 1.02$ for low growth rate populations and $f = 1.12$ for high growth rate populations).

For species with overlapping generations:
Genetic factor for fecundity: $wf_i = (1 - h.sf/2)^{n1} \cdot (1 - sf/2)^{n2}$
Genetic factor for survival: $ws_i = (1 - h.ss / ls)^{n1} \cdot (1 - ss / ls)^{n2}$
with:
sf: magnitude of the deleterious effect on fecundity ($sf = 0.025$)
ss: magnitude of the deleterious effect on survival ($ss = 0.025$)
ls: life expectancy at birth of the species.
The survival rate of the individual i of age a is then given by $ws_i.v_a$; where v_a is the basic survival rate for all individuals of age a.
The number of offspring from the pair (i, j) is then given by $w_i.w_j.f.$; where f is the basic fecundity (values are given in Table 1).