Releasing Adults versus Young in Reintroductions: Interactions between Demography and Genetics

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Abstract: *We integrated genetics and demography into population modeling in the context of species restorations, in which both the origin of released individuals and the management strategy may influence the success of introduction. Through an explicit individual-based simulation approach, we investigated the effects of the age of released individuals by exploring the relative merits of releasing juveniles or adults to establish populations. We included the effect of genetic variability responsible for inbreeding depression and mutational meltdown. Our general analysis uncovered an interaction between the age of founders and the extent of intrapopulation fitness variability, which substantially influenced the efficiency of selection in populations founded by juveniles and had subsequent positive consequences for long-term persistence compared with the case in which adults were released. We then applied the model to the case of the reintroduction of the Griffon Vulture (*Gyps fulvus fulvus*) to southern France, for which post-release data were available. The demographic aspects of this reintroduction were already analyzed and published, suggesting that it is more efficient to release adults than juveniles, despite an observed reduction of demographic parameters following the release of adults. In that context, the inclusion of genetic considerations qualitatively changes the conclusion, predicting reduced long-term extinction risk if juveniles rather than adults are released.*

Key Words: demographic stochasticity, inbreeding, population viability analysis, reintroduction, release strategy

Liberación de Adultos versus Juveniles en Reintroducciones: Interacciones entre Demografía y Genética

Resumen: *Integramos genética y demografía a un modelo poblacional en el contexto de restauración de especies, donde, tanto el origen de los individuos liberados y la estrategia de gestión pueden influir en el éxito de la reintroducción. Investigamos los efectos de la edad de los individuos liberados mediante la exploración de los méritos relativos de la liberación de juveniles o adultos para establecer poblaciones, por medio de un método de simulación basada en un individuo explícito. Incluimos el efecto de la variabilidad genética responsable de la depresión por endogamia y catástrofe mutacional. Nuestro análisis general descubrió una interacción entre la edad de fundadores y el grado de variabilidad de adaptabilidad intrapoblacional, que influyó sustancialmente en la eficiencia de la selección en poblaciones fundadas por juveniles y tuvo consecuencias positivas subsecuentes sobre la persistencia a largo plazo en comparación con el caso de la liberación de adultos. Posteriormente aplicamos el modelo a la reintroducción del Buitre Griffon (*Gyps fulvus fulvus*) en el sur de Francia, de la que había datos post-liberación disponibles. Los aspectos demográficos de esta reintroducción ya habían sido analizados y publicados, sugiriendo que es más eficiente liberar adultos que juveniles, a pesar de una reducción de parámetros demográficos observada después de la liberación de adultos. En ese contexto, la inclusión de consideraciones genéticas cambia la conclusión*

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cualitativamente, prediciendo la reducción en el riesgo de extinción a largo plazo si se liberan juveniles en lugar de adultos.

Palabras Clave: análisis de viabilidad poblacional, endogamia, estrategia de liberación, estocacidad demográfica, reintroducción.

Introduction

Although the relative contributions of genetic, demographic, and environmental stochastic processes to the extinction of small populations have not been clearly discriminated, it is well established that the persistence of such populations is tightly linked to these processes (May 1991). The negative impact of demographic stochasticity, caused by chance realizations of individual probabilities of death and birth events, is important only in small populations (Leigh 1981), whereas environmental stochasticity and catastrophes are likely to endanger both large and small populations (Shaffer 1987; Hedrick & Miller 1992; Lande 1993). The main genetic effect associated with small effective population size is the loss of genetic variation through drift and inbreeding. In the short run, the major consequence of this reduction of genetic variation is associated with 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 (Lande 1994; Lynch et al. 1995, 1999) and a subsequent decrease in fitness. These genetic processes are likely to substantially affect the persistence of populations, as suggested by recent theoretical (Hedrick 1994) and empirical studies (Newman & Pilson 1997; Bouzat et al. 1998; Saccheri et al. 1998).

Although the necessity for using multidisciplinary approaches in conservation has previously been emphasized (Soulé 1985), the integration among scientific disciplines, such as population dynamics and genetics, has generally been lacking in modern conservation research (Clarke & Young 2000). Similarly, the gap between conservationists and scientists slows down the improvement of practical conservation methods (Sarrazin & Barbault 1996). Here, we integrate genetics and demography into population modeling in the context of species restorations, where both the origin of released individuals and the management strategy may influence the success of an introduction (Griffith et al. 1989; May 1991; Wolf et al. 1996). We investigated the effects of the age of released individuals on the success of reintroduction by exploring the relative merits (in terms of population viability) of using juveniles or adults to establish populations. To investigate this question, we used an explicit demographic, individual-based simulation approach. The effect of genetic variability responsible for inbreeding depression and mutational meltdown was incorporated into the model to test whether genetic mechanisms can affect the relative efficiencies of these release strategies.

First we compared the two release methods—release of adults versus release of juveniles—in a general restoration context with different scenarios, in which we examined the impact of different life-history categories, initial conditions, and genetic and demographic parameters.

Second, we applied the model to the case of the Griffon Vulture reintroduction (*Gyps fulvus fulvus*) in southern France, for which post-release monitoring data were available. The demographic aspects of this reintroduction have already been analyzed by Sarrazin and Legendre (2000), suggesting that it is more efficient to release adults than juveniles, despite the observed reduction of demographic parameters following the release of adults. In that context, the addition of genetic considerations qualitatively changes the conclusion, leading to better long-term efficiency in the release of juveniles.

Methods

Life Cycle

We used a two-sex, individual-based model with overlapping generations. The N_0 individuals were released in the first time step (year). Then, in each year adult males and females paired according to their social mating system (monogamous or polygamous), with the success of reproduction (stochastically determined) depending on the genomes of parents. A distinction was made between situations where adults paired randomly in each year and situations with fidelity of the pairs from one year to the next. The sex of each newborn individual was randomly determined according to a 1:1 mean sex ratio. Each survival event was drawn from a Bernouilli function, with age-specific survival rates. To examine the effect of the age of founders for species presenting different realistic life-history traits, we investigated different types of life cycles, in terms of growth rate, age at first reproduction, and generation time. The demographic parameters used for these analyses were computed with a deterministic matrix model (computer program ULM; Legendre & Clobert 1995; Ferrière et al. 1996). These parameters are presented in Appendix 1.

Table 1. Demographic parameters of Griffon Vultures with environmental stochasticity.

Parameters	*Mean value*	*SD*
Juvenile survival (s_0)	0.858	0.3
Immature survival (s_1)	0.858	0.2
Immature survival (s_2)	0.858	0.1
Subadult survival (s_3)	0.987	0.05
Adult survival (v)	0.987	0.05
Productivity (P)	0.818	0.3
Proportion of breeders (α)	0.8	—
Age at maturity (a)	4	—

Because the type of density dependence we considered had little effect on extinction rates at the time scale considered, population size was truncated to the carrying capacity K in each year in the main simulations. Carrying capacity was equal to 1000 individuals in all figures except where we explicitly examined the effect of the carrying capacity. Truncation was made independent of the genetic qualities of individuals to keep constant selection coefficients. Negative events caused by environmental stochasticity were obtained by drawing survival and fecundity rates at age x in each year from a truncated normal distribution with a standard deviation σ_x. Following Sarrazin and Legendre (2000), this truncation resulted in reduced effective demographic rates. The values of demographic parameters and standard deviations for the case of the Griffon Vulture are presented in Table 1 (Sarrazin et al. 1994, 1996; Sarrazin & Legendre 2000).

Genetic Characteristics

The genome of each individual was explicitly represented as two series of 500 different diploid loci. Each of these two series could carry two types of alleles at each locus: a wild type and a deleterious allele. Each series corresponded to a given type of deleterious mutation (mildly deleterious mutations and lethal-sterility mutations). We selected the number of loci large enough to allow the segregation and accumulation of numerous detrimental mutations within the period considered without saturating the genome. The probability of transmission of a given mutation then depended on its selective effect s and its coefficient of dominance h, but it also was influenced by the background variance in fitness caused by other segregating loci which, in particular, decreases the effective population size and then reduces the efficiency of selection at this particular locus (Hill & Robertson 1966; Charlesworth et al. 1993). In simulations where changes in the genetic parameters were explicitly examined, we considered only one series of diploid loci.

In all cases, we assumed that all released individuals descended from large outcrossing populations. Thus, the initial frequencies of mildly deleterious and lethal alleles were given by the mutation-selection balance in large populations. Using these mean frequencies, we then stochastically determined the initial number of each type of deleterious alleles present in each founder from a Poisson distribution. During fertilization, the probability of transmission of each allele at each locus was given by the Mendelian rules. New mutations stochastically occurred in each diploid genome (Poisson-distributed) with a mean number of mutations per zygote per generation U. The genetic parameters were $s = 0.05$, $h = 0.3$, and $U = 1$ for mildly deleterious mutations and $s = 1$, $h = 0.02$, and $U = 0.05$ for lethal mutations (Simmons & Crow 1977; Drake et al. 1998; Lynch et al. 1999). We assumed multiplicative interactions for fitness (no epistasis) and free recombination of all loci (no linkage). The individual-based structure of the model allowed the alleles on the independent loci to evolve in interaction with the demographic characteristics of the population. Deleterious alleles acted at the individual level by decreasing the demographic rates of each individual. The number of homozygous and heterozygous deleterious mutations carried by a given individual was used to compute a genetic factor, representing its relative overall reproductive fitness. This factor was then used to decrease survival and/or fecundity rates of the individual (see details in Appendix 2). We investigated changes in genetic and demographic characteristics and probabilities of extinction in several scenarios of reintroduction by using Monte Carlo simulations in which 1000 population trajectories were drawn over 300 years.

Results

General Model

Each deleterious mutation induced a life-time disadvantage in individual fitness, according to its coefficient of selection s. This disadvantage could be expressed as a decrease in survival, fecundity, or both. In our simulations, we investigated situations where deleterious mutations acted on juvenile survival, fecundity, or both juvenile survival and fecundity, corresponding to the most commonly documented reductions in demographic traits (Frankham et al. 2002). For each of these scenarios, the life-time disadvantage in total individual reproductive fitness induced by each deleterious allele was equivalent (in the case where mutations act on both survival and fecundity, their effect was equally divided among these two components).

In all cases, we observed an increase of extinction probabilities compared with the demographic models that did not include genetic considerations. In cases where the genetic load was assumed to decrease juvenile survival or juvenile survival and fecundity, it caused a long-term advantage to the juvenile strategy compared with the adult strategy (Fig. 1). The population founded by juveniles tended to go extinct more during the first 40 years for demographic reasons—an absence of reproduction and lower survival rates in juveniles engendering smaller

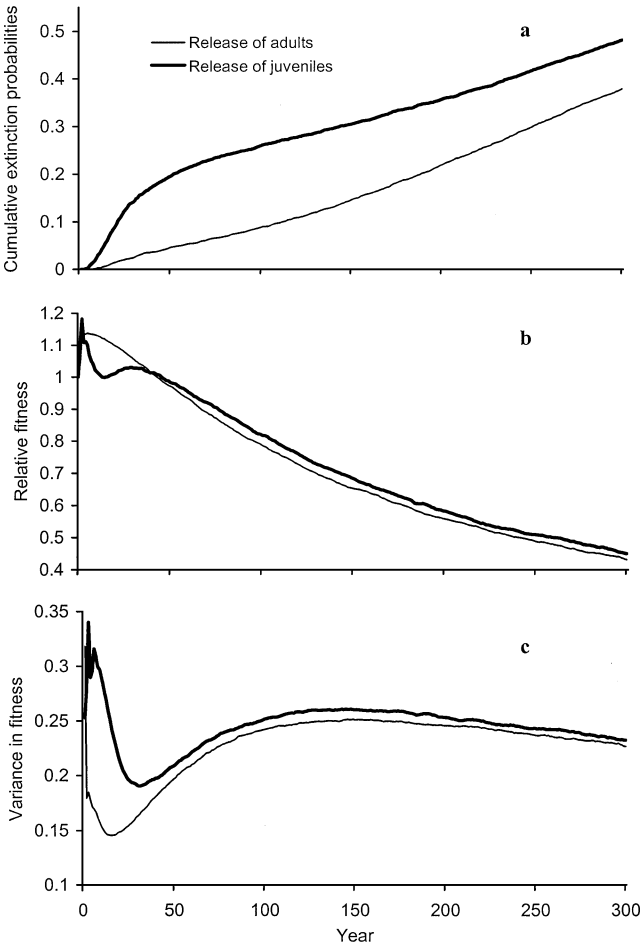

Figure 1. Influence of the release strategy on the change in fitness and extinction risk over 300 years: (a) extinction probabilities, (b) relative mean population fitness, and (c) interindividual variance in fitness. Model includes lethal and detrimental mutations acting on juvenile survival and fecundity (G3 life cycle $N_0 = 50$*). Environmental stochasticity with SD = 0.2 for all survival rates and for fecundity.*

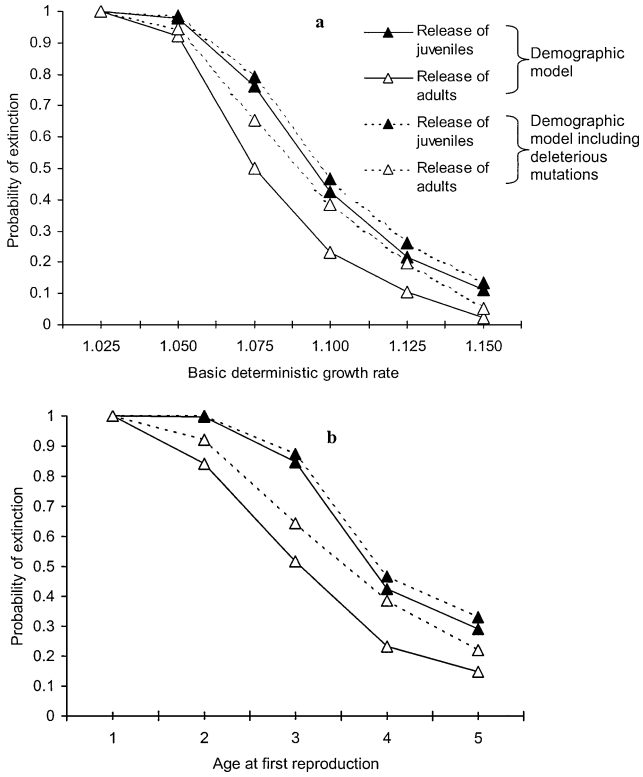

Figure 2. Influence of the release strategy on extinction risk for different types of life cycles: (a) impact of the deterministic growth rate and (b) impact of the generation time (life cycles presented in Appendix 1). Results presented after 300 years in the presence or absence of mutations acting on juvenile survival and fecundity ($N_0 = 25$)*. Environmental stochasticity with SD = 0.2 for all survival rates and for fecundity.*

population size—whereas the slope of the extinction curve (from 40 to 300 years after release) became lower for the juvenile strategy on a longer time scale (Fisher's test on regression slopes, $p < 0.001$). Concurrently, the average frequency of deleterious mutations was lower in the juvenile strategy after 30–40 years (not shown), leading to a higher mean population fitness (Fig. 1b). Variance in relative fitness was much higher in the case of the juvenile release, especially during the first years following release (Fig. 1c).

These differences in the sensibility of extinction risk to genetic factors obtained with the two strategies occurred in the presence or absence of environmental stochasticity and could be generalized to several life-history categories under different demographic and genetic scenarios.

In all cases, the genetic load had a stronger impact on the viability of populations founded by adults (Fig. 2). The difference became small, however, for species with high annual rates and species with long generation lengths. Extinction probabilities decreased with both an increasing number of released individuals and with an increasing carrying capacity, although these parameters did not identically affect short- and long-term viabilities (Fig. 3a & 3b). At the time scale considered, variation in K had a substantial effect only for unrealistically low values of K ($K < 300$ individuals). We performed additional simulations with a logistic function on fecundity and/or juvenile survival for density dependence (not shown) and uncovered no substantial effect of the type of density dependence on extinction. Similarly, the mating system (monogamy vs. polygamy) had little effect on extinction and did not effectively change our conclusion (all results are presented for monogamy).

The pattern of extinction rates obtained with different values of selection coefficient and coefficient of dominance were more complex (Fig. 3c & 3d), as a result of some differences in the contributions of the fixation load (maximum for relatively low values of s; Lande 1994)

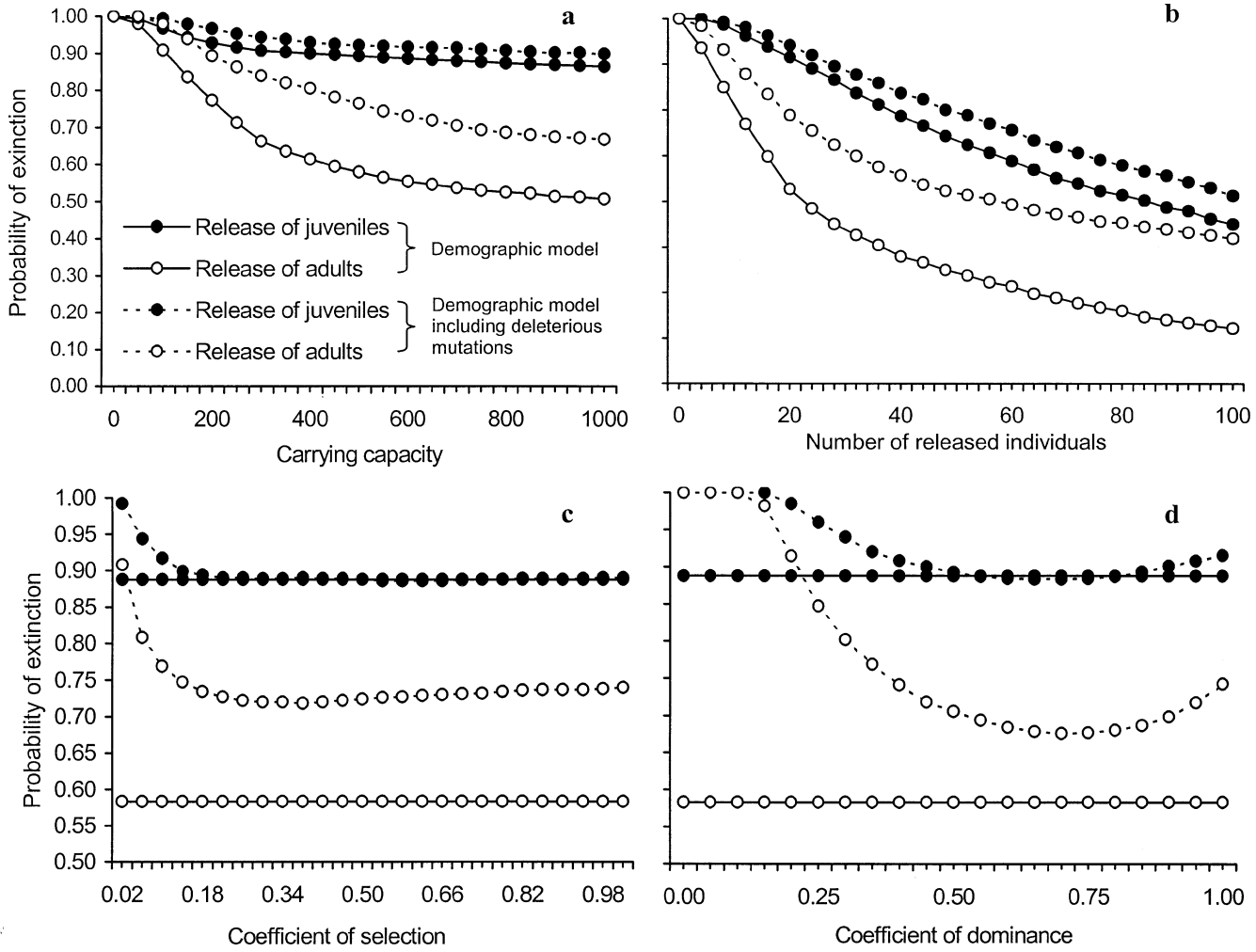

Figure 3. Impact of various genetic and demographic parameters on the relative efficiencies of the release strategies after 300 years: (a) influence of K, *(b) influence of* N_0 , *(c) influence of* s,h = *0.35, and (d) influence of* h,s = *0.1. The G3 life cycle is presented in Appendix 1. Environmental stochasticity with SD = 0.2 for all survival rates and for fecundity (*N_0 = *25 in all cases except [b]).*

and the segregation load (maximum for high *s* and *h*) to short- and long-term extinction risk. In all cases, however, the impact of the genetic load on extinction was stronger for the adult release than for the juvenile strategy.

Additional simulations in which mildly deleterious and lethal mutations were considered separately showed that lethal mutations had almost no effect on extinction, especially on a long-term scale, owing to the absence of accumulation of such mutations (Falconer 1989; Hedrick 1994; Kirkpatrick & Jarne 2000).

Case of the Griffon Vulture

We assessed the relative viability of the adult and juvenile release strategies in the case of the Griffon Vulture. Sarrazin and Legendre (2000) considered a permanent-release cost to fecundity and a short-term cost (1 year) to survival for adult release. These costs were estimated by previous analyses based on field data (Table 1; Sarrazin et al. 1994, 1996). The scenario of absence of cost in the case of juvenile release made by these authors is based on the assumption that the actual cost may be caused by some trauma associated with the adult translocation protocol. In the absence of further data, and to match the assumptions of Sarrazin and Legendre, we also considered this cost to exist only in the case of the adult release. As a first step, we investigated the hypothesis of an influence of the initial genetic load on this cost, especially on the short-term cost of survival. For that purpose, we compared the mean genetic load of the population at the release time and after 1 year. The observed pattern (i.e., a release factor of 0.752 for survival in the first year and no cost in the following years) could not be generated by the purging of the initial genetic load via a differential survival during the first year. Because the efficiency of the purging of deleterious mutations was expected to increase with an increasing extent of fitness variability among individuals (Couvet & Ronfort 1994), we performed additional

simulations with different extents of initial fitness variance. The extent of fitness variability within the released population was given by a Gaussian distribution on the number of deleterious mutations initially present in each individual. Our analysis indicated that the short-term release cost used by Sarrazin and Legendre (2000) could not be generated by selection against deleterious mutations only, even if the initial variance in fitness caused by deleterious alleles was 100 times the variance expected from a Poisson distribution. Similarly, the release ratio observed for fecundity (i.e., a permanent cost of 0.509 for the released as adults and no cost for the offspring) was not consistent with the type of genetic variability we considered. Therefore, the release cost of adults had no genetic basis, and the initial fitness variance was the Poisson variance.

For various numbers of released individuals considered, in the absence of deleterious mutations, the extinction probabilities obtained with our individual-based model were equivalent to those of Sarrazin and Legendre (2000) within 50 years. The absence of cost in juveniles was insufficient to compensate for their demographic disadvantage resulting from an absence of reproduction for 4 years and lower survival rates. The release of adults therefore led to less extinction than the release of juveniles in all cases. This result remained true on a 300-year time scale. As for the general results, however, we observed that the incorporation of genetic aspects into the model caused a relative long-term advantage to the juvenile strategy (Fig. 4a). Further, the release of juveniles became the optimal strategy on a 100- to 300-year time scale. As for the general results, this pattern was unchanged for scenarios in which mutations affected juvenile survival or juvenile survival and fecundity. When mutations affected only fecundity, we detected no effect on the relative efficiencies of the two strategies. Mate fidelity had a slightly positive effect (not significant after 300 years over 1000 simulations) on viability. It had no impact, however, on the relative efficiencies of the two strategies. These conclusions held when we added the influence of the environmental stochasticity, whatever the number of released individuals (Fig. 4b).

To account for selection before adult releases—the mortality of the weakest birds in captivity or in the natural source population—we also performed simulations in which the genetic load of reintroduced adults was reduced, a reduction corresponding to 4 years of selection on survival. Although the relative efficiency of the juvenile strategy decreased under these conditions, the results were not qualitatively different.

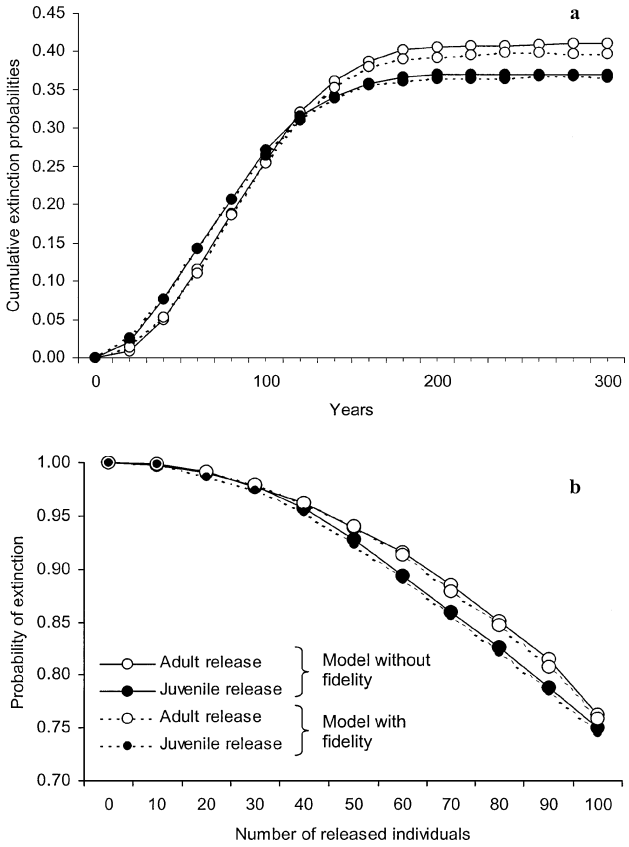

Figure 4. Influence of the release strategy on extinction risk in the Griffon Vulture in the presence of mutations acting on juvenile survival and fecundity: (a) $N_0 = 6$*, no environmental stochasticity, and (b) after 300 years, environmental stochasticity.*

Discussion

Our results indicate that populations founded by adults may generally be more affected by mutation accumulation than those founded by juveniles. The age of released individuals not only influences population dynamics from a demographic viewpoint but also has a substantial impact on the within-population extent of fitness heterogeneity. The variance in fitness in turn influences the process of purging of mildly deleterious alleles, which is more efficient when fitness variance is high (Couvet & Ronfort 1994). In our simulations, intrapopulation fitness variance decreased during the first years following release, due to the process of fertilization, which averaged the performances of reproducers (Fig. 1c). Variance then increased during the following years because inbred individuals (low fitness) appeared in the population, whereas noninbred individuals (high fitness) were still present. At a longer time scale, variance progressively decreases as founders disappear and as a proportion of the genetic load becomes fixed (Lynch et al. 1995).

Moreover, changes in fitness variance strongly interacted with the release strategy, especially during the first years following release. Whatever the life cycle considered, the release of juveniles (individuals of age 0) implies no reproduction for a_r years, a_r being the age at first

reproduction. During this period, efficient purging consists of a differential mortality between the individuals of heterogeneous fitness. By contrast, in the case of the adult release, reproduction occurs immediately after release, rapidly decreasing the intrapopulation fitness variance. Selection consequently operates on a more variable population (in terms of selective value) in the case of juvenile release, where reproduction is delayed, positively influencing the efficiency of purging and population mean fitness (Robert et al. 2002). This process occurred in all situations where mutations acted on juvenile or immature survival. We performed additional simulations with mutations similarly affecting survival rates at all age classes (juvenile + immature + adult), with the species life span as an indicator of compute annual reduction factors in survival. No substantial difference was detected compared with the case in which mutations acted on juvenile survival only.

Genetic load had a stronger impact on the relative efficiencies of the two strategies for short-lived species than for long-lived species (Fig. 2b). This result is mostly due to some difference in the magnitude of the impact of genetic deterioration, and in particular mutation accumulation, which becomes negligible for very long-lived species at the time scale considered (for the G5 life cycle, 300 years approximately represents 13 generations, whereas this time span represents 213 generations for the G1 life cycle). Because genetic processes operate on a per-generation basis (Falconer 1989), gradual genetic processes (as mutation accumulation) are expected to be more detrimental in a short-lived species than in a long-lived species in the same time span because of the larger number of generations elapsed. As emphasized by Gilligan et al. (1997), typical time frames of concern for captive propagation programs are 100–200 years, which typically represents <50 generations for most species of conservation concern. According to these authors, this represents a period that may be insufficient to detect substantial impacts of gradual genetic processes on population viability. Our results emphasize the necessity for a high level of specificity in answering such questions by showing that some genetic aspects may have a general qualitative impact on population dynamics but may remain of negligible effect on extinction for some categories of species within realistic conservation time frames.

The basic population growth rate also has an influence on the impact of genetic changes on extinction because it partly determines the time scale at which extinction occurs. Reintroduction projects generally involve relatively low numbers of released individuals, and a positive rate of increase is expected. In all scenarios considered, we observed a relatively rapid increase of population size. Extinction risk was therefore maximal during the short period following release and decreased as a consequence of population growth. Mechanisms acting over the long term (such as genetic mechanisms) had therefore less impact when population growth was high (Fig. 2a). More generally, various nongenetic factors—such as intrinsic population growth rate, initial population size, carrying capacity, extent of environmental stochasticity, frequency, and severity of environmental perturbations—may induce some variation in the relative contribution of short- and long-term extinction to overall extinction risk. The susceptibility of the population to genetic deterioration is likely to depend on the combination of these factors.

Sarrazin and Legendre (2000) developed both deterministic and stochastic demographic models (without formal genetic consideration) to predict the relative efficiencies of releasing juveniles versus adults in the case of the Griffon Vulture reintroduction in southern France, showing that it may be more efficient to release adults than juveniles, despite the observed reduction of demographic parameters following the release of adults. When applying the general approach including genetics that we developed above to this specific context, we observed that the juvenile strategy led to lower long-term extinction probabilities than the adult strategy, which contradicted the results obtained from the purely demographic model. This impact of deleterious mutations on the qualitative conclusion was largely a result of the variance effect described above. However, the presence of a release cost in the case of the adult release induced an additional relative advantage for the juvenile strategy.

The release cost had two main effects on population dynamics. From a demographic viewpoint, it decreased the short-term growth rate of populations founded by adults. From a genetic viewpoint, it further decreased the interindividual variance in individual performance. Because population size was relatively small, at least during the first years following release, both mean genetic load and mean inbreeding coefficient increased over time. Consequently, released individuals had on average a higher fitness than individuals of the following generations. The opposing effects of the genetically induced performance of the individuals released as adults and their nongenetic release cost reduced the intrapopulation variance in individual performance, affecting the efficiency of purging and long-term population fitness.

Demographic data on the reintroduced Griffon Vulture population in southern France suggest that pairs remain the same from one year to the next with an extremely high probability in this species (Sarrazin et al. 1996). From a demographic viewpoint, mate fidelity does not affect population viability at all. From a genetic viewpoint, it had a minor effect on our results.

Contrary to the more general results presented above, the Griffon Vulture results depended partly on the assumption that there is a demographic cost for individuals released as adults and no cost for those released as juveniles. As emphasized by Sarrazin & Legendre (2000), a reduction in survival and/or fecundity parameters is

characteristic of many reintroductions (Gogan & Barret 1987; Saltz & Rubenstein 1995). In several cases, this cost is higher for individuals released as adults than for those released as juveniles (Kleiman et al. 1991; Massot et al. 1994). If we consider a cost for individuals released as juveniles (not shown), as with adults we observe a reduction of the purging effect due to the negative covariance between the release cost and the individual genetic load. The purging effect is always better in populations founded by juveniles, however, because of the absence of reproduction during the first years following release.

For simplicity, we assumed multiplicative interactions for fitness (no epistasis) and free recombination of all loci (no linkage). Population genetic theory (Hill & Robertson 1966; Felsenstein 1974) and empirical results (Betancourt & Presgraves 2002) show that the efficacy of natural selection is generally limited by linkage. Thus, the consideration of linkage or nonrandom association is not expected to qualitatively modify the results in terms of the relative efficiencies of the release strategies, but it may engender a more detrimental effect of mutation accumulation for both release strategies. Similarly, synergistic or antagonistic epistasis is likely to influence the strength of selection. In particular, if deleterious mutations interact synergistically, they may be more efficiently removed by selection, which may result in a reduced load (Charlesworth 1990). However, no clear pattern of epistasis (synergistic or antagonistic) is apparent from empirical studies (Elena & Lenski 1997), and the short-term effect of epistasis on population dynamics is difficult to assess in the case considered here in which mutations are initially at the selection-mutation equilibrium and accumulate rapidly following a brutal bottleneck.

We have assumed that the frequency of mutations was at the mutation-selection balance, a plausible assumption in situations where individuals originate from large natural populations. This assumption is unlikely to be justified, however, in cases where individuals descend from captive breeding stocks. The effects of inbreeding we found therefore ignore previous inbreeding. A similar assumption is generally made by generic population viability analyses programs, which assume at time zero an outbred population in which individuals carry two unique alleles at a single locus. Admittedly, population history is important for accurately assessing the impact of inbreeding on the dynamics of the released population. In particular, fitness may evolve differently according to whether all individuals come from a single small stock or from different small, independent populations. Further, it should be carefully considered how the specific demographic parameters used in modeling are obtained (in particular in cases where demographic parameters are directly obtained from empirical measures of the population under consideration) to determine to what extent the impact of genetic deterioration is already included in the demographic rates used in modeling.

In both our general and the Griffon Vulture results, the influences of the life-history category and initial conditions emphasize the necessity for considering specifically and explicitly every aspect of the demographic processes when assessing the impact of genetics on the viability of restored populations and, more generally, on the viability of small populations. The consideration of every biological aspect having an effect on persistence is of course impossible in real cases, and one should therefore interpret results from population viability analyses with caution (Morris & Doak 2002). The consideration of some biological or ecological aspects that have not been considered in our study or in the study of Sarrazin and Legendre (2000) may change the conclusion. For example, for the adult release, captivity until age at maturity can constitute a useful period in which to acclimatize individuals to the release area (Bright & Morris 1994). In the case of the Griffon Vulture, preliminary analyses show that conspecific attraction plays a role in aggregation, potentially leading to a better settlement ability in individuals released as adults before the reproduction period than for juveniles (Sarrazin et al. 2000). The probability of establishment of the release population in the release site may strongly depend on such behavioral aspects that we did not consider.

Our general conclusions are consistent with the idea that genetic processes contribute to rates of extinction (Soulé 1987; Hedrick 1994; Mills & Smouse 1994; Lynch et al. 1999) and emphasize the influence of the operation of selection during inbreeding (Lande 1988) as well as its effect on viability. Sarrazin and Legendre (2000) have pointed out the potential role of management strategy in the success of reintroduction through a demographic approach. We uncovered the additional influence of genetic processes, underlining the necessity for more integration among the fields of demography and genetics in restorative conservation and management.

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Appendix 1. Theoretical demographic parameters used to compare species with different generation times and rates of increase.

	Life-cycle name				
Demographic parameter	*G1*	*G2*	*G3*	*G4*	*G5*
Juvenile survival (s_0)	0.2558	0.415	0.6	0.8	0.8
Immature survival (s_1)	—	0.5025	0.7	0.8	0.82
Immature survival (s_2, s_3)	—	—	0.75	0.9	0.95
Immature survival (s_4)	—	—	—	—	0.9726
Adult survival (s_a)	0.3	0.6	0.8	0.9445	0.982
Age at maturity	1	2	3	4	5
Annual individual fecundity (f)	3.125	2.64	1.1525	0.4	0.3
Deterministic growth rate	1.1	1.1	1.1	1.1	1.1
Generation time	1.41	3.34	6.25	13.80	23.73

	Life-cycle name					
	Lambda1	*Lambda2*	*Lambda3*	*Lambda4*	*Lambda5*	*Lambda6*
Juvenile survival (s_0)	0.725	0.75	0.77	0.8	0.825	0.835
Immature survival (s_1)	0.725	0.75	0.77	0.8	0.85	0.86
Immature survival (s_2, s_3)	0.85	0.86	0.87	0.9	0.92	0.925
Adult survival (s_a)	0.908	0.9135	0.9342	0.9445	0.9515	0.9525
Age at maturity	4	4	4	4	4	4
Annual individual fecundity (f)	0.3318	0.38	0.39	0.4	0.4163	0.458
Deterministic growth rate	1.025	1.050	1.075	1.1	1.125	1.15
Generation time	12.74	12.35	13.51	13.80	13.82	13.19

**Parameters were computed to obtain similar annual growth rates and equivalent generation times.*

Appendix 2

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

$$w_i = (1 - h_d \cdot s_d)^{nd1} \cdot (1 - s_d)^{nd2} \cdot (1 - h_l \cdot s_l)^{nl1} \cdot f_{bl}(nl2),$$

with s_d, h_d, $nd1$, and $nd2$, respectively, being the magnitude of mutational effect, coefficient of dominance, and number of heterozygous and homozygous mutations carried by i for detrimental mutations and s_l, h_l, $nl1$, and $nl2$, respectively, being the magnitude of mutational effect, coefficient of dominance, and number of heterozygous and homozygous mutations carried by i for lethal mutations. The f_{bl} is the function "homozygous lethal," defined by $f_{bl}(0) = 1$ and $f_{bl}(x) = 0$ for any $x \neq 0$.

For cases in which the genetic load is applied to juvenile survival, the deterministic juvenile survival rate of the individual i is then $w_i \cdot s_0$. In cases where the genetic load is applied to fecundity, the deterministic number of offspring for the pair (i,j) is $2w_i \cdot w_j \cdot f$, where f is the basic annual individual fecundity. In cases where the genetic load is applied to both juvenile survival and fecundity, the coefficient $(w_i)^{1/2}$ is used to reduce each demographic rate instead of w_i.

Queries

Q1 Author: Sarrazin et al. 2002: not cited in text; please spell out publisher's name in full.

Q2 Author: Use of periods in the equation and in the terms below OK? please replace with the appropriate mathematical symbol if necessary.