Conservation Genetics **2:** 363–378, 2001.

Captive breeding and the genetic fitness of natural populations

Michael Lynch & Martin O'Hely
Department of Biology, University of Oregon, Eugene, Oregon 97403, USA

Received 1 March 2001; accepted in revised form 20 April 2001

Key words: captive breeding, domestication selection, extinction risk, genetic load, hatchery, supplementation

Abstract

Many populations of endangered species are subject to recurrent introductions of individuals from an alternative setting where selection is either relaxed or in a direction opposite to that in the natural habitat. Such population structures, which are common to captive breeding and hatchery programs, can lead to a scenario in which alleles that are deleterious (and ordinarily kept at low levels) in the wild can rise to high frequencies and, in some cases, go to fixation. We outline how these genetic responses to supplementation can develop to a large enough extent to impose a substantial risk of extinction for natural populations on time scales of relevance to conservation biology. The genetic supplementation load can be especially severe when a captive population that is largely closed to import makes a large contribution to the breeding pool of individuals in the wild, as these conditions insure that the productivity of the two-population system is dominated by captive breeders. However, a substantial supplementation load can even develop when the captive breeders are always derived from the wild, and in general, a severe restriction of gene flow into the natural population is required to reduce this load to an insignificant level. Domestication selection (adaptation to the captive environment) poses a particularly serious problem because it promotes fixations of alleles that are deleterious in nature, thereby resulting in a permanent load that cannot be purged once the supplementation program is truncated. Thus, our results suggest that the apparent short-term demographic advantages of a supplementation program can be quite deceiving. Unless the selective pressures of the captive environment are closely managed to resemble those in the wild, long-term supplementation programs are expected to result in genetic transformations that can eventually lead to natural populations that are no longer capable of sustaining themselves.

1. Introduction

Managers of endangered populations face a double-jeopardy situation. On the one hand, failure to intervene when it is clear that a species is declining deterministically can virtually guarantee extinction. On the other hand, the use of captive propagation for supplementation purposes can result in genetic changes that may reduce the sustainability of a wild population. Captive environments can be radically different from natural habitats, and there is little question that their inhabitants can often rapidly undergo significant evolutionary change in morphological, behavioral, and physiological traits in ways that compromise fitness in a more natural setting (Kohane and Parsons 1988; Arnold 1995; Frankham and Loebel 1992; Ruzzante and Doyle 1993). These types of problems have become particularly apparent in the case of hatchery populations of salmonids (Waples 1991; Fleming and Gross 1993; Utter et al. 1993; Campton 1995; Reisenbichler and Rubin 1999), where conditions associated with captive rearing alter the selective pressures operating on fitness-related traits in a variety of ways. First, due to the absence of predators, provisioning of food, and medical treatment, selection can either be relaxed in hatchery populations or phenotypes that would otherwise be maladaptive in the wild may be selected for inadvertently. Second, managers of captive-breeding populations may intentionally select for phenotypes that are advantageous to their immediate needs, e.g., early adult return times, high juvenile growth rates, and/or rapid time to smol-

tification. Third, captive breeding populations with small genetic effective sizes will enhance the likelihood of accumulation of deleterious alleles by random genetic drift even in the absence of an altered selective environment.

Introgression from a genetically altered captive population can impose a genetic load on the wild population. Our purpose is to investigate whether this load can develop to a high enough level to significantly increase the risk of extinction of natural populations on time scales of relevance to conservation biologists. Three conditions contribute to the build-up of a genetic supplementation load. First, all populations harbor a genetic load from segregating deleterious mutations. Under constant conditions, this load eventually reaches a quasi-steady state value resulting from the balance between mutation, migration, selection, and random genetic drift. In principle, a natural population can recover from an excess segregational load after being isolated from a supplementation program, because beneficial (wild-type) alleles are still present and can be returned to high frequency by natural selection. Second, deleterious mutations can become fixed by random genetic drift in populations that remain sufficiently small over a sufficiently long time. Under these conditions, although an equilibrium load associated with segregating mutations still evolves, the wild population does not attain an equilibrium mean fitness level. Rather, a quasi-steady state rate of loss of fitness results from the recurrent fixation of deleterious alleles, the rate depending on the sizes of the wild and captive populations, the degree of relaxation of selection in captivity, and migration patterns. Third, selection for alleles that are beneficial in captivity but otherwise deleterious in nature can lead to the loss of wild-type alleles even in very large populations. Recovery from both types of fixation load may be difficult, requiring back or compensatory mutations or the replenishment of adaptive alleles from exogenous sources.

To explore the quantitative significance of these problems, we will consider the general situation in which two partially connected populations, one in the native setting and the other in captivity, are exposed to different selection regimes. This type of population structure covers a number of scenarios of practical significance with different motivations. For example, gene flow may be intentional and bidirectional, as in the case of augmentation programs whose goal is to increase the size of the wild population to a level that permits harvesting of the excess resource. A central question in this case is whether such management is consistent with long-term self-sustainability of the naturally breeding population. Alternatively, gene flow may be inadvertent and unidirectional, as with escapes from aquaculture programs in coastal waters. Because such programs often involve the use of stock specifically bred for attributes that enhance production in a captive setting, this type of gene flow may be especially harmful to the fitness of the recipient wild population. Our goal is to use population-genetic principles to develop a theoretical framework for the genetic impact of these and other types of migration structure on the genetic fitness of the natural population. The definition of natural (or wild) population becomes blurred in the face of recurrent gene flow from a captive population, and we operationally define it to be the population of individuals breeding in nature, regardless of previous ancestry.

Although there are substantial technical challenges in developing a quantitative theory for the genetics of two-population systems, where possible we have attempted to reduce our results to formulations that depend largely on parameters that are directly measurable and/or under the potential control of population managers, e.g., rates of gene flow, degree to which selection is altered in captivity, and relative sizes of the wild and captive populations. We will first evaluate the magnitude of the transient supplementation load that is likely to result from an increase in segregating mutations, and then consider the more permanent load resulting from fixations. In addition, we will provide some results on the time scales for the development of increased genetic load following the establishment of a supplementation program and for purging the load when such a program is ended.

Previous work on these topics has been limited. Byrne et al. (1992) attempted to model the genetic effects of hatcheries, but their model assumed that the summed genetic effects could be treated as a single locus, a rather unrealistic situation. Ryman and Laikre (1991), Waples and Do (1994), and Ryman et al. (1995) considered the influence of supplementation on the genetic effective size of a population (N), the implicit assumption being that population fitness is likely to be compromised in populations with low N. However, as we will show below, maximization of the effective size of a subdivided population does not necessarily minimize the genetic supplementation load. Adkison (1994) considered the consequences of gene flow on the evolution of a quantitative trait in a population subdivided into captive and wild breeding

pools, an issue that was taken up subsequently by Ford (in press). Some of the results from these latter two studies parallel those reported herein, although the analyses involving quantitative traits under optimizing selection are focused on genes with conditionally deleterious effects and assume populations that are effectively infinite in size.

2. The equilibrium supplementation load

Throughout, we will assume that individual fitness is a multiplicative function of the allele-specific fitnesses (within and between loci) and that the deleterious effects of mutant alleles are the same at all loci. Individual fitness in the wild, $(1-s)^k$, is then fully characterized by the number of deleterious alleles carried by the individual (k) and the reduction in fitness per mutation (s). This type of fitness function assumes an absence of epistatic fitness effects across loci, and with the small selection coefficients that we employ, the fitness effects of alleles within loci behave in an essentially additive fashion. To allow for reduced effects of deleterious mutations, individual fitness in the captive environment is defined to be $(1-\theta s)^k$, with $\theta = 0$ implying complete relaxation of selection. The order of events in the life cycle is assumed to be: mutation, followed by random mating, then selection, and finally migration. New mutations are assumed to be Poisson distributed among individuals, with u being the rate of mutation from a wild-type to a deleterious allele, and the rate of back-mutation being assumed to be negligible.

The fractions of breeders in the captive and wild populations that are derived from the home site are defined by the retention coefficients r_c and r_w, with $(1-r_c)$ and $(1-r_w)$ denoting the fractions of breeders that are immigrants from the alternative sites (Figure 1). It should be noted that the retention coefficients are functions of population sizes and productivities, which in turn can depend on genetic influences on fitness. Suppose, for example, that the number of adults breeding in captivity and in the wild are $N_c = 100$ and $N_w = 200$ respectively, with $r_c = 0.5$ and $r_w = 0.3$. This implies that 50 of the 100 individuals breeding in captivity are born in captivity and 50 come from the wild, and that 60 of the 200 wild breeders are derived from the wild and 140 from captivity. To fulfill such conditions, the 100 captive individuals must contribute 190 adults to the next generation, while the 200 wild individuals must contribute only 110.

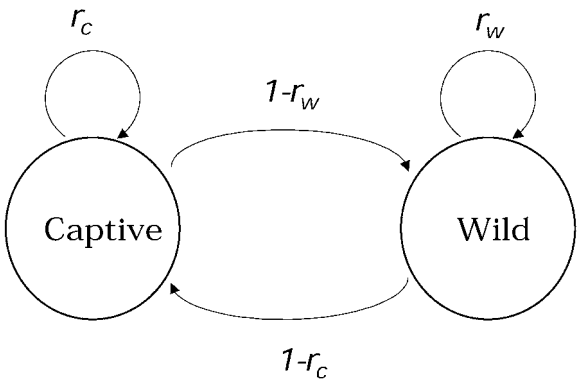

Figure 1. The migration scheme. r_c is the fraction of captive breeders that were born in captivity, and r_w is the fraction of wild breeders that were born in the wild. The populations need not be equal in size.

We first assume that both the captive and wild populations are sufficiently large that random genetic drift is of negligible significance. Under these conditions, deleterious alleles are not expected to go to fixation within the time frame of consideration, so the full genetic impact of the captive population derives from the magnified load resulting from inflated frequencies of segregating deleterious mutations. Given constant mutation and migration rates, a mutation-selection-migration equilibrium will then exist for the distribution of deleterious mutations carried by individuals, and this will define the equilibrium mean fitness of the populations in the two settings. Our initial results make no assumptions about the constancy of population sizes, so long as the minimum sizes are large enough to reduce the force of genetic drift to a negligible level. However, as noted above, population sizes may dictate the realizable bounds on the retention coefficients.

An important consideration with respect to the genetics of two-population systems is the degree of gametic-phase disequilibrium that inevitably results from restricted migration. Members of a captive population under relaxed selection are expected to carry deleterious alleles at multiple loci, and the efficiency of purging of such mutations after their export to the wild will depend on the degree to which they remain associated with each other. Positive associations magnify the efficiency with which deleterious alleles are removed because the carriers of such alleles have lower fitness than under random association. We

may then anticipate that the magnitude of the supplementation load will increase with increasing levels of recombination.

We have developed an analytical framework for the evolution of fitness in subdivided populations that fully accounts for linkage disequilibrium (O'Hely, in prep.), which we refer to as the Poisson-mixture approach, but the details are quite technical. Rather than present this method, we will describe results under the assumption of free recombination and gametic-phase equilibrium within both the captive and wild populations. The first assumption is a reasonable approximation for most multi-locus characters because most pairs of loci will lie on different chromosomes (with x chromosomes of equal length, the probability that a pair of random genes lies on the same chromosome is $1/x$). Although the assumption of gametic-phase disequilibrium is certainly violated, it yields some relatively simple analytical expressions for the upper bound on the expected supplementation load, and work that we will report elsewhere (O'Hely, in prep.) demonstrates that these expressions overestimate the true load by no more than a few per cent, justifying their use for our purposes.

For an arbitrary locus, let $q_c(t)$ and $q_w(t)$ denote the frequencies of the wild-type allele in the captive and natural populations at time t, with $1 - q_c(t)$ and $1 - q_w(t)$ denoting the frequencies of the alternative (deleterious) allele. Letting $W_c(t)$ and $W_w(t)$ denote the mean locus-specific fitnesses in the two populations, it can then be shown using standard approaches that after a generation of mutation, selection, and migration,

$$q_c(t+1) = (1-u)\left[\frac{r_c q_c(t)}{\sqrt{W_c(t)}} + \frac{(1-r_c)q_w(t)}{\sqrt{W_w(t)}}\right], \quad (1a)$$

$$q_w(t+1) = (1-u)\left[\frac{r_w q_w(t)}{\sqrt{W_w(t)}} + \frac{(1-r_w)q_c(t)}{\sqrt{W_c(t)}}\right], \quad (1b)$$

with

$$W_c(t) = [1 - \theta s(1 - q_c(t))]^2,$$
$$W_w(t) = [1 - s(1 - q_w(t))]^2.$$

Although the equilibrium allele frequency, q_w, can be obtained from Equations (1a,b), the general form of the resulting expression is complex and not very revealing. However, a number of relevant special cases reduce to a simple form.

2.1 *Complete relaxation of selection in captivity*

There are several plausible situations in which a captive population may be essentially released from the pressures of natural selection. For example, in most captive breeding programs, including hatcheries, every effort is made to minimize losses to predators and disease and to eliminate problems with food and mate acquisition. In addition, many captive breeding programs involving endangered species attempt to equalize the contributions of all members of the captive population to future generations (Lacy 1989; Allendorf 1993; Ballou et al. 1995). Although the desired outcome of such a breeding design is the maximization of effective population size, minimization of the loss of genetic variation, and avoidance of domestication selection, there is a significant genetic cost to equalizing family sizes – the complete elimination of the among-family component of selection.

When selection is relaxed completely in the captive environment ($\theta = 0$), the equilibrium mean locus-specific fitnesses are $W_w = [1 - s(1 - q_w)]^2$ and $W_c = 1$. In addition, it can be shown that the fraction of the time that an allele spends in the wild environment is $\omega = (1 - r_c)/(2 - r_c - r_w)$. The solution of Equations (1a,b) can then be expressed as

$$q_w \simeq 1 - \frac{u}{\omega s}, \quad (2a)$$

and

$$W_w \simeq [1 - (u/\omega)]^2. \quad (2b)$$

Assuming global gametic-phase equilibrium over all loci, the mean equilibrium fitness of the wild population simplifies to

$$\widetilde{W}_w \simeq e^{-U/\omega}. \quad (3)$$

where U is the genomic deleterious mutation rate, i.e., the sum of $2u$ over all loci. Thus, when selection is completely relaxed in captivity, the mean equilibrium fitness of the wild population decreases with increasing genomic mutation rate and increases with the fraction of time that an allele spends in the wild. For a closed wild population ($r_w = 1$), $\widetilde{W}_w = e^{-U}$, a well-known result (Haldane 1937; Bürger and Hofbauer 1994).

Rewriting Equation (3) as $\widetilde{W}_w = e^{-U}(1 - \widetilde{L}_s)$, $\widetilde{L}_s$ then defines the fractional reduction of fitness in the wild population caused by regular supplementation from a captive population. Rearranging, $\widetilde{L}_s = 1 - e^{U}\widetilde{W}_w$, showing that when selection is absent

in captivity, the equilibrium supplementation load is a simple function of three observable parameters (U, r_w, and r_c),

$$\widetilde{L}_s = 1 - e^{-U(1-\omega)/\omega}. \quad (4)$$

Because the equilibrium frequency of a deleterious allele is inversely proportional to s (from Equation 2a), whereas the reduction in fitness is directly proportional to s, the supplementation load is entirely independent of the selection coefficient. This is a useful result because s is ordinarily quite difficult to estimate. The genomic deleterious mutation rate, U, appears to fall in the range of 0.1 to 1.0, with the likely value for vertebrates being closer to 1.0 (Lynch et al. 1999).

The behavior of $\widetilde{L}_s$ when $\theta = 0.0$, given by the bold lines in Figure 2, illustrates several points. First, when the captive population is completely self-sustaining and closed to import ($r_c = 1$), the equilibrium load in the wild population is equal to one, regardless of the mutation rate, provided there is some gene flow to the wild ($r_w \neq 1$). This occurs because with no import from the wild, a captive population under relaxed selection builds up a load of deleterious mutations without limit, and even a small amount of gene flow eventually drives the fitness of the wild population to zero. Second, when both populations contribute equally to the breeding pools in both settings ($r_w = r_c = 0.5$), $\widetilde{L}_s = 1 - e^{-U}$. Thus, even with free and equal gene flow between both populations, a load is imposed due to the fact that half of deleterious alleles are free from selection each generation. With $U = 1$, the resultant load is an expected 63% loss of fitness in the wild population. Third, when the captive population relies entirely on imports from the wild ($r_c = 0$), such that no lineage spends consecutive generations in captivity, the load is minimized, but still can be appreciable, declining nearly linearly from $1 - e^{-U}$ when $r_w = 0$ to zero when $r_w = 1$.

2.2 *Intermediate levels of selection in captivity*

The preceding results assume the extreme case of complete relaxation of selection in captivity. A more likely situation is one in which the deleterious effects of mutant alleles are only partially masked in the captive environment ($0 < \theta < 1$). We have found no tractable equilibrium solutions for Equations (1a,b) for this mathematically challenging situation. However, a simple solution is possible for the extreme case in which the captive population is closed ($r_c = 1$) and the wild population is entirely derived from captive propagation ($r_w = 0$), i.e., no individuals born in the wild survive to breed in the natural environment. Such conditions represent a reintroduction program in which the transplants routinely fail to survive (not necessarily for genetic reasons), and provided $\theta > 0$, the mean equilibrium fitness expressed in the captive environment will then equal that expected for a single population in selection-mutation balance, e^{-U}. This implies an average of $U/(\theta s)$ mutations carried per individual and an equilibrium load on the wild population of

$$\widetilde{L}_s = 1 - e^{-U(1-\theta)/\theta}. \quad (5)$$

With $U = 1.0$, for $\theta = 0$, 0.2, 0.4, 0.6, 0.8 and 1.0 respectively, Equation (5) yields $\widetilde{L}_s = 1.00$, 0.98, 0.78, 0.49, 0.22, and 0.00 (given as the intercepts in the upper left panel of Figure 2).

A more complicated solution for the case of a closed captive-breeding population ($r_c = 1$) that allows for any r_w yields an equilibrium fitness for the wild population of

$$\widetilde{W}_w = e^{-\frac{U}{\theta}\left(\frac{1-r_w(1-\theta s)}{1-r_w(1-s)}\right)}. \quad (6)$$

with the load again being defined as $L_s = 1 - e^U \widetilde{W}_w$. As can be seen from Figure 2 (upper left), when the captive population is closed to import, the load on the wild population is nearly independent of the degree of purity of the wild population provided $r_w < 0.9$. Thus, for situations in which the wild population of a species experiences significant gene flow from a closed captive population, substantial build-up of genetic load can be expected even if selection against deleterious alleles is moderately high in captivity.

For more general conditions in which $r_c < 1$ and $\theta > 0$, we used the Poisson-mixture approach to obtain the equilibrium supplementation loads given in Figure 2. However, a very useful approximation applies provided $r_c < 0.8$ or so,

$$\widetilde{L}_s \simeq (1 - \theta)\left(1 - e^{-U(1-\omega)/\omega}\right). \quad (7)$$

Recalling Equation (4), this shows that for an arbitrary level of selection in captivity (θ), the equilibrium supplementation load is approximately $(1 - \theta)$ of that when selection is absent in captivity, again demonstrating that a substantial load is expected even with moderate selection in captivity, particularly if the captive population contributes significantly to the breeding stock in the wild (small r_w).

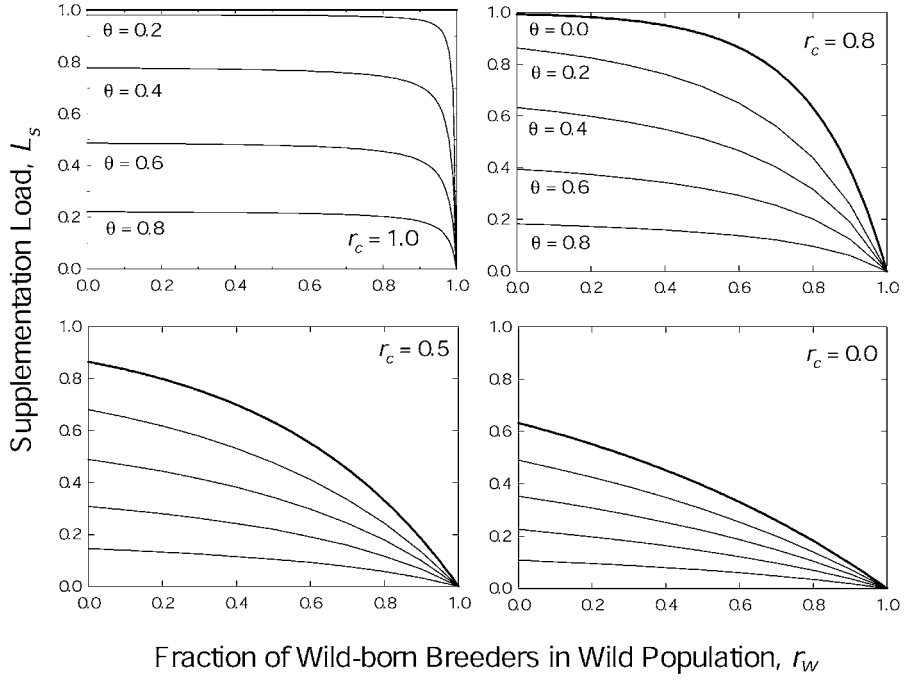

Figure 2. The equilibrium supplementation load as expressed in the natural environment, with effectively infinite sizes. Each panel provides results for a specific value of r_c, where $r_c = 1$ denotes a captive population for which the breeders are always born in captivity and $r_c = 0$ denotes a captive population for which the breeders are always born in the wild. Within each panel, the curves of increasing height denote results for decreasing selection in captivity, with $\theta = 0$ denoting completely relaxed selection. Note that when $r_c = 1$ and $\theta = 0$, the load is equal to one for all r_w. When $r_c = 1$, there is a slight dependency of the load on the selection coefficient (s) when $r_c > 0.9$, and we assumed $s = 0.025$. In all cases, the genomic deleterious mutation rate (U) is assumed to equal 1.0.

2.3 *Time to acquire the equilibrium supplementation load*

On what time scales are the equilibrium supplementation loads that we have outlined likely to be approximated? To obtain insight into this matter, we consider a base wild population with deleterious-allele frequencies assumed to be in mutation-selection balance (the numbers of deleterious mutations per individual being Poisson distributed with expectation U/s) and assume free recombination between all pairs of loci. An analytical approximation to the asymptotic approach to the new equilibrium load can then be obtained by noting that the effect of a supplementation program is to increase the equilibrium number of mutations per individual from U/s to approximately U/s_e, where

$$s_e = s[\omega + \theta(1 - \omega)] \tag{8}$$

is the effective selection coefficient against a deleterious allele experiencing the subdivided population structure (As a check on the validity of this approach, we note that defining $\widetilde{W}_w = e^{-Us/s_e}$ yields results that are essentially identical to those in Figure 2, provided $r_c \leq 0.8$). It can be shown that the deviation between initial and final equilibrium number of mutations is reduced by a fraction $(1-s_e)$ per generation, and using this relationship, the expected number of generations for the supplementation load to reach a fraction P of its final equilibrium level is found to be

$$t_a = \frac{\ln\{[\ln(1 - P\widetilde{L}_s)]/[U((s/s_e) - 1)] + 1\}}{\ln(1 - s_e)}. \tag{9}$$

To obtain more precise insight into this matter, we also used the Poisson-mixture approach, again starting with a base population with deleterious-allele frequencies assumed to be in mutation-selection balance, and iterating the two-population system with free recombination between all pairs of loci to generate the progressive change in the distribution of mutation numbers per individual. Because the results obtained by the Poisson mixture approach were in excellent agreement with this analytical approximation, we only present the latter.

For unconditionally deleterious alleles, the supplementation load approaches its equilibrium level at a rate that depends on the degree of gene flow between captive and wild populations (r_c and r_w) and on the extent to which selection is relaxed in captivity (θ), but for the most part, this dependence is quite weak (Figure 3). Generally speaking, 50% of the equilibrium supplementation load is typically reached within 10 to 20 generations, with the time to achieve 90% of the equilibrium being on the order of four-fold longer. Not very visible in Figure 3 is the fact that at very high r_c and very low θ (a nearly closed captive population with very relaxed selection), the times to attain the equilibrium load drop precipitously.

2.4 *Time to purge the equilibrium supplementation load*

Supplementation programs are sometimes viewed as temporary mechanisms to restore a natural population to a large enough size that the risk of extinction from demographic or environmental stochasticity is reduced to a low level (although few such programs have actually achieved this goal). The enhanced productivity of the captive population can yield a temporary boost in the size of a wild population despite the accumulation of a genetic supplementation load, and not until after elimination of the supplementation program will the accumulated genetic load be revealed as a reduction in the replacement rate of the natural population. Under the assumption that the system has initially evolved to its equilibrium fitness properties under supplementation, we now evaluate the time scale necessary for the recovery to wild-type fitness once the captive breeding program is ended.

In general, we expect the time to recovery to be somewhat faster than the time to acquisition of the segregational load, as the former occurs at the rate s (the magnitude of selection in the pure wild environment) whereas the latter occurs at the lower rate s_e. For the case in which selection is relaxed completely in captivity, Equations (1a,b) can be solved explicitly to yield the number of generations required for recovery to a fraction P of the expected equilibrium fitness in the absence of supplementation,

$$t_r \simeq -\frac{1}{s}\ln\left(\frac{s-[(\ln P)/U]}{s+[\omega/(1-\omega)]}\right). \qquad (10a)$$

A more general approximation that allows for some selection in captivity can be derived from the Poisson-mixture approach,

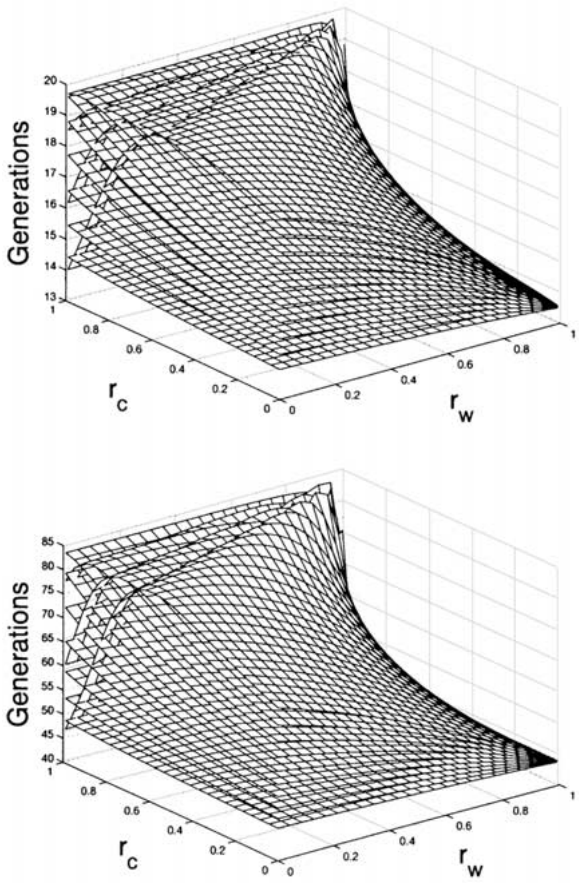

Figure 3. The time to achieve 50% (upper panel) and 90% (lower panel) of the equilibrium supplementation load for the situation in which all deleterious alleles segregating in the initial wild population are either mildly deleterious or neutral in captivity (i.e., there is no antagonistic selection). The base population is assumed to be in selection-mutation balance, with a genomic deleterious mutation rate $U = 1$ and selection against heterozygous and homozygous mutations being 0.05 and 0.10 respectively. All loci are assumed to be freely recombining. Within each panel, the uppermost set of results denotes completely relaxed selection in captivity ($\theta = 0$), and the descending sheets of results apply to increasing levels of θ in increments of 0.1.

$$t_r \leq -\frac{1}{s}\ln\left(\frac{\ln P}{U(1-s)+\ln \widetilde{W}_w}\right), \qquad (10b)$$

where $\widetilde{W}_w$ is defined as above. The results show that it can take several dozens of generations for a population to recover a substantial proportion of wild-type fitness, particularly if the captive population has contributed substantially to the natural breeding population in the past (small r_w) (Figure 4).

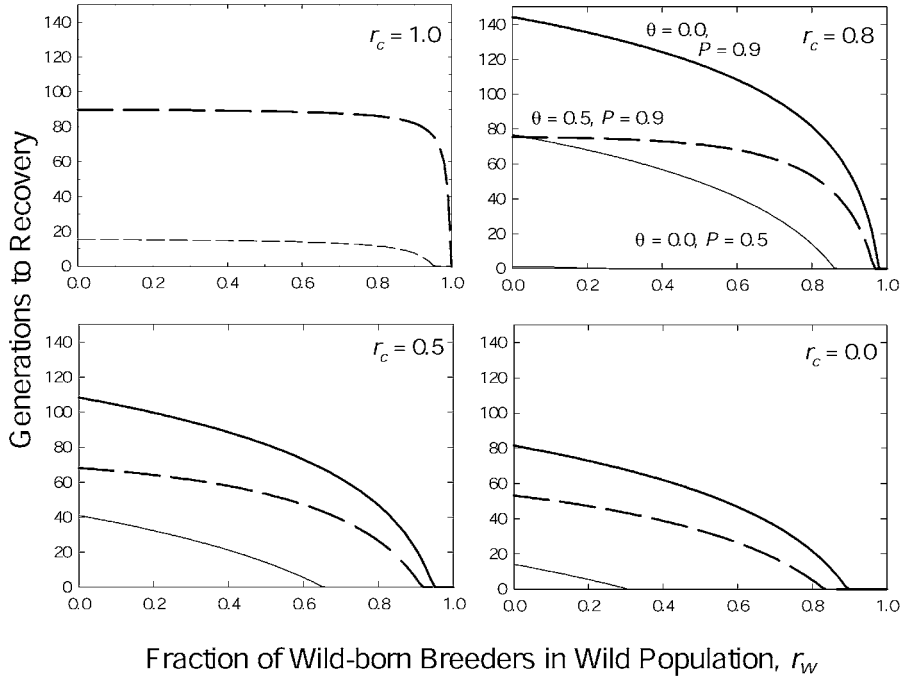

Figure 4. The time for recovery to a proportion P of original wild-type fitness after release from exposure to a captive breeding program. The solid lines refer to situations in which selection is relaxed completely in captivity and were obtained from Equation (15a). The dashed lines refer to situations in which selection is relaxed by 50% in captivity and were obtained by use of Equation (15b), with Equation (7) being used to approximate the equilibrium fitness in the presence of a captive population. These solutions assume a selection coefficient of $s = 0.025$; a two-fold increase or decrease in s would, respectively, decrease or increase the recovery times by approximately a factor of two. Bold lines denote 90% recovery, while thin lines denote 50% recovery. Note that where the recovery time is zero, the equilibrium fitness of the wild population in the presence of a captive population is greater than the recovery goal. For the case in which $r_c = 1$ and $\theta = 0$ (upper left panel), the equilibrium wild-population fitness converges to zero, so the expected recovery time is infinite (so not shown).

3. Antagonistic selection in the captive environment

Alleles that are deleterious in nature may sometimes be selectively advantageous in the captive setting. For example, certain feeding, social, or predator-avoidance behaviors that are energetically costly but essential in nature may be disadvantageous in captivity (Reisenbichler and McIntyre 1977). Such antagonistic (or domestication) selection can magnify the load on a natural population considerably, as mutant alleles that contribute to low fitness in the wild may be actively promoted, and even driven to fixation, by selection in captivity.

Here we assume additive gene action such that the three genotypes at a diallelic locus have fitnesses of 1, $1 - s$, and $1 - 2s$ in the wild population, but $1 - 2\alpha s$, $1 - \alpha s$, and 1 in the captive population, with $\alpha \geq 0$ being a measure of the magnitude of reversal in the selective advantage of an allele in captivity that is otherwise deleterious in nature. Implicit in this approach is the assumption that the deleterious effects of such alleles are expressed through quantitative traits involving morphology and/or behavior. Following Eyland (1971), the dynamics of allele-frequency change under this model can be described, to first order in s, as

$$\begin{aligned} q_c(t+1) = {} & q_c(t) + (1 - r_c)[q_w(t) - q_c(t)] + \\ & s\alpha q_c(t)[1 - q_c(t)] + s(1 - r_c) \\ & \{q_w(t)[1 - q_w(t)] - \alpha q_c(t)[1 - q_c(t)]\}, \\ q_w(t+1) = {} & q_w(t) + (1 - r_w)[q_c(t) - q_w(t)] + \\ & s q_w(t)[1 - q_w(t)] + s(1 - r_w) \\ & \{\alpha q_c(t)[1 - q_c(t)] - q_w(t)[1 - q_w(t)]\}. \end{aligned}$$

These formulae ignore mutation, which we regard as a second-order effect for the time scale under consideration, under the assumption that domestication selection generally involves the promotion of rare alleles segregating in the natural population.

There are three possible outcomes under this model. First, if the selective advantage experienced during the time that a gene spends in captivity is sufficiently low, the allele with deleterious effects in nature will be kept at a frequency not significantly greater than that at the outset. Second, a balance may be struck between the opposing forces of selection in nature and captivity, raising the frequency of the allele to a higher level in the wild population than expected under pure mutation-selection balance. Third, if the strength of selection in captivity is sufficiently high, the allele will be driven to fixation despite its disadvantages in the wild. Using standard techniques, but with considerable algebra, it can be shown that the requirements for the maintenance of a balanced polymorphism by antagonistic selection (the second possible outcome) are

$$\frac{1-r_c}{1-r_w-s|1-r_w-r_c|} < \alpha < \frac{1-r_c}{1-r_w+s|1-r_w-r_c|}. \quad (11)$$

Because we typically expect s to be quite small (on the order of 0.1 or smaller), the right and left sides of this inequality are nearly equal, implying that these conditions are quite stringent. This simple result indicates that the key determinant of whether an antagonistically selected allele will go to fixation is the ratio of the time spent in the wild to that spent in captivity, $(1-r_c)/(1-r_w) = \omega/(1-\omega)$. If α exceeds this ratio by even a moderate amount, the disadvantageous effects of an allele in nature will be offset by its advantages in captivity, and the allele will move towards fixation, eventually resulting in a permanent fitness loss in the wild.

To incorporate antagonistic selection into our expressions for the genetic supplementation load, it is necessary to partition the genomic deleterious mutation rate into components related to the classes of mutations with different expected fates. To simplify the presentation, we will consider the situation in which there are only two classes of mutations (distinguished by the subscripts $+$ and $-$), the first representing unconditionally deleterious mutations (arising at the rate of U_- per genome per generation, and kept at equilibrium frequencies defined by selection-mutation balance) and the second representing antagonistically selected mutations (arising at the rate of U_+, and driven towards fixation in the presence of supplementation). Under these conditions, the expected equilibrium fitness of the wild population becomes

$$\widetilde{W}_w = e^{-U}(1-\widetilde{L}_{s-})(1-\widetilde{L}_{s+}), \quad (12)$$

where $U = U_- + U_+$ is the total mutation rate to alleles that lower wild-population fitness, $\widetilde{L}_{s-}$ is the equilibrium load resulting from alleles that are deleterious (or neutral) in captivity (defined by using the expressions in the previous section, but replacing U by U_-), and $\widetilde{L}_{s+}$ is the equilibrium load resulting from fixations of alleles that are advantageous in captivity but deleterious in nature. The total supplementation load is defined as

$$\widetilde{L}_s = 1 - [(1-\widetilde{L}_{s-})(1-\widetilde{L}_{s+})]. \quad (13)$$

To obtain an expression for the load due to antagonistic selection, we note that with n loci subject to fixation, the fitness of the wild population associated with fixations at such loci will ultimately be reduced to e^{-2sn} of its previous level, i.e.,

$$\widetilde{L}_{s+} = 1 - e^{-2sn}. \quad (14)$$

Unlike the load resulting from unconditionally deleterious mutations, $\widetilde{L}_{s+}$ depends on the selection coefficient (s) as well as on the number of loci subject to strong antagonistic selection. Lack of specific information on these two parameters makes it difficult to surmise the magnitude of the genetic load that can result from antagonistic selection. However, it is clear that conditions do not have to be extreme for this load to approach its maximum value of one. For example, if the product $sn > 0.5$, domestication selection will reduce the wild population's fitness by more than 50%.

4. Influence of small population size

In the previous analyses, we assumed effectively infinite sizes for both the captive and breeding populations, ensuring that the fitness of the wild population will asymptotically approach an equilibrium level and that wild-type alleles will never be completely lost (except under strong antagonistic selection). However, in cases of endangered species management, the sizes of the wild and/or captive breeding populations can be small enough to raise the possibility of a permanent loss of wild-type alleles as deleterious mutations drift to fixation. A particularly serious situation can arise when the number of captive breeders is relatively small and the integration of their progeny into the

wild breeding population is high, as this causes the effective size of the total population to approach that of the captive segment, even when the actual number of breeders in the wild is quite high (Ryman and Laikre 1991; Waples and Do 1994; Ryman et al. 1995).

For finite populations, a quasi-steady state equilibrium supplementation load associated with segregating mutations is expected (as in the case of an effectively infinite population) but with random genetic drift playing a role along with the forces of mutation, selection, and migration. With a single panmictic population of effective size N_e, the magnitude of this segregational load attains a maximum at an intermediate size of $N_e \simeq 1/(2s)$ and converges to zero as $N_e \to 0$ (because most mutations are either rapidly lost or fixed) and to the usual large-population size approximation, $1 - e^{-U}$, as $N_e \to \infty$ (Kimura et al. 1963; Crow 1993; Lynch et al. 1995). However, these results do not imply that a minimum supplementation load arises at very small N_e, because when the population size is finite, the load resulting from segregating mutations incompletely describes the mean population fitness. Even though the load from segregating mutations may be relatively constant, the contributing loci experience allelic turnover as a result of fixations. As a consequence, the total genetic supplementation load continues to increase, eventually doing so at a relatively constant rate as the rate of fixation of deleterious alleles reaches a quasi-steady state level.

The extent to which fixation of deleterious mutations is likely to contribute to a permanent genetic supplementation load is a function of the time scale of consideration, the global effective population size (N_e), and the magnitude of selection (s_e). Because the mean time to fixation of a completely neutral mutation is approximately $4N_e$ generations (Kimura and Ohta 1969), one might surmise that it is safe to assume that fixation will not be a serious problem if the number of generations is smaller than N_e. However, it can be shown (through numerical integration of Equation 8.9.1 in Crow and Kimura 1970) that the mean time to fixation for a deleterious mutation (conditional on fixing) is less than N_e generations to a degree that depends on the magnitude of selection and on N_e. To make further progress, we develop the concept of the effective size for the two-population system. We then apply this definition, along with the effective selection coefficient (Equation 12), to the usual expressions for panmictic populations to evaluate the consequences of finite population size for the load associated with both segregating and fixed mutations.

The effective size of a subdivided population raises substantial technical difficulties (Chesser et al. 1993; Whitlock and Barton 1997), but a relatively simple expression can be obtained for the two-population case by defining N_e to be the reciprocal of the probability that two gametes drawn randomly from the total population are derived from the same adult in the previous generation (Ryman and Laikre 1991; after Crow and Kimura 1970). For two gametes to be derived from the same adult in the previous generation, they must both have come from the same subpopulation, and the metapopulationis derived from $2(N_w + N_c)$ gametes. There are $2N_w r_w$ gametes in the wild that come (in the previous generation) from the wild and $2N_w(1 - r_w)$ that come from captivity, while in captivity there are $2N_c(1 - r_c)$ gametes that come from the wild and $2N_c r_c$ that come from captivity. Two gametes that have come from the wild come from the same adult with probability $1/N_w$, and two gametes that have come from captivity come from the same adult with probabilty $1/N_c$. Therefore, the probability that a randomly selected pair of gametes will be derived from the same adult in the previous generation is

$$\frac{1}{4(N_w + N_c)^2}\left(\frac{(2N_w r_w + 2N_c(1 - r_c))^2}{N_w} + \frac{(2N_c r_c + 2N_w(1 - r_w))^2}{N_c}\right) \qquad (15)$$

so, inverting and writing $N_T = N_w + N_c$, $N_w = p_w N_T$, and $N_c = p_c N_T$,

$$N_e = N_T\left(\frac{[r_w p_w + (1 - r_c)p_c]^2}{p_w} + \frac{[r_c p_c + (1 - r_w)p_w]^2}{p_c}\right)^{-1}. \qquad (16a)$$

Although this construction strictly defines the inbreeding effective size, whereas we are concerned with the variance effective size, these two measures are essentially the same provided the population sizes remain constant (Caballero 1994). Equation (16a) provides a simple qualitative description of the effective size of a two-population system in terms of four measurable parameters (the two retention coefficients and the two breeding population sizes). This formula is similar in structure to that of Ryman and Laikre (1991) but provides a more explicit description of the mechanisms influencing the reproductive contributions of different population segments.

Several assumptions have been made in arriving at this expression. First, we have assumed that gene flow occurs at the gamete stage, whereas migration will be at the zygote stage in most situations with animals. Since zygotic migration simply postpones inbreeding by one generation, we do not expect this assumption to have a substantive effect on N_e. Second, we have ignored the differences among the sexes (assuming, for example, an unbiased sex ratio), nonrandom variation in family size, and potential temporal variation in population sizes. Excess variance in family sizes, temporal fluctuations in population density, and unequal numbers of breeding males and females can all substantially depress the local effective numbers of breeders below the actual numbers. Such reductions can be accommodated by making appropriate adjustments to N_c and N_w in the previous expressions for p_c and p_w (Lande and Barrowclough 1987; Caballero 1994). Finally, it should be noted that the dynamics of random genetic drift and inbreeding implied by Equation (16a) are not immediate, but are approached asymptotically following the initiation of a two-population system.

Some limiting cases of Equation (16a) are instructive. First, as the number of breeders in captivity dominates the total population size, i.e., $N_c \to N_T$, then

$$N_e \simeq \left(\frac{(1-r_c)^2}{N_w} + \frac{r_c^2}{N_c} \right)^{-1}. \qquad (16b)$$

Under these conditions, N_e converges on N_c as the captive population becomes increasingly closed ($r_c \to 1$), but converges on N_w as the captive breeding stock is increasingly derived from the wild ($r_c \to 0$). These results clearly show that the effective size of the total population depends on both the current sizes of the wild and captive populations and on their contributions to their respective breeding stocks in the following generation. Second, with equal migration between the two populations ($r_c = r_w = 0.5$),

$$N_e = \frac{4N_wN_c}{N_T}. \qquad (16c)$$

Under these conditions, if one population size is much smaller than the other, the total effective size will be close to four times that of the smaller population, e.g., if $N_c \gg N_w$, then $N_e \simeq 4N_w$. This shows that, all other things being equal, the effective size of the total population is generally much more dependent on the breeding size of the smallest component (Equation 16c is identical in structure to the expression for N_e for a single population with unequal numbers of males and females; Crow and Kimura 1970). Third, it can be shown that N_e is always less than or equal to N_T, and that for fixed N_w and N_c, the maximum N_e is attained when the combination of retention coefficients satisfies $(1-r_c)N_c = (1-r_w)N_w$. This latter condition implies equal numbers of immigrants into the captive and wild populations, i.e., an absence of any demographic boost to the natural population from the captive population.

With expressions for the effective size of a subdivided population and the effective selection coefficient in hand, we are now in a position to evaluate the consequences of finite population size for the development of the supplementation load. Following the methods developed in Lynch et al. (1995), an analytical expression for the expected supplementation load associated with segregating unconditionally deleterious mutations is developed in the Appendix. As $N_e \to \infty$, this expression converges to that given by Equation (3), and as in the case of a single population, the load associated with segregating mutations is maximized at an intermediate value of N_e.

The asymptotic rate of fitness decline resulting from fixations of new mutations (ΔW_m) is a function of the number of mutations arising in the total population per generation (UN_T), the probability of fixation of such mutations (u_f), and the reduction in natural-population fitness per fixation, which we approximate as $1-(1-s)^2 \simeq 2s$. Taking the product of these three terms,

$$\Delta W_m = 2UN_Tu_fs, \qquad (17)$$

where from Crow and Kimura (1970),

$$u_f \simeq \frac{e^{2N_es_e/N_T} - 1}{e^{4N_es_e} - 1}. \qquad (18)$$

As $N_T \to 0$, the fixation probability converges on $1/(2N_T)$, the expectation for an effectively neutral allele, at which point the recurrent fixation load is equal to its maximum, Us. For more general N_T, the highest possible value of ΔW_m is expected when selection is completely relaxed in captivity ($\theta = 0$) and the captive population is closed to import ($r_c = 1$), as both of these circumstances minimize the effective selection coefficient. Under these conditions, the solution of Equation (17) shows that if U is on the order of 1.0 and s is in the range of 0.01 to 0.05, ΔW_m can exceed 0.5% per generation if the total effective population size is on the order of a

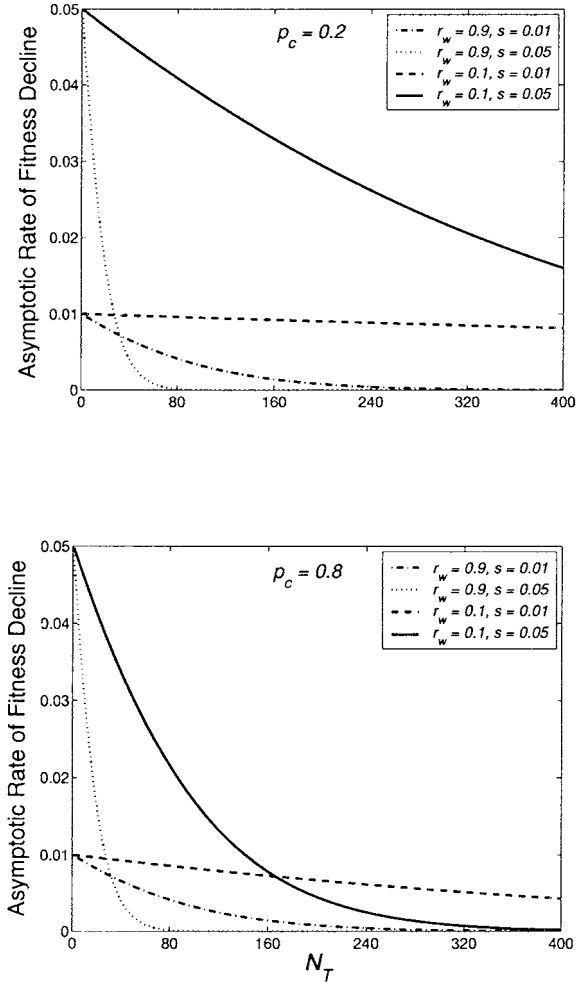

Figure 5. Expected asymptotic loss rates of wild-population fitness, given as a fractional loss per generation, obtained by use of Equation (17). It is assumed that the captive population is entirely closed to import from the wild and experiences complete relaxation of selection ($r_c = 1$ and $\theta = 0$), and that the genomic deleterious mutation is $U = 1$. Results are given for two different proportional sizes of the captive population (p_c).

few hundred breeders or smaller (Figure 5). Much higher estimates of ΔW_m arise when there is substantial gene flow from the captive to the wild population (small r_w) and when the fraction of the population in captivity (p_c) is large, as these conditions result in the productivity of the total metapopulation being dominated by members of the captive segment.

Finally, we note that although ΔW_m defines the eventual steady-state rate of decline in fitness for a finite population resulting from new mutations, there is also an initial, expected permanent loss in fitness owing to mutations pre-existing in the base population. This loss can in some cases be considerable even though ΔW_m may be small. To see this, recall that the equilibrium frequency of a deleterious allele in a large randomly mating base population is approximately u/s under the multiplicative model, where u is the genic mutation rate to deleterious alleles. Letting n be the total number of loci influencing fitness, the expected equilibrium number of deleterious genes carried per individual is then $2nu/s = U/s$. This shows that the average individual in an equilibrium population carries the equivalent of s^{-1} generations of new mutations. Thus, for large t, the cumulative permanent reduction in fitness resulting from fixations of deleterious alleles both residing in the base population and arising subsequent to the initiation of a supplementation program is

$$\Delta W(t) \simeq 1 - e^{-2UN_T u_f(1+st)}. \tag{19}$$

Further incorporating the equilibrium load due to segregating mutations, the mean fitness of the wild population can then be described as

$$W_w(t) \simeq e^{-U}(1 - \widetilde{L}_s)[1 - \Delta W(t)]. \tag{20a}$$

Rearranging, the approximate time for the mean genetic fitness of the wild population to be reduced to a fraction P of that in the original base population (e^{-U}) is

$$\overline{t} \simeq -\frac{1}{s}\left[1 + \frac{\ln[P/(1 - \widetilde{L}_s)]}{2UN_T u_f}\right]. \tag{20b}$$

This expression is most appropriate for $\overline{t}$ on the order of N_e or higher, as we have assumed that the time to settle into the equilibrium segregational variance and to fix pre-existing deleterious mutations is less than $\overline{t}$.

5. Discussion

We have focused on the situation in which a natural population is exposed to a recurrent pattern of gene flow from another population segment that is subject to an altered selective environment. In some respects, our results are related to previous theory concerned with the evolutionary origin of ecological specialization (Kawecki 1994; Holt and Gomulkiewicz 1997; Kawecki et al. 1997; Kirkpatrick and Barton 1997). The fundamental principle underlying all of these

studies is that selection operates on genes in a metapopulation system in proportion to their relative residence times and selective advantages in various habitats. Mutations that are deleterious in one particular habitat will nevertheless accumulate if selection is sufficiently relaxed (or is antagonistic) in other more productive habitats.

Our results clearly show that captive breeding programs that impose relaxed (or positive) selection on alleles that are otherwise deleterious in nature can have pronounced negative effects on the genetic fitness of natural populations on time scales no greater than a few dozen generations. Although we have not explicitly related the development of the genetic supplementation load to the dynamics of population size (see Ford (in press) for one approach to this problem), the potential consequences for the viability of a natural population can be understood relatively easily. Let R_{max} be the maximum number of adult progeny produced per adult per generation in the wild population (at low population density after ecological forces have been taken into account) prior to exposure to supplementation, with $R_{max} = 2$ being the minimum level of productivity necessary for a stable population (assuming a population with separate sexes). A supplementation load reduces this reproductive potential to $R_{max}(1 - L_s)$. Thus, if $R_{max} = 4$, an approximate value for many vertebrates, a load greater than 50% would eliminate the ability of the population to sustain itself demographically. In principle, habitat improvement could mitigate the consequences of a genetic supplementation load through an increase in R_{max}, e.g., a doubling in R_{max} would completely offset a 50% genetic supplementation load. On the other hand, habitat deterioration in the face of a growing genetic supplementation load can only put a wild population in a more precarious situation. The major findings of this study can be summarized as follows:

First, for a population subject to external supplementation, the loss of adaptation to the natural habitat is expected to be most severe when genes spend substantial fractions of time in the captive environment (a high value of r_c) and migrants from the captive population make a large contribution to the pool of breeding individuals in the wild (a low value of r_w) each generation. Under these conditions, which are common attributes of augmentation programs whose primary aim is to provide a demographic boost to a wild population, the captive population segment is the dominant contributor to the entire productivity of the species. In addition, when r_c is high, mutant alleles reside in captivity for many consecutive generations, allowing them to drift to higher frequencies before they experience selection in nature. The net result is the accumulation of alleles that are neutral (or beneficial) in captivity but deleterious in nature and the progressive transformation of the wild population to a genetic state such that complete collapse can occur in the absence of continued supplementation. Such a condition presents a particularly difficult management problem. On the one hand, the wild population has become entirely reliant on the captive population for subsidization. On the other hand, prolongation of the captive breeding program will only exacerbate the situation by promoting the accumulation of more deleterious mutations, particularly if domestication selection is a significant contributor to the genetic deterioration. As noted above, the time to reach a genetic state of non-self-sustainability can be delayed by habitat improvement (through an increase in R_{max}), but it can not be postponed indefinitely if fixations of deleterious mutations are occurring.

Second, although the supplementation load declines with decreasing degree of purity of the captive population, the magnitude of improvement is much less than proportional to r_c. For example, captive breeding programs that rely entirely on wild-born individuals each generation ($r_c = 0$) provide little advantage ($\sim$ 20%) over programs that rely on as much as 50% captive- born stock each generation (lower half of Figure 2). Although complete replenishment of the captive population from the wild each generation does minimize the segregational load, this load can still exceed 50% if selection is largely relaxed in captivity. Such a high load in the face of high gene flow is a consequence of the fact that mutant alleles still pass through the captive population every other generation, thereby experiencing substantial sheltering from selection.

Third, the degree of purity of the captive population (r_c) and its reproductive isolation from the wild population (r_w) interact synergistically to define the equilibrium segregational load. Although the supplementation load declines to only an intermediate level as r_c is reduced to a minimum, the load declines at an accelerating rate towards zero with increasing degree of purity of the natural population (r_w). For example, increasing r_w from 0.8 to 1.0 yields a much greater reduction in the load than increasing r_w from 0.6 to 0.8, especially if the captive population is

largely closed to import. If the captive population is quite pure ($r_c > 0.8$), the load on the wild population is nearly independent of the purity of the wild population until r_w exceeds a threshold value on the order of 0.8 or higher. Thus, from a management standpoint, situations in which the captive population is kept largely free from import put very high demands on the need to prevent gene flow into the wild population. This raises practical concerns because, in general, it is much easier to insure that some breeding members of the natural population are born in the wild than to ensure almost no contribution from the captive population. Moreover, because the goal of many supplementation programs is to provide a demographic boost to the wild population, the conditions that maximize the likelihood of such a boost (high r_c and low r_w) are precisely the ones that are likely to maximize the genetic supplementation load.

It is worth noting that all of the results summarized above are remarkably consistent with those obtained by Ford (in press), who modeled the fitness consequences of a single quantitative trait under stabilizing selection for different phenotypic optima in two effectively infinite subpopulations. Under this type of quantitative-genetic model, the fitness of a particular allele depends on both the environmental setting and on the genetic background, and quite unlike the situation that we modeled, no allele is unconditionally deleterious or advantageous in any environment. That the Ford model yields estimates of the segregational load that are quantitatively similar to those reported here suggests strongly that our results are not a peculiar feature of one specific genetic model but rather a general outcome of divergent selection in two environments.

Fourth, although most of our results were derived under the assumption that the effective population size is large enough to safely ignore random genetic drift, virtually all captive breeding programs will enhance the likelihood of fixation of unconditionally deleterious mutations through reductions in the effective population size and/or the effective selection coefficient. Moreover, it is important to note that substantial genetic deterioration resulting from fixations does not require the input of new mutations, as most populations are expected to harbor the equivalent of several dozens of generations of mutation resulting from prior input.

Fifth, alleles that are deleterious in nature but favored in captivity can become fixed in large as well as small populations. Indeed, it is possible that the rate of genetic deterioration resulting from via domestication selection may increase with population size, as larger populations are more likely to harbor rare conditionally advantageous alleles. Thus, it is an open question as to whether captive populations with large effective sizes are less likely to compromise the fitness of a recipient wild population.

In principle, a wild population can readapt to the natural environment after it has been released from a supplementation program. However, if the prior supplementation load is so high that the population is unable to replace itself on a per-generation basis, recovery will require that the rate of readaptation offset the consequences of demographic decline (unless, as noted above, ecological modifications can be made to elevate R_{max}). Even if the primary source of genetic deterioration is simply an elevation in frequencies of deleterious alleles, a return to wild-type fitness can require several dozens of generations, especially if the wild population had previously experienced substantial gene flow from the captive population (Figure 4). In this case, the rate of recovery will depend primarily on the average selection coefficient against the deleterious alleles that had accumulated under the previous conditions of relaxed selection. If, on the other hand, a significant fraction of the load is associated with fixed mutations, the time to recovery may be substantially greater, as this will require the input of new back or compensatory mutations and/or the importation of wild-type alleles from alternative population sources.

Throughout, we have assumed constant migration rates among populations, but even under the most carefully managed systems r_w and r_c are likely to vary from generation to generation due to environmental stochasticity. For example, the breeding segment of the wild population may be dramatically reduced in some years by natural fluctuations in the environment and/or unforeseen human disturbance. For populations with N_e sufficiently large to avoid fixations, the expressions that we derived under the assumption of constant migratory patterns should still yield a close approximation to the long-term average load provided the time-average values of r_w and r_c are utilized. In this case, because the expected frequency of a deleterious allele is a function of the long-term average fraction of time spent in alternative environments over the (many generation) life-time of the gene, temporal fluctuations should not alter the long-term average allele frequency (although temporal variations around

this average will occur). However, in the case of small populations where drift plays an important role, we expect that the fixation probabilities will depend on the long-term distribution of the product $N_e s_e$ (Equation 18), noting that variation in r_c and r_w introduce nonindependent fluctuations in both N_e and s_e.

In summary, the consequences of gene flow from domesticated populations raise serious concerns about the use of supportive breeding programs to enhance the ability of a natural population to sustain harvesting (Larkin 1980; Cuenco et al. 1993) or to enhance genetic diversity (Kapuscinski and Lannon 1984; Wohlfarth 1993). While there may be good reasons for short-term efforts to boost the size of wild populations with propagules from a captive stock (Olney et al. 1994), long-term supplementation programs appear to be incompatible with the permanent maintenance of self-sustaining wild populations, unless the two population segments are kept in a state of long-term reproductive isolation.

Acknowledgements

This work has been supported by a grant to ML from the Oregon Department of Fish and Wildlife. We thank M. Ford, K. Kostow, K. Higgins, S. Ratner, R. Waples, and an anonymous reviewer for helpful comments.

Appendix

Once N_e and s_e have been established, methods from Crow and Kimura (1970) may be used to determine the probabilities of and time until fixation or loss of a mutant allele. Noting that the initial frequency of a new mutant allele is $1/(2N_T)$, where N_T is the total census size of the metapopulation, formulae similar to Equations (A3), (A7) and (A12) from Lynch et al. (1995) can be derived. The expected cumulative segregational load caused by a mutant allele during its sojourn through the metapopulation is

$$L = 2(1-u_f)\times\left[-e^{4N_e s_e}\left(\mathrm{Ei}(4N_e s_e)-\mathrm{Ei}\left(4N_e s_e\left(1-\frac{1}{2N_T}\right)\right)\right)-\ln\left(1-\frac{1}{2N_T}\right)\right]+2u_f\left[\mathrm{Ei}\left(4N_e s_e\left(1-\frac{1}{2N_T}\right)\right)-\ln\left(4N_e s_e\left(1-\frac{1}{2N_T}\right)\right)-\gamma\right],$$

where u_f is defined by Equation (18), Ei is the exponential integral, and γ is Euler's constant. The mean time to absorption of a newly arisen mutant allele is

$$\bar{t}_a = \frac{L}{2s_e}+\frac{1-u_F}{s_e}\left[\mathrm{E}_1\left(\frac{4N_e s_e}{2N_T}\right)+\ln\left(\frac{4N_e s_e}{2N_T}\right)+\gamma\right]+\frac{u_F}{s_e}\left[e^{4N_e s_e}\left(\mathrm{E}_1\left(\frac{4N_e s_e}{2N_T}\right)-\mathrm{E}_1\left(4N_e s_e\right)\right)+\ln\left(\frac{1}{2N_T}\right)\right]$$

where E_1 is the exponential integral of order one. From these two expressions, the equilibrium segregational fitness can be calculated as

$$\left(1-\frac{L}{\bar{t}_a}\right)^{UN_T\bar{t}_a}. \quad (21)$$

References

Adkison MD (1994) *Application of Mathematical Modeling to Problems in Salmon Biology and Management*. Ph. D. Dissertation, University of Washington, Seattle.

Allendorf FW (1993) Delay of adaptation to captive breeding by equalizing family size. *Conserv. Biol.*, **7**, 416–419.

Arnold SJ (1995) Monitoring quantitative genetic variation and evolution in captive populations. In: *Population Management for Survival and Recovery* (eds. Ballou JD, Gilpin M, Foose TJ), pp. 295–317. Columbia University Press, New York.

Ballou JD, Gilpin M, Foose TJ, eds. (1995) *Population Management for Survival and Recovery*. Columbia University Press, New York.

Bürger R, Hofbauer J (1994) Mutation load and mutation-selection-balance in quantitative genetic traits. *Journal of Mathematical Biology*, **32**, 193–218.

Byrne A, Bjornn TC, McIntyre JD (1992) Modeling the response of native steelhead to hatchery supplementation programs in an Idaho river. *North American Journal of Fisheries Management*, **12**, 62–78.

Caballero A (1994) Developments in the prediction of effective population size. *Heredity*, **73**, 657–679.

Campton DE (1995) Genetic effects of hatchery fish on wild populations of Pacific salmon and steelhead: What do we really know? *American Fisheries Society Symposium*, **15**, 337–353.

Chesser RK, Rhodes EO, Sugg DW, Schnabel A (1993) Effective sizes for subdivided populations. *Genetics*, **135**, 1221–1232.

Crow JF (1993) Mutation, mean fitness, and genetic load. *Oxford Surv. Evol. Biol.*, **9**, 3–42.

Crow JF, Kimura M (1970) *An Introduction to Population Genetics Theory*. Harper & Row, New York.

Cuenco ML (1994) A model of an internally supplemented population. *Transactions of the American Fisheries Society*, **123**, 277–288.

Eyland EA (1971) Moran's island migration model. *Genetics*, **69**, 399–403.

Fleming IR, Gross MR (1993) Breeding success of hatchery and wild coho salmon *(Oncorhynchus kisutch)* in competition. *Ecological Applications*, **3**, 230–245.

Ford M (in press) The effects of selection during supportive breeding. *Conserv. Biol.*

Frankham R, Loebel DA (1992) Modeling problems in conservation genetics using captive *Drosophila* populations: rapid genetic adaptation to captivity. *Zoo Biology*, **11**, 333–342.

Haldane JBS (1937) The effect of variation on fitness. *American Naturalist*, **71**, 337–349.

Holt RD, Gomulkiewicz R (1997) How does immigration influence local adaptation? A reexamination of a familiar paradigm. *American Naturalist*, **149**, 563–572.

Kapuscinski ARD, Lannan JE (1984) Application of a conceptual fitness model for managing Pacific salmon fisheries. *Aquaculture*, **43**, 135–146.

Kawecki TJ (1994) Accumulation of deleterious mutations and the evolutionary cost of being a generalist. *American Naturalist*, **144**, 833–838.

Kawecki TJ, Barton NH, Fry JD (1997) Mutational collapse of fitness in marginal habitats and the evolution of ecological specialization. *J. Evol. Biol.*, **10**, 407–429.

Kimura M, Maruyama T, Crow JF (1963) The mutation load in small populations. *Genetics*, **48**, 1303–1312.

Kimura M, Ohta T (1969) The average number of generations until fixation of a mutant gene in a finite population. *Genetics*, **61**, 763–771.

Kirkpatrick M, Barton NH (1997) Evolution of a species' range. *American Naturalist*, **150**, 1–23.

Kohane MJ, Parsons PA (1988) Domestication: evolutionary change under stress. *Evolutionary Biology*, **23**, 31–48.

Lacy RC (1989) Analysis of founder representation in pedigrees: founder equivalents and founder genome equivalents. *Zoo Biology*, **8**, 111–123.

Larkin PA (1980) A perspective on population genetics and salmon management. *Can. J. Fish. Aquatic Sci.*, **38**, 1469–1475.

Lande R, Barrowclough GF (1987) Effective population size, genetic variation, and their use in population management. In: *Viable Populations for Conservation* (ed. Soule ME), pp. 87–123. Cambridge University Press, UK.

Lynch M, Blanchard J, Houle D, Kibota T, Schultz S, Vassilieva L, Willis J (1999) Spontaneous deleterious mutation. *Evolution*, **53**, 645–663.

Lynch M, Conery J, Bürger R (1995) Mutation accumulation and the extinction of small populations. *American Naturalist*, **146**, 489–518.

Olney PJS, Mace GM, Feistner ATC (1994) *Creative Conservation Interactive Management of Wild and Captive Animals*. Chapman and Hall, London, UK.

Reisenbichler RR, McIntyre JD (1977) Genetic differences in growth and survival of juvenile hatchery and wild steelhead trout, *Salmo gairdneri. J. Fish. Res. Bd. Canada* **34**, 123–128.

Reisenbichler RR, Rubin SP (1999) Genetic changes from artificial propagation of Pacific salmon affect the productivity and viability of supplemented populations. *ICES Journal of Marine Science*, **56**, 459–466.

Ruzzante DE, Doyle RW (1993) Evolution of social behavior in a resource-rich structured environment: selection experiments with medaka *(Oryzias latipes)*. *Evolution*, **47**, 456–470.

Ryman N, Laikre L (1991) Effects of supportive breeding on the genetically effective population size. *Conservation Biology*, **5**, 325–329.

Utter FM, Hindar K, Ryman N (1993) Genetic effects of aquaculture on natural salmonid populations. In: *Salmon Aquaculture* (eds. Heen K, Monahan RR, Utter F), pp. 144–165. Fishing News Books, Oxford, UK.

Waples RS (1991) Genetic interactions between hatchery and wild salmonids: lessons from the Pacific northwest. *Canadian Journal of Fisheries and Aquatic Sciences*, **48**, 124–133.

Waples RS, Do C (1994) Genetic risk associated with supplementation of Pacific salmonids: captive broodstock programs. *Canadian Journal of Fisheries and Aquatic Sciences*, **51**, 310–329.

Whitlock MC, Barton NH (1997) The effective size of a subdivided population. *Genetics*, **146**, 427–441.

Wohlfarth GW (1993) Genetic management of natural fish populations. In: *Genetic Conservation of Salmonid Fishes* (eds. Cloud JG, Thorgaard GH), pp. 227–230. Plenum Press, New York.