Selection in Captivity during Supportive Breeding May Reduce Fitness in the Wild

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Abstract: *I used a quantitative genetic model to explore the effects of selection on the fitness of a wild population subject to supportive breeding. Supportive breeding is the boosting of a wild population's size by breeding part of the population in captivity and releasing the captive progeny back into the wild. The model assumes that a single trait is under selection with different optimum trait values in the captive and wild environments. The model shows that when the captive population is closed to gene flow from the wild population, even low levels of gene flow from the captive population to the wild population will shift the wild population's mean phenotype so that it approaches the optimal phenotype in captivity. If the captive population receives gene flow from the wild, the shift in the wild population's mean phenotype becomes less pronounced but can still be substantial. The approach to the new mean phenotype can occur in less than 50 generations. The fitness consequences of the phenotypic shift depend on the details of the model, but a >30% decline in fitness can occur over a broad range of parameter values. The rate of gene flow between the two environments, and hence the outcome of the model, is sensitive to the wild environment's carrying capacity and the population growth rate it can support. The results have two important implications for conservation efforts. First, they show that selection in captivity may significantly reduce a wild population's fitness during supportive breeding and that even continually introducing wild individuals into the captive population will not eliminate this effect entirely. Second, the sensitivity of the model's outcome to the wild environment's quality suggests that conserving or restoring a population's habitat is important for preventing fitness loss during supportive breeding.*

La Selección en Cautiverio Durante la Reproducción de Apoyo Puede Reducir la Adaptabilidad en Condiciones Silvestres

Resumen: *Se empleó un modelo genético cuantitativo para explorar los efectos de la selección en la adaptabilidad de una población silvestre sujeta a reproducción de apoyo. La reproducción de apoyo involucra el fomento del tamaño poblacional silvestre mediante la reproducción de parte de la población en cautiverio y la liberación de progenie cautiva al medio silvestre. El modelo asume que una sola característica se encuentra bajo selección con diferentes valores óptimos de esta característica en los ambientes de cautiverio y silvestres. El modelo muestra que cuando la población cautiva está cerrada al flujo de genes de la población silvestre, aún niveles bajos de flujo de genes de la población cautiva a la población silvestre sesgaría la media del fenotipo de la población silvestre de tal manera que se aproxime al óptimo del fenotipo en cautiverio. Si la población cautiva recibe un flujo de genes de la población silvestre, el sesgo del fenotipo promedio de la población silvestre sería menos pronunciado pero aún sustancial. El acercamiento a la nueva media del fenotipo puede ocurrir en menos de 50 generaciones. Las consecuencias de adaptación del sesgo del fenotipo depende de los detalles del modelo, pero puede ocurrir una disminución de hasta <30% en la adaptabilidad en un rango amplio de valores de parámetros. La tasa de flujo de genes entre los dos ambientes y por lo tanto el producto del modelo es sensible a la capacidad de carga del ambiente silvestre y de la tasa de crecimiento poblacional que puede soportar. Los resultados tienen dos implicaciones importantes para los esfuerzos de conservación. Primero, muestran que la selección en cautiverio puede reducir significativamente la adapt-*

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ación de una población durante reproducción de apoyo y que aunque se introduzcan continuamente individuos silvestres a la población cautiva, no se eliminaría completamente este efecto. Segundo, la sensibilidad del resultado del modelo a la calidad del ambiente silvestre sugiere que la conservación o restauración de la calidad del hábitat silvestre es importante para prevenir la pérdida de adaptabilidad durante la reproducción de apoyo.

Introduction

Supportive breeding is a method of boosting population size that involves breeding part of a population in captivity and then releasing the captive progeny back into the wild (Ryman & Laikre 1991). If captive individuals have high rates of survival compared with those of wild individuals, and if their progeny can be successfully returned to the wild, supportive breeding can be used to increase the size of the population and lower its risk of extinction (Cuenco et al. 1993). Supportive breeding has been used for a variety of species (Olney et al. 1994), and it has recently become an especially common tool for increasing the size of fish populations. For example, many of the proposed management alternatives for the conservation and recovery of Pacific salmonid populations (*Oncorhynchus* sp.) currently listed under the U.S. Endangered Species Act involve some form of supportive breeding (or "supplementation," Hedrick et al. 1994; National Marine Fisheries Service 1995).

In addition to the potential benefit of increasing abundance, supportive breeding poses risks to wild populations. For example, Ryman and Laikre (1991), Waples and Do (1994), and Ryman et al. (1995) found that supportive breeding can result in a reduction in effective population size. Waples (1991), Busack and Currens (1995), and Campton (1995) review other deleterious genetic effects of supportive breeding. One potential deleterious effect discussed in all of these reviews is selection that occurs in the captive environment. Selection that occurs in captivity can be deleterious because traits that are advantageous in the captive environment may not be advantageous in the wild. If the trait distribution in a wild population is at an optimum that has been shaped by selection in the wild environment, releasing individuals into the population that have a different distribution as a result of selection in captivity will result in a reduction in the mean fitness of the population. If supportive breeding proceeds for many generations, the trait distribution of the wild population might evolve far away from its wild optimum. Further, selective change could occur in captivity even if there is no deliberate plan for artificial selection. In Pacific salmonids, for example, there is evidence that conditions in captivity inadvertently select for behavioral and morphological traits that are not optimal in the wild (e.g., Reisenbichler & McIntyre 1977; Nickelson et al. 1986; Fleming & Gross 1989; Swain & Riddell 1990; Fleming & Gross 1992, 1993, 1994; Flagg et al. 1995).

Most well-understood adaptations involve traits such as morphology and behavior whose distributions in a population are influenced by multiple genetic and environmental factors (reviews by Hard 1995, Lynch 1996). Because these traits are influenced by many genes whose individual effects on the phenotype are unknown, a detailed understanding of the genetic architecture of these traits is usually impossible to attain. Instead, they must be studied with the tools of quantitative genetics (Falconer & Mackay 1996; Roff 1997). This means that the genetic and environmental influences on the distribution of a set of traits is described statistically, with no attempt to describe the genetic basis of the traits in detail.

I used a quantitative genetic model to explore how the combined effects of selection in two environments, captive and wild, can influence the distribution of a phenotypic trait and the fitness of a population. This model makes numerous simplifying assumptions, but it is expected to provide a more realistic understanding of how selection might operate during supportive breeding than has been possible with previous models, which have generally assumed that selection in captivity occurs at a single genetic locus (e.g., Byrne et al. 1992). Doyle (1983) used a quantitative genetic model to explain how domestication could occur in aquacultural settings, but assumed a completely closed captive population. Tufto (2000) recently employed several quantitative genetic models to explore a situation in which a population continually receives migrants from a genetically divergent population, but did not examine the case of two-way migration. As part of a broader study of supportive breeding, Adkison (1994) employed a quantitative genetic model similar to the one I describe here. I employed a much simpler demographic model, however, and focused more narrowly on exploring the conditions under which the model predicts changes in the distribution of a phenotypic trait.

My primary goals were to (1) develop a simple model of selection on a quantitative trait during supportive breeding and (2) evaluate the sensitivity of the model to its parameters, which include the strength of selection in the captive and wild environments, the rate of exchange between the two environments, the maximum reproductive rate of the population in each environ-

ment, and the duration of supportive breeding. The model does not take into account all the genetic factors that could affect the outcome of supportive breeding. Instead, I used a simple model to gain some insight into the possible effects of artificial or natural selection on supportive breeding and to determine the sensitivity of these potential effects to the different parameters in the model.

Phenotypic Model

I used a simple deterministic model of phenotypic evolution drawn from Lande (1976), Via and Lande (1985), and Bulmer (1985). Assuming a population of infinite size and discrete generations, the response of a single phenotypic trait to selection in one generation can be described by

$$\Delta\bar{z} = [\bar{z}_s - \bar{z}]h^2, \quad (1)$$

where $\bar{z}$ is the mean trait value before selection, $\bar{z}_s$ is the mean trait value after selection, and h^2 is the realized heritability of the trait. Lande (1976) showed that if the trait is normally distributed with mean $\bar{z}$ and constant variance σ^2, and is subject to bell-shaped (Gaussian) selection with an optimal trait value of θ and range of high-fitness trait values of ω (Fig. 1), the relative fitness, W, of an individual with trait value z is

$$W(z) = \exp\left(\frac{-(z-\theta)^2}{2\omega^2}\right) \quad (2)$$

the mean relative fitness of the population, $\overline{W}$, is

$$\overline{W}(\bar{z},\theta,\omega) \propto \exp\left(\frac{-(\bar{z}-\theta)^2}{2(\omega^2+\sigma^2)}\right) \quad (3)$$

and the change in mean trait value from one generation to the next is described by

$$\bar{z}' = \bar{z} + \left[\frac{\bar{z}\omega^2 + \theta\sigma^2}{\omega^2+\sigma^2} - \bar{z}\right]h^2. \quad (4)$$

Under this model, selection causes $\bar{z}$ to change over time so that the mean trait value approaches the opti-

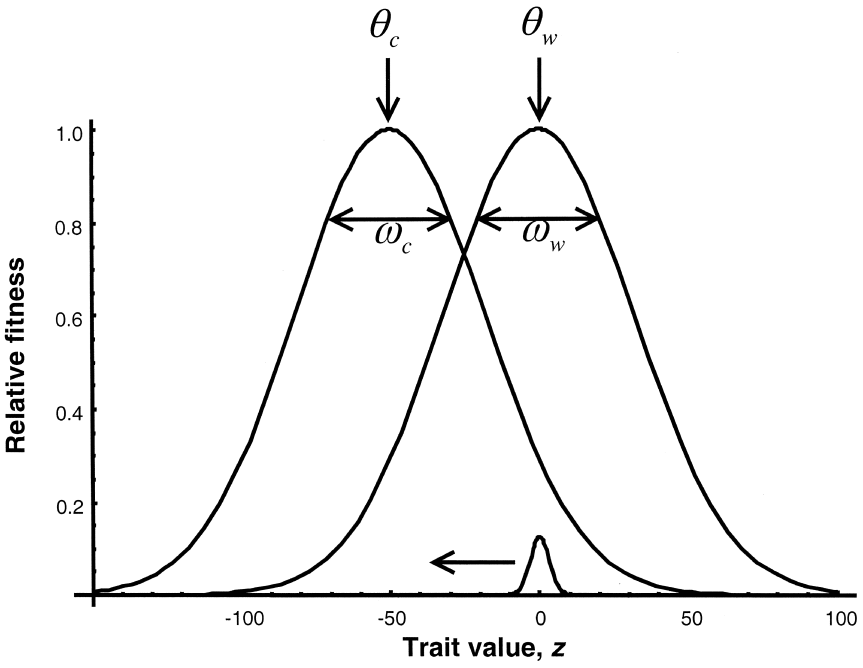

Figure 1. The basic concept of the phenotypic model. The curve on the right is the function describing the fitness in the wild environment of an individual with trait value z *(equation 2). Fitness in the wild is maximized for individuals with trait value* $z = \theta_w$. *The curve on the left is the function describing fitness in the captive environment. The optimal trait value in the captive environment is* $z = \theta_c$. *The width of the fitness curves, and hence the strength of selection, is described by the parameters* ω_w *and* ω_c *for the wild and captive environments, respectively* ($\omega_w^2 = \omega_c^2 = 1000$). *The smaller curve under the wild fitness function describes a population that has evolved so that its mean phenotype is equal to the optimal phenotype in the wild. The phenotypic variance in this population,* σ^2, *is 10, and is assumed to remain constant. If part of the population is taken into captivity and used for supportive breeding, the captive-fitness function slowly moves the trait distribution in the population to the left, toward the captive optimum.*

mum trait value (Fig. 1). The model assumes that selection is weak enough for mutation and recombination to continually replenish genetic variation so that the total variance and heritability of the trait do not change over time (Lande 1976). This assumption is supported by numerous empirical examples of continual phenotypic change from long-term artificial selection (Roff 1997). The model also assumes that all changes in the mean trait value are due to selection and that other potential causes of phenotypic change, such as random genetic drift or phenotypic plasticity, can be ignored. For the time scales and strengths of selection I examined, phenotypic changes produced by drift will be small as long as populations are reasonably large ($N_e >> 10$) (Lande 1976). For small populations in which drift cannot be ignored, the model results can be interpreted as the average outcome that would be expected were the populations to be replicated many times. This model does not take into account short-term phenotypic changes caused by developmental plasticity.

Bulmer (1985) extended this selection model to the case of two populations, and I followed a similar approach (Fig. 1). I let $\bar{z}_w$ and $\bar{z}_c$ be the means of a normally distributed trait in the wild and captive populations, respectively. For simplicity, I assumed that in each population the trait has the same (constant) phenotypic variance, σ_2. I let θ_w, ω_w, θ_c, and ω_c be the parameters for the Gaussian fitness function (equation 3) in the wild and captive environments, respectively. Assuming Gaussian selection and random mating in each environment, the recursion equations for the change in the mean value of the trait in each population are

$$\bar{z}_w' = p_w\left\{\bar{z}_w + \left[\frac{\bar{z}_w\omega_w^2 + \theta_w\sigma^2}{\omega_w^2 + \sigma^2} - \bar{z}_w\right]h^2\right\} + (1-p_w)\left\{\bar{z}_c + \left[\frac{\bar{z}_c\omega_w^2 + \theta_w\sigma^2}{\omega_w^2 + \sigma^2} - \bar{z}_c\right]h^2\right\} \quad (5)$$

and

$$\bar{z}_c' = p_c\left\{\bar{z}_c + \left[\frac{\bar{z}_c\omega_c^2 + \theta_c\sigma^2}{\omega_c^2 + \sigma^2} - \bar{z}_c\right]h^2\right\} + (1-p_c)\left\{\bar{z}_w + \left[\frac{\bar{z}_w\omega_c^2 + \theta_c\sigma^2}{\omega_c^2 + \sigma^2} - \bar{z}_w\right]h^2\right\}, \quad (6)$$

where p_w is the proportion of the individuals in the wild population that originated from the wild population the previous generation and p_c is the proportion of individuals in the captive population that originated from the captive population the previous generation.

Setting $\bar{z}_w'$ equal to $\bar{z}_w$ and $\bar{z}_c'$ equal to $\bar{z}_c$ and solving for $\bar{z}_w$ and $\bar{z}_c$, the equilibrium mean trait values in each population are

$$\hat{z}_w = \frac{\sigma^2((1+p_c(h^2-1))\theta_w + (h^2-1)(p_w-1)\theta_c) + \sigma^2(2-p_w-p_c+h^2(p_w+p_c-1)) + \dfrac{\theta_c(\omega_w^2-\omega_w^2p_w) - \theta_w\omega_c^2(p_c-1)}{\omega_w^2(1-p_w)+\omega_c^2(1-p_c)}}{} \quad (7)$$

and

$$\hat{z}_c = \frac{\sigma^2((h^2-1)\theta_w(p_c-1) + (1+(h^2-1)p_w)\theta_c) + \sigma^2(2-p_w-p_c+h^2(p_w+p_c-1)) + \dfrac{\theta_c(\omega_w^2-p_w\omega_w^2) - \theta_w\omega_c^2(p_c-1)}{\omega_w^2(1-p_w)+\omega_c^2(1-p_c)}}{}. \quad (8)$$

The equilibrium mean fitnesses of each population can be found by substituting the equilibrium mean trait values into equation 3.

In a plot of equation 7, it is apparent that the level of gene flow from the captive population to the wild population is a key parameter in determining the equilibrium mean trait value in the wild population (Fig. 2). Figure 2a illustrates a worst-case scenario in which the captive population is completely closed to gene flow from the wild population ($p_c = 1$), but the wild population is subject to continual gene flow from the captive population ($p_w < 1$). This situation may occur, for example, in the case of many salmon hatchery or aquaculture programs. In this scenario, the wild population's mean phenotype is shifted so that it approaches the optimal phenotype in captivity unless the level of gene flow from the captive population is very small. In other words, when the captive population is closed to gene flow from the wild, even a small amount of gene flow from the captive into the wild population will shift the wild population's mean equilibrium phenotype toward the optimal phenotype in captivity.

At the other extreme, when the captive population is completely replenished with wild-origin individuals every generation ($p_c = 0$), the relationship between the wild population's mean phenotype and the level of gene flow from the captive population is more linear (Fig. 2d). For any particular level of selection in captivity, the absolute shift in the wild population's mean phenotype is less than half what it was in the case of a closed captive population. Intermediate levels of gene flow from the wild into the captive population produce intermediate shifts in mean phenotype (Fig. 2b & 2c). The relationship between gene flow into the wild population and the wild population's mean trait value also becomes increasingly nonlinear the more the captive population is closed to wild gene flow. These relationships are essentially insensitive to the strength of selection in each population (ω_w and ω_c), so long as the strength of selection is equal in each population. When the strength of selection is unequal in the two populations, the equilib-

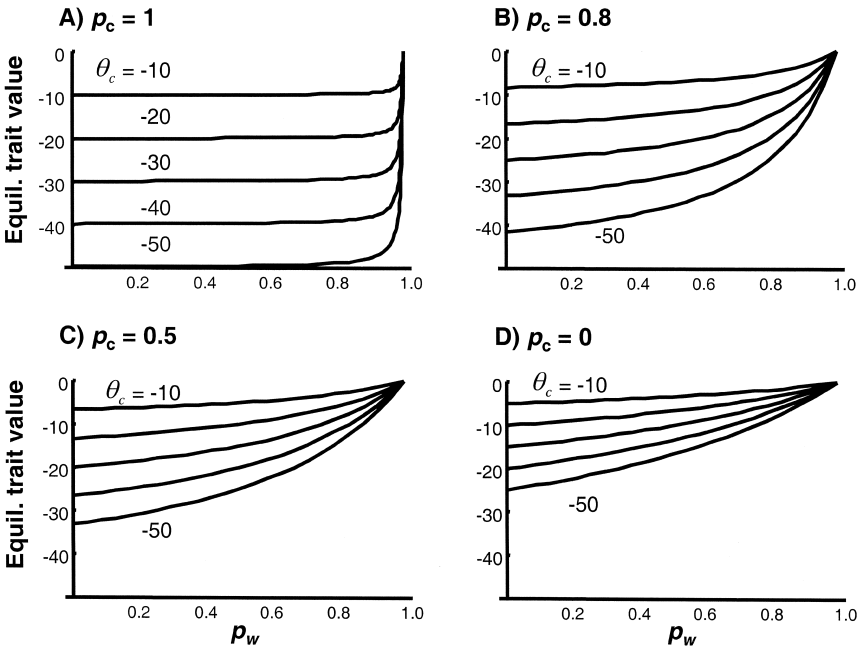

Figure 2. Equilibrium trait values in the wild population as a function of the proportion of wild-origin individuals in the wild population (p_w), captive-origin individuals in the captive population (p_c), and optimal phenotypic value in captivity (θ_c). In plot A, the captive population consists entirely of captive-origin individuals every generation. In plot D, the captive population consists entirely of wild-origin individuals every generation. Plots B and C show intermediate cases. In each case, heritability (h^2) is 0.5, the phenotypic variance (σ^2) is 10, the optimal wild-trait value (θ_w) is 0, and the widths of the selection functions (ω_w^2 and ω_c^2) are both 1000.

rium phenotype tends toward the optimum of the environment with stronger selection.

The fitness consequences of shifts in mean phenotype depend on the difference between the optimal trait values and the strength of selection in each environment, but they can be large for a broad range of parameter values (Fig. 3). When selection is relatively strong ($\omega^2 = 10\sigma^2$), for example, the fitness of the wild population can be reduced by 80% or more if the captive population is closed to immigration from the wild (Fig. 3a). The wild population is protected from this decline in fitness only when gene flow from the captive into the wild population approaches zero. Weaker selection results in a less dramatic loss of fitness, but even very weak selection ($\omega^2 = 100\sigma^2$) can produce large reductions in fitness if the captive population is closed to immigration

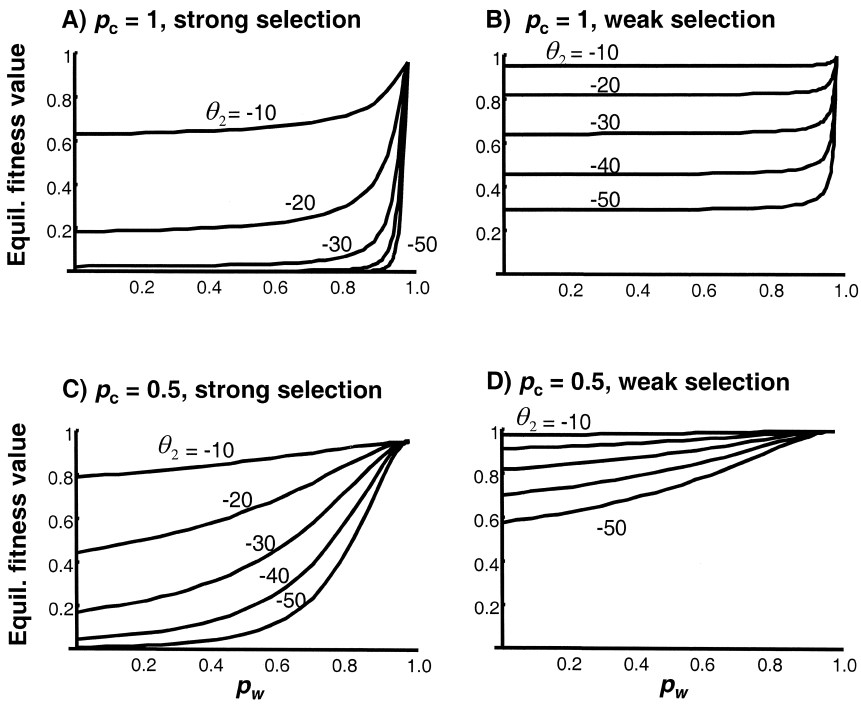

Figure 3. Equilibrium mean fitness values in the wild population as a function of the proportion of wild-origin individuals in the wild population (p_w), captive-origin individuals in the captive population (p_c), and optimal phenotypic value in captivity (θ_c). In all plots, the optimal wild-trait value (θ_w) is 0, and heritability (h^2) is 0.5. In plots A and C, selection is relatively strong ($\sigma^2 = 10$ and $\omega_w^2 = \omega_c^2 = 100$). In plots B and D, selection is relatively weak ($\sigma^2 = 10$ and $\omega_w^2 = \omega_c^2 = 1000$).

from the wild (Fig. 3b). As in the case of strong selection, the fitness response in the wild population is extremely nonlinear. Opening the captive population to immigration from the wild results in a much smaller—but still potentially significant—loss of fitness if selection is weak (Fig. 3d), but it can still produce large reductions when selection is strong (Fig. 3c & 3d). Remarkably similar results to those presented in Fig. 3 have also been found in a model of the accumulation of deleterious mutations during supportive breeding (Lynch & O'Hely 2001).

The rate of approach to the equilibrium fitness values can be rapid if selection is strong. For a population initially at its optimal wild trait value, selection can shift the mean phenotype 90% of the way to its new equilibrium value in <50 generations, and substantial reductions in wild fitness can occur in <20 generations (Table 1). For many short-lived organisms, such as Pacific salmon, this time frame is relevant to conservation efforts. The time to approach the new equilibrium value is sensitive to the strength of selection but relatively insensitive to the rate of gene flow between the captive and wild environments (Table 1).

Demographic Model

The phenotypic model is useful for illustrating relationships between key parameters, but it does not explicitly incorporate the demographics of supportive breeding. In particular, this model implicitly assumes that there are an effectively infinite number of individuals in each population so that the immigration proportions, $1 - p_w$ and $1 - p_c$ can be set at arbitrary values. In reality, the immigration proportions vary depending on the growth rates of each population, which are themselves functions of the mean population fitness in each population.

Table 1. Time, in generations, for a wild population's mean phenotype to reach 50% and 90% of the way toward its new equilibrium value after the start of supportive breeding.

	Strong selection[a]				*Weak selection*[b]			
	$p_c = 1$		$p_c = 0.5$		$p_c = 1$		$p_c = 0.5$	
p_w	t_{50}	t_{90}	t_{50}	t_{90}	t_{50}	t_{90}	t_{50}	t_{90}
0.1	16	50	16	48	138	456	135	446
0.25	17	48	15	47	139	458	135	440
0.50	17	49	16	44	140	464	127	404
0.75	19	52	16	44	144	444	123	422
1.0	23	53	15	42	147	459	113	313

[a]*The width of the fitness functions, ω_w^2 and ω_c^2, is 100; the phenotypic variance, σ^2, is 10; t_i is the number of generations needed to reach* i% *of the way to the equilibrium mean fitness value; and* p_c *and* p_w *are the proportion of captive breeders that originated from captivity and wild breeders that originated from the wild, respectively.*
[b]$\omega_w^2 = \omega_c^2 = 1000$, $\sigma^2 = 10$.

Under many conditions, the immigration proportions are therefore expected to be influenced by selection.

To address this issue, I combined the phenotypic model with a simple, discrete generation-demographic model of supportive breeding similar to that described by Ryman and Laikre (1991) and Cuenco (1994). The life cycle starts when adults are either taken into captivity or allowed to breed in the wild environment. Reproduction occurs separately in each environment, and the number of offspring produced is determined by multiplying the maximum potential reproductive rate of the population in the environment by the mean relative fitness of the population. All captive offspring are then released back into the wild environment, where they grow to adulthood and the life cycle starts over again. The model assumes that captive adults are marked in some way, so that it is possible to determine the rate of exchange between the captive and wild environments. The recursion equations describing the number of breeding adults in one generation as a function of the number of breeders in the previous generation are

$$N_w' = (1 - m_w) f_w(N_w) \overline{W}(\bar{z}_w, \theta, \omega_w) + m_c f_c(N_c) \overline{W}(\bar{z}, \theta, \omega_w) \quad (9)$$

and

$$N_c' = (1 - m_c) f_c(N_c) \overline{W}(\bar{z}_c, \theta, \omega_c) + m_w f_w(N_w) \overline{W}(\bar{z}_w, \theta_c, \omega_c), \quad (10)$$

where N_w' and N_c' are the number of breeders in the wild and captive populations, respectively, in generation $t + 1$; N_w and N_c are the number of breeders in generation t; m_w is the fraction of the adult progeny from the wild population that returns to breed in the captive population; m_c is the fraction of the adult progeny from the captive population that returns to spawn in the wild population; $f_w(N_w)$ and $f_c(N_c)$ are functions that describe the maximum per-capita reproductive rate in the wild and captive environments, respectively. The m_w and m_c are emigration rates prior to selection, and $1 - p_w$ and $1 - p_c$ are net immigration rates after selection.

Any number of functions could be used in equations 9 and 10 to describe reproduction. For simplicity, all the scenarios assume reproduction is independent of density in each environment up to an environment-specific limit, such that

$$f_w(N_w) = \begin{cases} N_w R_w, \text{ for } N_w < K_w \\ K_w R_w, \text{ for } N_w \geq K_w \end{cases} \quad (11)$$

and

$$f_c(N_c) = \begin{cases} N_c R_c, \text{ for } N_c < K_c \\ K_c R_c, \text{ for } N_c \geq K_c \end{cases}, \quad (12)$$

where R_w and R_c are the maximum per-capita reproductive rates the wild and captive environments can sup-

port, respectively, and K_w and K_c are the carrying capacities of the wild and captive environments, respectively. The maximum per-capita reproductive rate for an environment is defined as that which a population at low density with $\overline{W} = 1$ would have in that environment. Carrying capacity is defined here as the breeding population size above which no further increases in recruitment are possible. The maximum possible number of adult recruits produced by the wild and captive environments are therefore K_wR_w and K_cR_c, respectively. Like the phenotypic model, the demographic model is entirely deterministic.

It is worth briefly examining the outcome of the demographic model with no selection in either the wild or captive environments. To do this, I initially made four additional simplifying assumptions. First, I assumed that at the start of supportive breeding the wild population is declining at a constant rate and that breeders brought into captivity will produce more than one adult offspring per breeder ($R_w < 1$ and $R_c > 1$). Second, I assumed that the first priority of the supportive breeding project is to exactly fill the captive environment to its capacity, K_c. Third, I assumed that wild-origin and captive-origin adults are taken into captivity in proportion to their abundance. Fourth, I assumed that the population will remain below the carrying capacity of the wild environment. The third assumption means that m_w equals the proportion of the entire population (of both captive and wild origin) that returns to breed in captivity. These assumptions mean that

$$m_w = \left\{ \begin{array}{c} \dfrac{K_c}{N_wR_w + N_cR_c}, \text{ for } N_wR_w + N_cR_c > K_c \\ 1, \text{ for } N_wR_w + N_cR_c < K_c \end{array} \right\} \quad (13)$$

and

$$m_c = 1 - m_w .$$

Assuming that R_w, R_c, and K_c remain constant, equations 9 and 10 can be solved to find the equilibrium number of breeders in the captive and wild populations,

$$\hat{N}_w = \frac{K_cR_c - K_c}{1 - R_w} \quad (14)$$

and

$$\hat{N}_c = K_c .$$

At equilibrium, the proportion of each population that consists of adults born in the wild environment will be

$$\hat{p}_w = \frac{R_w(R_c - 1)}{R_c - R_w} . \quad (15)$$

Under these assumptions, the equilibrium size of the wild population is determined solely by the capacity of the captive environment and the maximum per-capita reproductive rates in the wild and captive environments. The equilibrium proportion of each population born in the wild is determined solely by the per-capita reproductive rates in each environment so that as $R_w \to 1$, $p_w \to 1$, and as $R_c \to \infty$, $p_w \to R_w$.

The demographic and phenotypic models can be combined by substituting

$$p_i = \frac{(1 - m_i)\overline{W}(\bar{z}_i, \theta, \omega_i)f_i(N_i)}{N_i^*} \quad (16)$$

into equations 5 and 6, where N_i^* is the total population size of the ith population after migration and selection. The resulting system of phenotypic and demographic equations does not appear to be readily mathematically tractable, but it can be easily iterated to explore the model's behavior. Below, I explore how several key parameters, including the relative reproductive rates and carrying capacities of the wild and captive environments, affect the outcome of the model.

The dynamics of the combined demographic and phenotypic models can be complicated. As an example, consider a situation where at the time supportive breeding is initiated the wild population is declining at a rapid rate ($R_w = 0.05$) and the maximum potential reproductive rate in captivity is considerably higher than in the wild ($R_c = 3$). The captive population is founded by wild-origin individuals, with a mean phenotype initially equal to the optimum in the wild. The optimal phenotype in captivity is substantially different (approximately 16 phenotypic standard deviations) from the wild optimum. The first 100 generations of this scenario are plotted in Fig. 4. Initially, the wild population rapidly decreases and the captive population increases as wild individuals are brought into captivity. The fitness of the captive population is initially low (because the wild individuals' mean phenotype is far from the captive optimum), so once the wild population goes extinct the captive population begins to decline as well (Fig. 4b). The rate of decline slows, however, as the population adapts to the captive environment (Fig. 4c), and eventually the captive population starts to increase in size (Fig. 4b). After about 25 generations, the captive population has become productive enough that it has "surplus" individuals that can be reintroduced into the wild (Fig. 4a). In generations 25 to 100, the population continues to evolve toward the captive optimum, so its fitness in captivity continues to improve while its fitness in the wild slowly declines (Fig. 4c & 4d).

A key result from the combined demographic and phenotypic model is that, when the wild population is well below its carrying capacity, the effect of supportive breeding on the wild population's mean fitness is highly dependent on the maximum potential reproductive rates in both the wild and captive environments (Fig. 5a). When R_w is relatively large and R_c is large enough

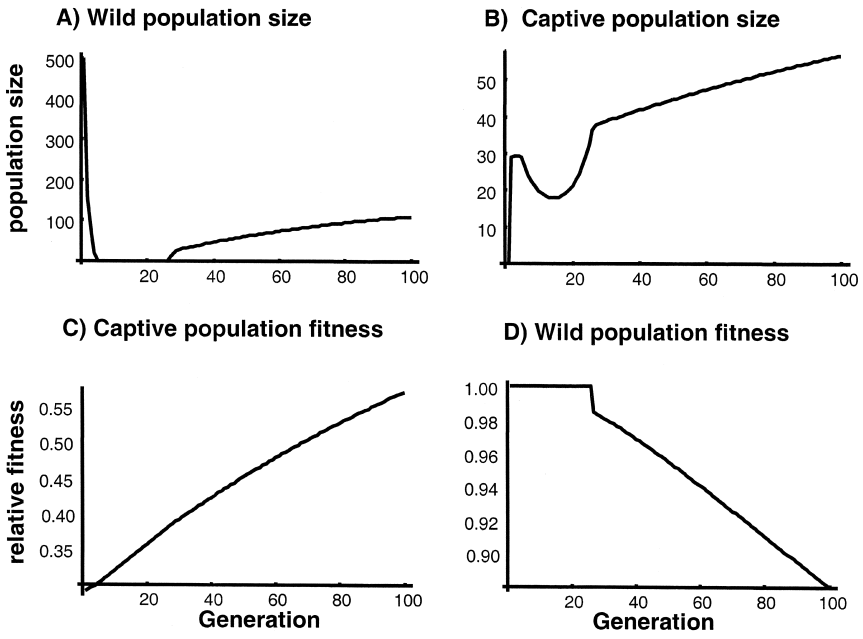

Figure 4. Population size and mean fitness of the wild and captive populations over the course of 100 generations of supportive breeding. Plot A shows the wild population's size, plot B shows the captive population's size, plot C shows the captive population's fitness, and plot D shows the wild population's fitness. The optimal phenotype in the wild (θ_w) is 0; the optimal phenotype in captivity (θ_c) is −50; the phenotypic variance (σ^2) is 10, and selection is weak in both environments ($\omega_w^2 = \omega_c^2 = 1000$). Heritability ($h^2$) is 0.5.

that the captive population has surplus production to contribute individuals to the wild population, the proportion of wild-produced individuals remains relatively high, and, as expected from the results of the purely phenotypic model, the mean fitness of the wild population remains relatively unchanged by the supportive breeding program. On the other hand, when either R_w or R_c is relatively small, the proportion of wild-produced individuals also becomes small, and the mean fitness of the wild population declines as its mean phenotype evolves away from its optimum value.

If the wild population has a finite carrying capacity, the simple relationship between the reproductive rates in the two environments and the proportion of wild-origin individuals no longer applies, and it becomes possible to "overload" the wild environment with captive individuals. In this case, if the carrying capacity is low relative to the surplus productivity of the captive population, the fitness of the wild population can decline substantially (Fig. 5b). This result is again consistent with the results from the purely phenotypic model. When the wild environment is overloaded with captive-origin individuals, the proportion of wild-origin individuals, p_w, becomes small, and relatively few fish that have been selected for the wild phenotype are produced compared with the large number of captively reared migrants.

Model Assumptions and Limitations

The phenotypic model makes several simplifying assumptions, the most important of which are that the phenotypic trait of interest is normally distributed with constant variance, that selection on the trait is constant and stabilizing, that all changes in the mean trait value are due to selection, and that there is no concurrent selection on correlated traits. The demographic model makes additional assumptions, such as discrete generations and a specific form of density-dependent reproduction. These assumptions are unlikely to be met exactly but are close enough to reality to provide insight into the likely outcomes of supportive breeding. For example, the assumption that a trait will remain normally distributed with constant variance will be approximately met if selection is sufficiently weak ($\omega^2 >> \sigma^2$), migration rates are sufficiently high, and mutation and recombination act to continually normalize variation.

Likewise, the assumption of stabilizing selection in wild populations is probably qualitatively correct for many traits. For example, wild populations of Pacific salmonids often appear to have local adaptations that increase fitness in their spawning and rearing habitats (Taylor 1991), and many behavioral and morphological traits in Pacific salmonids (such as run timing) are probably under some form of stabilizing selection. The assumption that selection on wild populations is constant is unlikely to be exactly true, but it will be qualitatively correct as long as the wild optimum does not change appreciably over the course of the supportive breeding project or if the difference between the wild and captive optima is much larger than the range of variation within either optima. The assumption that only one trait is subject to selection is obviously not true, but modeling additional traits adds considerably to the number of parameters in the model without providing much additional insight. The demographic assumptions are certainly not

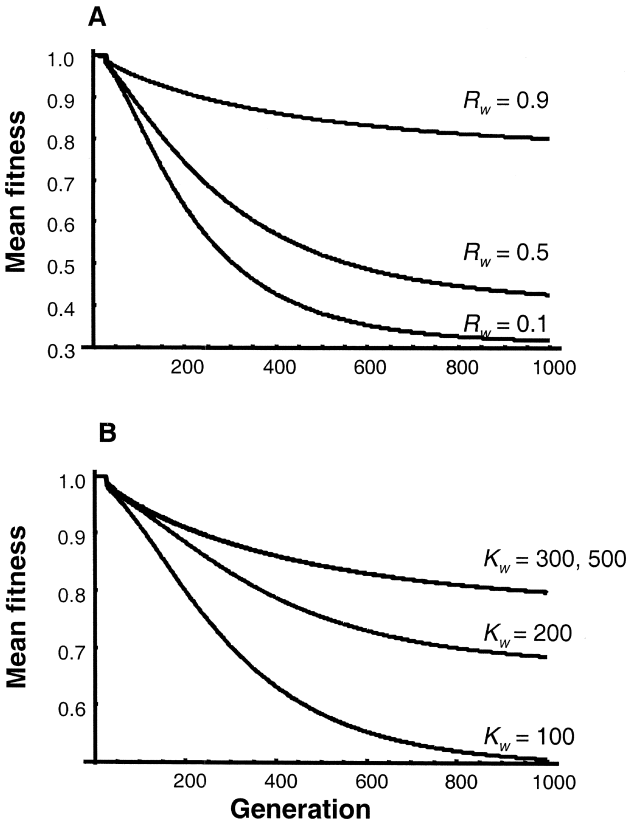

Figure 5. Effect of varying maximum reproductive rate in the wild (R_w) (plot A) and carrying capacity in the wild (K_w) (plot B) on mean fitness of the wild population during 1000 generations of supportive breeding. The optimal phenotype in the wild (θ_w) is 0; the optimal phenotype in captivity (θ_c) is −50; the phenotypic variance (σ^2) is 10, and selection is weak in both environments ($\omega_w^2 = \omega_c^2 = 1000$). The maximum reproductive rate in captivity (R_c) is 3. The maximum wild reproductive rate (R_w) in part B is 0.9. Heritability (h^2) is 0.5.

exactly correct for many species, but they are nonetheless commonly used in fisheries management as a first approximation to more complex demographics.

Conservation Implications

For salmonids there are several examples of reductions in fitness caused by captive propagation (e.g., Reisenbichler & McIntyre 1977; Leider et al. 1990; Fleming et al. 1996). The results of the model show that these fitness reductions are not unexpected, although they are expected to vary depending on factors such as the strength of selection and the rate of exchange between captive and wild populations. The model provides some insight not immediately obvious from existing empirical studies. First, most experimental studies of the fitness effects of captive breeding focus on the survival or reproductive success of captively propagated fish when they are released into the wild. The long-term effects of such releases on the mean fitness of a supplemented wild population have not been measured, but the model shows that these effects can be significant (Figs. 3 & 5).

Second, most empirical studies to date have focused on genetic changes in captive populations that received few if any wild immigrants subsequent to the initial founding of the captive population. In contrast, nearly all of the more recent supportive-breeding projects for Pacific salmonids involve deliberately managed migration between captive and wild populations (e.g., Carmichael & Messmer 1995). The primary purpose of regularly bringing wild-origin broodstock into these programs is to avoid domestication of the captive stock. I explicitly explored this two-population scenario and found that substantial phenotypic changes and fitness reductions can occur even if a large fraction of the captive broodstock is brought in from the wild every generation (Figs. 2 & 3). This suggests that regularly bringing wild-origin broodstock into captive populations cannot be relied upon to eliminate the effects of inadvertent domestication selection, although the rate and level of domestication will be reduced compared with those of a completely closed captive population.

The sensitivity of the degree of phenotypic change to the relative proportions of captive and wild individuals—and hence indirectly to the maximum potential reproductive rates in the wild and captive environments—has two interesting implications. First, it means that wild-origin breeders are important to a population's viability in the wild even in cases where the wild population is not able to sustain itself without the aid of supportive breeding. All the scenarios I explored had wild per-capita reproductive rates of <1, so the wild population in each case would have gone extinct deterministically without the aid of supportive breeding. Those wild populations with relatively high reproductive rates ($R_1 > 0.9$), however, were much less prone to phenotypic change during supportive breeding than less-healthy populations (Fig. 5).

The dependence on the potential reproductive rate in the wild environment means that conserving or restoring a population's habitat (or addressing other factors that limit the population's reproductive rate) may be the most effective method of preventing phenotypic change during supportive breeding, even if these improvements are not sufficient to allow the population to sustain itself entirely naturally. The results also suggest that controlling the exchange rate between captive and wild populations would be an effective way of limiting domestication of wild populations. In a supportive-breeding situation, however, where the goal is to use captive individuals to increase the size of a declining wild popula-

tion, it may be impossible to achieve the desired demographic boost while keeping the proportion of captive-origin individuals in the wild population low (Cuenco 1994). On the other hand, for some fish species there exist aquaculture programs that result in the inadvertent release of captively bred individuals into wild populations. My results suggest that such inadvertent releases can, given enough time, result in substantial phenotypic changes and fitness loss in wild populations even at low rates of introgression (Fig. 2a).

The sensitivity of the model to the wild reproductive rate also implies that those populations most in need of supportive breeding are also those most vulnerable to phenotypic change due to selection in captivity. This suggests that in situations where improving the wild reproductive rate is not feasible, it is especially critical to avoid strong selection in captivity. Representative sampling of the population for broodstock (Hard et al. 1992) and the use of "natural" methods of breeding and rearing (e.g., Maynard et al. 1995) may be particularly important in situations where wild reproductive rates are low, although the ability of these measures to adequately mimic the selection that occurs in the wild environment is unknown.

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Literature Cited

Adkison, M. D. 1994. Application of mathematical modeling to problems in salmon biology and management. Ph.D. dissertation. University of Washington, Seattle.

Bulmer, M. G. 1985. The mathematical theory of quantitative genetics. Clarendon Press, Oxford, United Kingdom.

Busack, C. A., and K. P. Currens. 1995. Genetic risks and hazards in hatchery operations: fundamental concepts and issues. American Fisheries Society Symposium **15:**71–80.

Byrne, A., T. C. Bjornn, and J. D. McIntyre. 1992. Modeling the response of native steelhead to hatcher supplementation programs in an Idaho river. North American Journal of Fisheries Management **12:**62–78.

Campton, D. E. 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.

Carmichael, R. W., and R. T. Messmer. 1995. Status of supplementing chinook salmon natural production in the Imnaha River basin. American Fisheries Society Symposium **15:**284–291.

Cuenco, M. L. 1994. A model of an internally supplemented population. Transactions of the American Fisheries Society **123:**277–288.

Cuenco, M. L., T. W. H. Backman, and P. R. Mundy. 1993. The use of supplementation to aid in natural stock restoration. Pages 269–288 in J. G. Cloud and G. H. Thorgaard, editors. Genetic conservation of salmonid fishes. Plenum Press, New York.

Doyle, R. W. 1983. An approach to the quantitative analysis of domestication selection in aquaculture. Aquaculture **33:**167–185.

Falconer, D. S., and T. F. C. Mackay. 1996. Introduction to quantitative genetics. Fourth edition. Longman Limited, Essex, United Kingdom.

Flagg, T. A., F. W. Waknitz, D. J. Maynard, G. B. Milner, and C. V. W. Mahnken. 1995. The effect of hatcheries on native coho salmon populations in the lower Columbia River. American Fisheries Society Symposium **15:**366–375.

Fleming, I. A., and M. R. Gross. 1989. Evolution of adult female life history and morphology in a Pacific salmon (coho: *Oncorhynchus kisutch*). Evolution **43:**141–157.

Fleming, I. A., and M. R. Gross. 1992. Reproductive behavior of hatchery and wild coho salmon (*Oncorhynchus kisutch*): does it differ? Aquaculture **103:**101–121.

Fleming, I. A., and M. R. Gross. 1993. Breeding success of hatchery and wild coho salmon (*Oncorhynchus kisutch*) in competition. Ecological Applications **3:**230–245.

Fleming, I. A., and M. R. Gross. 1994. Breeding competition in a Pacific Salmon (coho: *Oncorhynchus kisutch*): measures of natural and sexual selection. Evolution **48:**637–657.

Fleming, I. A., B. Jonsson, M. R. Gross, and A. Lamberg. 1996. An experimental study of the reproductive behavior and success of farmed and wild Atlantic salmon. Journal of Applied Ecology **33:** 893–905.

Hard, J. J. 1995. A quantitative genetic perspective on the conservation of intraspecific diversity. American Fisheries Society Symposium **17:**304–326.

Hard, J. J., R. P. Jones Jr., M. R. Delarm, and R. S. Waples. 1992. Pacific salmon and artificial propagation under the Endangered Species Act. National Oceanic and Atmospheric Administration technical memorandum. National Marine Fisheries Service, Northwest Fisheries Science Center, Seattle.

Hedrick, P. W., D. Hedgecock, and S. Hamelberg. 1994. Effective population size in winter-run chinook salmon. Conservation Biology **9:**615–624.

Lande, R. 1976. Natural selection and random genetic drift in phenotypic evolution. Evolution **30:**314–334.

Leider, S. A., P. L. Hulett, J. J. Loch, and M. W. Chilcote. 1990. Electrophoretic comparison of the reproductive success of naturally spawning transplanted and wild steelhead trout through the returning adult stage. Aquaculture **88:**239–252.

Lynch, M. 1996. A quantitative-genetic perspective on conservation issues. Pages 471–501 in J. C. Avise and J. L. Hamrick, editors. Conservation genetics: case histories from nature. Chapman and Hall, New York.

Lynch, M., and M. O'Hely. 2001. Captive breeding and the genetic fitness of natural populations. Conservation Genetics **2:**363–378.

Maynard, D. J., T. A. Flagg, and C. V. W. Mahnken. 1995. A review of seminatural culture strategies for enhancing the postrelease survival of anadromous salmonids. American Fisheries Society Symposium **15:**307–316.

National Marine Fisheries Service. 1995. Proposed recovery plan for Snake River salmon. Portland, Oregon.

Nickelson, T. E., M. F. Solazzi, and S. L. Johnson. 1986. Use of hatchery coho salmon (*Oncorhynchus kisutch*) presmolts to rebuild wild populations in Oregon coastal streams. Canadian Journal of Fisheries and Aquatic Sciences **43:**2443–2449.

Olney, P. J. S., G. M. Mace, and A. T. C. Feistner. 1994. Creative conservation: interactive management of wild and captive animals. Chapman and Hall, London.

Reisenbichler, R. R., and J. D. McIntyre. 1977. Genetic differences in growth and survival of juvenile hatchery and wild steelhead trout,

Salmo gairdneri. Journal of the Fisheries Research Board of Canada **34:**123–128.

Roff, D. A. 1997. Evolutionary quantitative genetics. Chapman and Hall, New York.

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

Ryman, N., P. E. Jorde, and L. Laikre. 1995. Supportive breeding and variance effective population size. Conservation Biology **9:**1619–1628.

Swain, D. P., and B. E. Riddell. 1990. Variation in agonistic behavior between newly emerged juveniles from hatchery and wild populations of coho salmon, *Oncorhynchus kisutch*. Canadian Journal of Fisheries and Aquatic Sciences **47:**566–571.

Taylor, E. B. 1991. A review of local adaptation in Salmonidae, with particular reference to Pacific and Atlantic salmon. Aquaculture **98:** 185–207.

Tufto, J. 2000. Quantitative genetic models for the balance between migration and stabilizing selection. Genetical Research **76:**285–93.

Via, S., and R. Lande. 1985. Genotype-environment interaction and the evolution of phenotypic plasticity. Evolution **39:**505–522.

Waples, R. S. 1991. Genetic interactions between hatchery and wild salmonids: lessons from the Pacific Northwest. Canadian Journal of Fisheries and Aquatic Sciences **48:**124–133.

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