White Paper No. 1[1]

Predicted Fitness Effects of Interbreeding between Hatchery and Natural Populations of Pacific Salmon and Steelhead

1 Introduction

The propagation of Pacific salmon and steelhead (*Oncorhynchus* spp.[2]) in hatcheries has raised concerns for more than 30 years regarding the long-term genetic effects of hatchery-origin fish on the mean fitness of natural populations (Reisenbichler and McIntyre 1977; Campton 1995; Naish et al. 2007). In general, hatchery-origin fish have lower smolt-to-adult survivals (*viability fitness*) and reproductive success (*reproductive fitness*) in nature than do natural-origin fish (Berejikian and Ford 2004; Araki et al. 2008). Environmental effects associated with artificial feeding and rearing in hatcheries are clearly factors contributing to those fitness differences under natural conditions. However, most traits related to fitness (e.g., fecundity, age at sexual maturity) in salmonid fishes have heritabilities[3] greater than zero (Carlson and Seamons 2008), thus providing a genetic mechanism for hatchery populations to respond phenotypically over multiple generations to *domestication selection* in the hatchery environment.[4] Moreover, phenotypic differences between hatchery and wild fish often increase as a function of the number of generations that fish are propagated artificially, consistent with expectations for heritable traits under selection (Araki et al. 2007). Perhaps the best-known example of heritable selection responses in hatchery populations of Pacific salmon and steelhead are

[1] This white paper was prepared by the HSRG to address topics relevant to hatchery reform. It is intended to provide background, documentation and explanations not included in the body of the HSRG's report.

[2] Species include Chinook salmon (*O. tshawytscha*), chum salmon (*O. keta*), coho salmon (*O. kisutch*), pink salmon (*O. gorbuscha*), sockeye salmon (*O. nerka*), and steelhead (*O. mykiss*).

[3] The *heritability* (h^2)of a trait is defined as the proportion of the total phenotypic variance (V_P) of a trait in a population that is heritable due to *additive* genetic variance (V_A) among individuals within that population ($h^2 = V_A/V_P$; $0 \leq h^2 \leq 1.0$). Most traits are also influenced significantly by environmental and non-inherited sources of genetic variation (i.e., *dominance* and *epistasis*). Indeed, h^2 has been estimated to be less than 0.5 for most traits related to survival or fitness.

[4] Artificial selection in a hatchery environment is often referred to as *domestication selection* (Doyle et al. 1983). Domestication selection includes "natural selection" in the hatchery environment, non-random selection of parents – including non-random culling of progeny - by hatchery personnel (aka "selective breeding"), and random genetic changes resulting from relaxation of natural selection that normally occurs in the "wild" environment (e.g., selection on spawning behavior). Single-generation responses (R) to selection in a population for a particular trait are commonly measured by $R = \mu_P' - \mu_P$, where μ_P' and μ_P are the mean value of the trait in the progeny and parental generations, respectively. This response can be predicted by $\hat{R} = h^2(\mu_S - \mu_P)$, where h^2 is the heritability of the trait in the population, and μ_S is the mean value of the trait for the selected parents (spawners) of the parental generation. In practice, the phenotypic value for each selected parent needs to be weighted by their respective number of progeny. The quantity $\mu_S - \mu_P$ is defined as the *selection differential* (SD) on the trait ($\hat{R} = h^2 \cdot SD$).

shifts in the mean and range of return and spawn dates of adults - as measured by Julian calendar day – relative to natural populations (Mackey et al. 2001; Quinn et al. 2002; Knudsen et al. 2006). Responses to selection for many other traits have been documented or inferred (Berejikian 1995; Fleming et al. 2002; Heath et al. 2003).

The natural spawning of hatchery fish clearly poses genetic risks to natural populations of Pacific salmon and steelhead (Busack and Currens 1995; Currens and Busack 2004). However, those risks and associated effects are difficult to quantify and detect. Based on known phenotypic differences between hatchery and wild fish for heritable traits, the natural spawning of hatchery-origin fish – including the direct interbreeding of hatchery and wild fish in nature – is expected to reduce the mean fitness of natural-origin fish and, hence, reduce the overall productivity[5] of natural populations (Reisenbichler and Rubin 1999; Chilcote 2003; Goodman 2005). Genetic effects are particularly difficult to detect because they are manifested over multiple generations and are usually confounded with other factors that can reduce productivity (e.g., habitat degradation, indirect harvests on wild fish in fisheries targeting hatchery fish, etc.).

The natural spawning of hatchery fish can also increase the total number of fish spawning in a watershed, thus potentially yielding increased numbers of natural-origin smolts and adult recruits in the progeny generation (Bugert 1998; Reisenbichler 2004; Baumsteiger et al. 2008). However, these latter single-generation demographic benefits are sustainable only if they *exceed* the predicted reductions in genetic viability and reproductive fitness of natural-origin fish in subsequent generations. Many hatchery programs for Pacific salmon and steelhead are characterized by large numbers of hatchery-origin adults that, each year, escape fisheries and spawn naturally in watersheds where those fish were released as juveniles. As a consequence, the long-term genetic effects of hatchery fish spawning naturally to natural populations need to be assessed relative to potential demographic benefits when evaluating the benefits and risks of any hatchery program.

The Hatchery Scientific Review Group (HSRG) was tasked with developing hatchery management *solutions* that would allow hatcheries to continue supporting fisheries in a sustainable manner while, at the same time, minimizing or reducing risks to natural populations (Mobrand et al. 2005). The HSRG specifically needed a quantitative method for assessing the long-term fitness effects to natural populations of hatchery fish spawning naturally over multiple generations.

Several theoretical models have been used for assessing the genetic effects of captively-bred animals reproducing in nature with natural populations (Lynch and O'Hely 2001; Ford 2002; Theodorou and Couvet 2004; Goodman 2005). Each of those models has strengths and weaknesses. Of the models currently available, the HSRG adopted the model described by Ford (2002) for its assessments. This model was selected because of its relative simplicity and well-established foundation in quantitative genetics (Bulmer 1985). The HSRG has included the equations of Ford (2002) as algorithmic components

[5] *Productivity* is commonly measured as the mean number of adult recruits (R) of the parental generation per adult spawner (S) of the parental generation, and is often symbolized as "*R/S*". However, *productivity* - in a population dynamics sense - is more precisely defined as the slope at the origin ($S = 0$, $R = 0$) of the spawner-recruitment curve, or function, that defines the empirical mathematical relationship between adult spawner abundance and adult recruit abundance one generation later.

of the *All-H Analyzer* (*AHA*)[6], a hatchery management planning tool designed to assess the combined effects of habitat, hydropower dams, harvest, and hatcheries on the abundance and overall population dynamics of hatchery and wild populations of Pacific salmon and steelhead in the Pacific Northwest.

The paper presented here provides a detailed explanation of the Ford (2002) *phenotypic fitness model* and its direct application to the management of hatchery and wild populations of salmon and steelhead in the Pacific Northwest. Although the mathematical and biological foundations of the model have been thoroughly described elsewhere (Lande 1976; Bulmer 1985; Via and Lande 1985; Ford 2002), the direct application of this model to the complex task of managing hundreds, perhaps thousands, of hatchery and wild populations of Pacific salmon and steelhead has not yet been described. The explanations provided here are intended to serve as a primer for the HSRG's analyses and for entry into the scientific literature.

2 The model: gene flow and selection in two environments (after Ford 2002)

Ford's (2002) *phenotypic fitness model* is a two-population extension of the classic one-population selection model (Bulmer 1985; Appendix). The model assumes the following (after Lande 1976):

- A single trait is under selection with different optimum values, θ_W or θ_H, for fish that are the product of reproduction and early rearing in the wild and hatchery environments, respectively;
- Phenotypic traits are normally distributed and are subject to *Gaussian* selection;
- All adults mate randomly within each environment, not assortatively by origin;
- Populations reproduce as discrete generations;
- Population sizes are large so that random genetic drift, phenotypic plasticity, and other stochastic forces can be ignored;
- All changes in the mean value of a trait between generations are due to the deterministic forces of selection and gene flow;
- Selection does not reduce population sizes, the total genetic variance, or heritability of the trait over time. This form of selection is commonly call "soft selection (Demeeus et al. 1993).

Under the two-population model (Fig. 1), the phenotypic distributions of hatchery and wild fish are assumed to have equal variances (σ^2) but different phenotypic optima, θ_H and θ_W, respectively, resulting from reproduction and early rearing in different environments (Fig. 2). The quantity $|\theta_W - \theta_H|$ measures the *magnitude* of domestication selection in the hatchery environment relative to natural selection in the wild environment.

[6] The *All-H Analyzer* (AHA) tool is a Microsoft Excel® program based on the Beverton-Holt spawner-recruit model. It quantifies the mean number and fate (harvest, hatchery, habitat) of adult recruits each generation. The model and User's Guide are available at *http://www.managingforsuccess.us/site/tools_aha/321/aha.aspx.*

When gene flow occurs between two populations (e.g., hatchery and wild), equation (A6) in the appendix can be extended to the following two, single-generation recursive equations (Ford 2002, eqs. 5 and 6):

$$\overline{P}_W' = p_W\left\{\overline{P}_W + \left[\frac{\overline{P}_W\omega_W^2 + \theta_W\sigma^2}{\omega_W^2 + \sigma^2} - \overline{P}_W\right]h_W^2\right\} + (1-p_W)\left\{\overline{P}_H + \left[\frac{\overline{P}_H\omega_W^2 + \theta_W\sigma^2}{\omega_W^2 + \sigma^2} - \overline{P}_H\right]h_W^2\right\} \quad (1)$$

$$\overline{P}_H' = p_H\left\{\overline{P}_H + \left[\frac{\overline{P}_H\omega_H^2 + \theta_H\sigma^2}{\omega_H^2 + \sigma^2} - \overline{P}_H\right]h_H^2\right\} + (1-p_H)\left\{\overline{P}_W + \left[\frac{\overline{P}_W\omega_H^2 + \theta_H\sigma^2}{\omega_H^2 + \sigma^2} - \overline{P}_W\right]h_H^2\right\} \quad (2)$$

where

$\overline{P}_W'$ and $\overline{P}_H'$ = the mean phenotypic values of wild and hatchery-origin fish, respectively, in the *progeny* generation,

$\overline{P}_W$ and $\overline{P}_H$ = the mean phenotypic values of wild and hatchery-origin fish, respectively, in the *parental* generation,

p_W and $1\text{-}p_W$ = the *proportional genetic contributions* of wild and hatchery-origin parents respectively, to the production of wild (natural-origin) fish in the progeny generation (natural reproduction),

p_H and $1\text{-}p_H$ = the *proportional genetic contributions* of hatchery and wild-origin parents, respectively, to the production of hatchery-origin fish in the progeny generation (hatchery reproduction), and

θ, σ^2, h^2, and ω^2 = the phenotypic optimum, phenotypic variance, heritability, and variance of the fitness function (Fig. 2), respectively, for a quantitative trait, where the subscripts "*W*" and *"H"* for those parameters refer to fish that are the product of natural and hatchery reproduction, respectively.

The parameter p_W can be defined also as the mean proportion of progeny genes in the wild population derived each generation from natural-origin parents. Similarly, the parameter p_H can be defined as the mean proportion of progeny genes in the hatchery population derived each generation from hatchery-origin parents. Equations (1) and (2) are identical to equations (5) and (6) of Ford (2002), except that Ford (2002) assumed that heritabilities in the two environments are equal.

The mean phenotypic value for a trait in each environment (hatchery or wild) is a function of selection acting on each of two components: selection acting on wild and hatchery fish in the wild environment with proportions p_W and $1.0\text{-}p_W$, respectively (eq. 1), and selection acting on hatchery and wild fish in the hatchery environment with proportions p_H and $1.0\text{-}p_H$, respectively (eq. 2). If $p_W = 1.0$, then equation (1) reduces to equation (A6) as a "closed" wild population. Similarly, if $p_H = 1.0$, then equation (2) reduces to equation (A6) as a "closed" hatchery population. When those parameters do not equal 1.0, then selection in one environment can affect phenotypic values and fitness of fish produced via reproduction in the other environment. For a large number of hatchery populations in the Pacific Northwest, p_H equals 1.0 while p_W is less than 1.0 for natural populations. As a result, significant one-way gene flow can occur each generation from a hatchery population to a natural population.

One-way or two-way gene flow between two populations and environments is expected to result in mean phenotypic values for hatchery and/or wild fish that are intermediate to the optimum phenotypic values for each of the two environments (Fig. 2). Stabilizing selection within each environment, coupled with divergent selection between environments, attempts to drive the mean phenotypic value of each population towards their respective optima in each environment. However, gene flow between environments (e.g., hatchery fish spawning naturally) attempts to homogenize populations genetically, thus yielding phenotypic means that are intermediate between the two phenotypic optima. In other words, stabilizing selection drives the mean phenotypic values and underlying gene frequencies of hatchery and wild fish apart towards their respective optima in each of the two environments, whereas gene flow between environments acts to homogenize gene frequencies between them.

If the gene flow parameters ($p_W$and p_H) and phenotypic optima ($\theta_W$and θ_H) are assumed to be constants[7], then - over many generations - a balance between gene flow and selection in the two environments is expected to occur resulting in a *stable equilibrium* in the mean phenotypic values of hatchery and wild fish, respectively. When an equilibrium between selection and gene flow is achieved, then the mean phenotypic values of hatchery and wild fish will not change between generations: $\overline{P}_W' = \overline{P}_W$ and $\overline{P}_H' = \overline{P}_H$.

Setting $\overline{P}_W' = \overline{P}_W = \hat{P}_W$ and $\overline{P}_H' = \overline{P}_H = \hat{P}_H$ in equations (1) and (2) and then solving for $\hat{P}_W$ and $\hat{P}_H$, where $\hat{P}_W$ and $\hat{P}_H$ are the mean phenotypic values of wild and hatchery fish, respectively, at equilibrium, yields the following two equations (after Ford 2002):

$$\hat{P}_W = \frac{\sigma^2\left[\theta_W h^2 + \left(1.0 - h^2\right)\left(\theta_W q_H + \theta_H q_W\right)\right] + \theta_W q_H \omega_H^2 + \theta_H q_W \omega_W^2}{\sigma^2\left[h^2 + \left(1.0 - h^2\right)\left(q_W + q_H\right)\right] + q_W \omega_W^2 + q_H \omega_H^2} \quad (3)$$

$$\hat{P}_H = \frac{\sigma^2\left[\theta_H h^2 + \left(1.0 - h^2\right)\left(\theta_W q_H + \theta_H q_W\right)\right] + \theta_W q_H \omega_H^2 + \theta_H q_W \omega_W^2}{\sigma^2\left[h^2 + \left(1.0 - h^2\right)\left(q_W + q_H\right)\right] + q_W \omega_W^2 + q_H \omega_H^2} \quad (4)$$

where

$q_W = 1.0 - p_W =$ the proportional genetic contribution of hatchery-origin parents to wild progeny each generation (natural reproduction),

[7] In practice, these parameters behave more like random variables than fixed constants, but their variances may vary widely depending on the trait. For example, we might expect the optimum spawn date for a particular natural population to vary widely from year to year depending on seasonal weather conditions. On the other hand, the optimum phenotype for traits related to morphology or egg size may have a relatively low variance and behave more like fixed parameters than random variables. For the purpose of understanding the combined effects of natural selection and gene flow, the aforementioned parameters can be assumed to reflect their long-term averages over many generations.

$q_H = 1.0 - p_H =$ the proportional genetic contribution of wild-origin parents to hatchery progeny each generation (hatchery reproduction),

and σ^2, θ_C, θ_W, h^2, ω_W^2, ω_C^2 are as described previously, but where the heritabilities of the trait are assumed to be equal in the two environments ($h_W^2 = h_H^2 = h^2$).

Equations (3) and (4) are identical to equations (7) and (8), respectively, of Ford (2002) except the terms have been rearranged in equations (3) and (4) above in terms of *1.0-h²* (instead of *h²-1.0*), and with the substitutions $q_W = 1.0 - p_W$ and $q_H = 1.0 - p_H$. These rearrangements show the inherent symmetry of the equilibrium relationships for $\hat{P}_W$ and $\hat{P}_H$: equations (3) and (4) are identical to each other except for the parameter θ_H or θ_W in the first term within brackets in the numerators of the two expressions.

3 Parameterization of the gene flow, selection equations

Equations (3) and (4) are complicated but can be parameterized to yield much simpler expressions. In the classic quantitative genetics model (Falconer and MacKay 1996), the phenotypic distributions of quantitative traits are assumed to be normally distributed ~ $N(\mu, \sigma^2)$ with expected mean value = μ and variance = σ^2 (Note: Non-normal traits can be *normalized* statistically by the appropriate transformation). As noted previously, the magnitude of the difference in the phenotypic optima for any particular trait in the wild and hatchery environments, $|\theta_W - \theta_H|$, is a measure of the strength of domestication selection in the hatchery environment relative to natural selection in the wild environment. Although the exact values of θ_W and θ_H may be unknown for any particular trait, their parameterized difference $\theta_W - \theta_H$ can be set as multiples of σ, the phenotypic standard deviation of the trait, such that $\theta_W - \theta_H = 1.0\sigma$, 2.0σ, or 3.0σ, etc., depending on the trait in question and the amount of domestication selection that may be occurring for any specific or hypothesized trait. If the phenotypic variances (σ^2) are equal for the two populations, then the phenotypic distributions for hatchery and wild fish will overlap by approximately 61%, 32%, or 13% when $\theta_W - \theta_H = 1.0\sigma$, 2.0σ, or 3.0σ, respectively, assuming each population is optimally adapted to the respective environment and no gene flow occurs between them.[8] Consequently, empirical information regarding the amount of overlap between the phenotypic distributions for hatchery and wild fish for one or more traits can be used to establish values of $\theta_W - \theta_H$ relative to σ. Moreover, any normally distributed trait with expected value = μ and variance = σ^2 can be "standardized" by subtracting the expected value of the trait from its observed value and dividing by the square root of the variance (σ = standard deviation). This transformation yields a *standardized* normal distribution with an expected value (μ) = 0 and a variance (σ^2) = 1.0. These latter substitution allowing further simplification of equations (3) and (4) by setting $\sigma^2 = 1.0$, and then establishing values of $\theta_W - \theta_H$ as potential multiples of σ.

[8] The extent of overlap of the phenotypic distributions can be determined easily from tables of the standardized normal distribution when σ^2 is equal in the two populations and the difference in their expected values (means) are expressed as multiples of σ.

If equations (3) and (4) are used to plot $\hat{P}_W$ and $\hat{P}_H$ (y-axis) versus q_W or q_H (x-axis) for various values of $\theta_W - \theta_H$, then one can easily show that the overall shapes of those curves are identical regardless of the actual value of $\theta_W - \theta_H$; only the *scales* (i.e., range of values) of the y-axis for those relationships change.[9] For example, if we assume the value of θ_W is greater than the value of θ_H, then neither $\hat{P}_W$ nor $\hat{P}_H$ can exceed θ_W, nor can they be less than θ_H. Indeed, plots of $\hat{P}_W$ vs. q_W or q_H ($0 \leq q_W$, $q_H \leq 1.0$) will each vary identically between θ_W and θ_H regardless of the actual parameter values of θ_W and θ_H, assuming all other parameters (e.g., h^2) are held constant. This simple relationship between (a) the mean phenotypic values of hatchery and wild fish, respectively, and (b) the gene flow parameters q_W and q_H, allow further simplification of equations (3) and (4). Consequently, for the purpose of evaluating the combined effects of natural selection in the wild environment, domestication selection in the hatchery environment, and gene flow between them, one can set $\theta_W - \theta_H = 1.0\,\sigma$, or simply $\theta_W - \theta_H = 1.0$ for $\sigma^2 = 1.0$. Moreover, one can further set $\theta_W = 1.0$ and $\theta_H = 0$ without changing the *relative values* of $\hat{P}_W$ and $\hat{P}_H$ with respect to each other *or* with respect to the phenotypic optima in the two environments. If heritabilities and selection intensities are further assumed to each be equal in the two environments ($h_W^2 = h_H^2 = h^2$, $\omega_W^2 = \omega_H^2 = \omega^2$), then equations (3) and (4) reduce to the following two simplified expressions:

$$\hat{P}_W = \frac{h^2 + (1.0 - h^2 + \omega^2) \cdot q_H}{h^2 + (1.0 - h^2 + \omega^2) \cdot (q_H + q_W)} \qquad (5)$$

$$\hat{P}_H = \frac{(1.0 - h^2 + \omega^2) \cdot q_H}{h^2 + (1.0 - h^2 + \omega^2) \cdot (q_H + q_W)} \qquad (6)$$

for $\sigma^2 = 1.0$, $\theta_W = 1.0$, and $\theta_H = 0$.

As noted previously, the terms q_W and q_H represent the mean proportional genetic contributions each generation of hatchery and wild fish to natural-origin and hatchery-origin progeny, respectively. In practice, those quantities are very difficult to estimate, particularly for natural populations. Alternatively, one can use the mean proportion of a hatchery broodstock composed of natural-origin fish (*pNOB*) and the mean proportion of naturally-spawning fish composed of hatchery-origin fish (*pHOS*) as approximate

[9] One can easily demonstrate this uniform relationship by setting up plotting routines of $\hat{P}_W$ or $\hat{P}_H$ vs. q_W or q_H, respectively, via equations (3) and (4), and then substituting various values of θ_W and θ_H while holding all other parameters constants. The scale of the y-axis will change, but the shape of the curves will remain constant.

surrogates for q_H and q_W, respectively.[10] These latter substitutions yield the following approximations:

$$\hat{P}_W \approx \frac{h^2 + (1.0 - h^2 + \omega^2) \cdot pNOB}{h^2 + (1.0 - h^2 + \omega^2) \cdot (pNOB + pHOS)} \quad (7)$$

$$\hat{P}_H \approx \frac{(1.0 - h^2 + \omega^2) \cdot pNOB}{h^2 + (1.0 - h^2 + \omega^2) \cdot (pNOB + pHOS)} \quad (8)$$

where

pNOB = mean proportion of a hatchery broodstock composed of natural-origin adults each year, and

pHOS = mean proportion of natural spawners in a watershed or stream composed of hatchery-origin adults each year.

$\hat{P}_W$ and $\hat{P}_H$ in equations (7) and (8) will each vary between $\theta_H = 0.0$ and $\theta_W = 1.0$ depending on the relative values of *pNOB* and *pHOS*. Also, $\hat{P}_W$ will always equal $\theta_W = 1.0$ if *pHOS* = 0, and $\hat{P}_H$ will always equal $\theta_H = 0.0$ if *pNOB* = 0. In other words, a wild population will be optimally adapted to a natural environment if no hatchery fish spawn naturally, and a hatchery population will be optimally adapted to the hatchery environment if no wild fish are included with the broodstock. Equations (7) and (8) quantify those relationships for traits where $\theta_H \neq \theta_W$.

4 Proportionate Natural Influence (*PNI*)

When the phenotypic distributions of hatchery and wild fish are *standardized* with $\theta_H = 0.0$ and $\theta_W = 1.0$, as was done for equations (5) through (8) above, then $\hat{P}_W$ and $\hat{P}_H$ can be interpreted as the *proportional genetic influence* of the natural environment on the mean phenotypic values of wild and hatchery fish, respectively. Thus, equations (7) and (8) can be further generalized to the following two expressions:

$$PNI_{Wild} \approx \frac{h^2 + (1.0 - h^2 + \omega^2) \cdot pNOB}{h^2 + (1.0 - h^2 + \omega^2) \cdot (pNOB + pHOS)} \quad (9)$$

$$PNI_{Hatch} \approx \frac{(1.0 - h^2 + \omega^2) \cdot pNOB}{h^2 + (1.0 - h^2 + \omega^2) \cdot (pNOB + pHOS)} \quad (10)$$

[10] The acronyms *pNOB* (proportion of *natural-origin broodstock*) and *pHOS* (proportion of *hatchery-origin spawners*) were first proposed in 2004 by Craig A. Busack, Washington Department of Fish and Wildlife, Olympia, WA, at an HSRG workshop held in Seattle, Washington, USA.

where *PNI* refers to the *proportionate natural influence* of the wild environment on the mean phenotypic values and genetic constitutions of wild (eq. 9) and hatchery (eq. 10) fish, respectively.[11] *PNI* varies from 0.0 to 1.0, where *PNI* = 0.0 or 1.0 imply that the genetic constitution and mean phenotypic values for a population are influenced only by the hatchery or natural environment, respectively.

PNI values for hatchery and wild fish will not be identical (eqs. 9 and 10). This difference occurs, even at equilibrium with two-way gene flow, because wild fish always have one extra generation of reproduction and selection (*natural*) in the wild environment, while hatchery fish always have one extra generation of reproduction and selection (*domestication*) in the hatchery environment. As a result, PNI_{Wild} will always be greater than zero, and PNI_{Hatch} will always be less than 1.0. For example, if *pHOS* = 1.0 and *pNOB* = 0, then $PNI_{Wild} = h^2/(1.0 + \omega^2)$, which is its lowest possible value (eq. 9). Similarly, if *pNOB* =1.0 and *pHOS* =0, $PNI_{Hatch} = 1.0 - h^2/(1.0 + \omega^2)$, which is its highest possible value (eq. 10).

5 Genetic consequences of gene flow between hatchery and wild populations

The relationships among PNI_{Wild}, PNI_{Hatch}, *pHOS*, and *pNOB* (eqs. 9 and 10) are illustrated in Figures 3 through 8 for various values of h^2 and ω. Two sets of heritabilities were used for generating those graphs: $h^2 = 0.2$ (*moderate* heritability) and $h^2 = 0.5$ (*high* heritability). Similarly, two selection intensities were used to generate Figures 3 through 8: $\omega = 10\sigma$ (*weak* selection) and $\omega = 3\sigma$ (*strong* selection). As noted in Appendix A, $\omega^2 = 100\sigma^2$ ($\omega = 10\sigma$) is considered *weak selection*, and $\omega^2 = 10\sigma^2$ ($\omega = 3.16\sigma$) is considered *strong selection* (Lande 1976; see also Fig. 2). The phenotypic variance (σ^2) was set equal to 1.0 in all plots based on a standardized normal distribution (eqs. 3 and 4).

The first conclusion to be drawn is that relatively small amounts of one-way gene flow between the hatchery and wild populations, continuously over many generations, can have a rather profound genetic effect on the recipient population (Figs. 3 and 4). When *pNOB* = 0 and a hatchery broodstock is composed of only hatchery-origin adults each year, the natural spawning of hatchery fish over many generations can significantly reduce *PNI* for wild fish (PNI_{Wild}), even for relatively low values of *pHOS* (Fig. 3). For example, when *pNOB* equals zero, a value of *pHOS* equal to only 0.05 (5%) results in $PNI_{Wild} < 0.5$ in all cases except when heritabilities and selection intensities are both high ($h^2 = 0.5$; $\omega = 3\sigma$, Fig. 3). Similarly, one-way gene flow from the natural environment to the hatchery environment can significantly increase *PNI* for hatchery-origin fish (PNI_{Hatch}) if *pHOS* equals zero (Fig. 4). Figures 3 and 4 also show that selection intensity has a greater influence than heritability on the shape of the *PNI* curves: as the value of ω decreases, selection intensity increases (Fig. 2), thereby increasing the ability of selection to resist the homogenizing effects of gene flow between populations. Figure 4 is clearly the mirror image of Figure 3, reflecting the symmetry of equations (3) and (4), and equations (9) and (10).

The relationship between PNI_{Wild} and *pHOS* for varying values of *pNOB* is particularly important for assessing long-term genetic risks of hatchery programs to naturally

[11] The term *proportionate natural influence* (*PNI*) was first proposed in 2004 by Craig A. Busack, Washington Department of Fish and Wildlife, Olympia, WA, at an HSRG workshop held in Seattle, Washington, USA.

spawning populations (Figs. 5 and 6). When *pHOS* is greater than 5% (0.05), then wild fish must be included with a hatchery broodstock to achieve $PNI_{Wild} > 0.5$ for traits with moderate heritability and high selection intensity (Figs. 5 and 6). Indeed, increasing the proportion of a broodstock composed of wild fish from *pNOB* = 0 to *pNOB* = 0.1 can increase PNI_{Wild} substantially, but only if *pHOS* is less than 30% (bottom two curves in Fig. 5; Fig. 6). However, *pNOB* must exceed *pHOS* to ensure a value of PNI_{Wild} greater than 0.5, the value at which the hatchery environment is having a 50% influence on the genetic make-up of a naturally spawning population. Moreover, increasing *pNOB* from 0.5 to 1.0 for *pHOS* > 0.3 is not nearly as effective at increasing PNI_{Wild} as increasing *pNOB* from 0 to 0.5 for *pHOS* < 0.3 (Fig. 6). In other words, the effectiveness of including wild fish in a hatchery broodstock to increase PNI_{Wild} decreases rapidly as *pHOS* increases (Figs. 5 and 6). These results indicate that, over a broad range of possible *pHOS* values, decreasing *pHOS* is a much more effective method for increasing PNI_{Wild} than increasing *pNOB*. These graphs also demonstrate the expected result that PNI_{Wild} and PNI_{Hatch} will both equal approximately 0.5 when *pNOB* = *pHOS*.

In practice, the abundance and viability of a naturally spawning population may limit the number of wild fish available for broodstock, further restricting the upper value of PNI_{Wild}. For example, if *pNOB* = 0.1, then the relationship between PNI_{Wild} and *pHOS* approximates a negative exponential such that all values of *pHOS* greater than approximately 30% result in very low *PNI* values (Fig. 5). In this latter situation, a naturally spawning population composed of 30% hatchery-origin fish over many generations is nearly equivalent genetically to a naturally spawning population composed of 100% hatchery-origin fish with no natural-origin spawners. In this case, a 10% gene flow rate from the natural environment to the hatchery environment is unable to compensate genetically for the large proportion of naturally spawning fish composed of hatchery fish. These results further illustrate the need to reduce *pHOS*, not increase *pNOB*, as the most effective way to increase PNI_{Wild}. These results also demonstrate the desirability of maintaining *pHOS* below a maximum value of 20-30% to achieve a value of $PNI_{Wild} > 0.5$, but only if wild fish can be included in the broodstock at a rate that allows *pNOB* to exceed *pHOS* (Fig. 5). Ultimately, the viability and abundance of a naturally spawning population will determine the absolute number of wild fish that can be included in a hatchery broodstock to maintain the desired *PNI* value for both hatchery and natural-origin fish.

When *pNOB* and *pHOS* are both greater than zero, the shapes of the *PNI* curves for wild and hatchery fish (PNI_{Wild} and PNI_{Hatch}, respectively) will be similar but not identical (Figs. 7 and 8; see also eqs. 9 and 10). The close similarity of PNI_{Wild} and PNI_{Hatch} under conditions of two-way gene flow is somewhat independent of the heritability of the trait. However, PNI_{Wild} and PNI_{Hatch} can differ substantially for traits under *strong selection*, particularly when *pNOB* or *pHOS* equal zero (Figs. 3 and 4).

6 Approximate *PNI* index

The close similarity of PNI_{Wild} and PNI_{Hatch} over a broad range of values for *pHOS* and *pNOB*, particularly when both are greater than zero (Figs. 7 and 8), suggests an approximation for *PNI* that can be used to quickly assess, with very few assumptions, the genetic risks posed by a hatchery population to a natural population:

$$PNI_{Approx} = \frac{pNOB}{pNOB + pHOS} \quad (11)$$

where PNI_{Approx} refers to an approximate value of PNI for both hatchery and wild fish in a particular watershed or geographic area.[12] The elegance of equation (11) is that it requires no assumptions regarding selection intensities or heritabilities associated with any specific trait; it simply approximates the relative influences of the natural and hatchery environments on the genetic constitution and mean phenotypic values of hatchery and wild fish when gene flow occurs between them (Figs. 9 and 10). PNI_{Approx} will be more similar to PNI_{Wild} when $pHOS < pNOB$ and more similar to PNI_{Hatch} when $pHOS > pNOB$ (Figs. 9 and 10). Moreover, PNI_{Approx} will always be slightly lower than PNI_{Wild} for all values of $pHOS$ if $pNOB > 0$.

Equation (11) can be used to calculate an approximate value of PNI_{Wild} (or PNI_{Hatch}) if $pNOB$ and $pHOS$ are both greater than zero. If $pNOB = 0$, then $PNI_{Hatch} = 0$ and equation (9) should be used to calculate PNI_{Wild}, assuming values for h^2 and ω similar to those presented here for this paper. Similarly, if $pHOS = 0$, then $PNI_{Wild} = 1.0$ and equation (10) should be used to calculate PNI_{Hatch}. Situations where $pHOS = 0$ and $pNOB > 0$ – that is, where no hatchery fish are spawning naturally, but wild fish are systematically included in a broodstock each year (or each generation) - are expected to be relatively rare, whereas the converse situations where $pNOB = 0$ and $pHOS > 0$ are known to be common. In these latter situations ($pNOB = 0$), equation (9) should be used to calculate PNI_{Wild} for the purpose of assessing genetic risks of a hatchery program to a natural population. Equation (9) should also be used if hatchery fish spawning naturally represent strays from another watershed, even for $pNOB > 0$ for that out-of-basin hatchery stock. In this latter situation, $pNOB$ should be set equal to zero ($pNOB = 0$) in equation (9) because the naturally-spawning population of interest makes no direct genetic contribution to the out-of-basin hatchery population that is spawning in the recipient watershed.

7 HSRG application of the selection and gene flow model

The HSRG has applied equations (1) and (2) to Beverton-Holt spawner-recruitment equations in the *AHA* model to adjust the number of natural-origin and hatchery-origin adult recruits returning each year to a watershed (see Appendix C of this HSRG report). The mean phenotypic values (eqs. 1 and 2) generated during each iteration of the *AHA* model are used to calculate a mean relative fitness ($\overline{F}$) of wild and hatchery fish each generation according to the following equations (eq. 3 of Ford 2002):

[12] *PNI = pNOB/(pNOB+pHOS)* was first proposed in 2004 by Craig A. Busack, Washington Department of Fish and Wildlife, Olympia, Washington, USA as a working index based on the equations provided by Ford (2002) and computer iterations that converged approximately to that relationship when *pNOB* and *pHOS* were both greater than zero. The HSRG adopted this index as a simple measure to assess the genetic risks of *genetically integrated* hatchery programs where wild fish are included in a broodstock and *pNOB* is greater than zero (Mobrand et al. 2005).

$$\overline{F}_W = e^{-\frac{1}{2}\frac{(\overline{P}_W-\theta_W)^2}{(\omega^2+\sigma^2)}} \quad (12)$$

$$\overline{F}_H = e^{-\frac{1}{2}\frac{(\overline{P}_H-\theta_H)^2}{(\omega^2+\sigma^2)}} \quad (13)$$

where $\overline{F}_W$ and $\overline{F}_H$ are the mean fitnesses of wild and hatchery fish, respectively, in a particular generation. The *AHA* model then apportions those mean fitnesses across each life history stage for each group of fish (hatchery or wild) to yield an adjusted number of hatchery and natural-origin progeny for each of those life history stages (eqs. 3 and 4 of Appendix C). Continued iterations of equations (1), (2), (12) and (13) presented here allow fitness effects in each parental generation to affect the mean fitness and number of adult recruits in each progeny generation via the Beverton-Holt spawner-recruit equations (see Appendix C for details). *AHA* then provides the expected mean number of adult recruits (both hatchery and wild) each year at equilibrium after many generations of iterations. This mode of selection, as implemented in *AHA*, is commonly called *hard selection* because population abundances are adjusted according to their mean relative fitnesses (Demeeus et al. 1993).

The HSRG used parameter values for the fitness functions in *AHA* that simulate traits of high heritability (h^2 = 0.5) and high selection intensity ($\omega^2 = 10\sigma^2$) in both the hatchery and natural environments. These types of traits are expected to undergo the quickest selection responses over the shortest number of generations. The equilibrium trait values resulting from those simulations (Figs. 12 and 13) yield graphs virtually identical to the *PNI* graphs for standardized traits (Figs. 5 and 6). As noted previously, the shapes of the equilibrium curves generated from equations (3) and (4) are largely independent of the optimum phenotypic values (θ_W and θ_H) and variance for the trait; rather, those curves are determined primarily by the relationship between *pNOB* and *pHOS* (q_W and q_H) and secondarily by the heritability and selection intensity of the trait (eqs. 5 and 6). These latter results (Figs. 12 and 13) further justify the use of equations (9) and (10) – and, more generically, equation (11) – to evaluate the genetic risks of hatchery programs to naturally spawning populations of salmon and steelhead in the Pacific Northwest.

8 Discussion

Many traits of anadromous salmonid fishes potentially have very different optimum values for hatchery and wild fish, especially traits subject to selective breeding by hatchery personnel (e.g., return and spawn dates of fish selected for broodstock) and traits related to natural reproduction that are relaxed in the hatchery environment (e.g., spawning behavior; see Quinn 2005 for an excellent discussion of this issue). If no *gene flow* occurs between the hatchery and natural environments, then stabilizing selection in each environment will drive the phenotypic means of each population towards their respective optima; that is, in the absence of gene flow between the two environments, hatchery and wild fish will represent two reproductively distinct populations, each *locally adapted* to their respective environments. However, if hatchery fish spawn naturally and/or wild fish are included with the broodstock each generation, then – over time – the mean phenotypic values of hatchery and/or wild fish will be influenced by the selection,

natural or *domestic*, in the other environment. The net result is that the mean phenotypic values of one or both groups of fish will be intermediate to the phenotypic optima in the two environments. The phenotypic fitness model of Ford (2002) allows assessment of those predicted effects as a function of *pNOB* and *pHOS*.

Lynch and O'Hely (2001) developed an alternative model for assessing the long-term fitness effects of captively bred populations reproducing in natural environments. Their analysis was based on relaxation of natural selection in a captive (hatchery) environment and the accumulation of mutations in the captive population that would otherwise be deleterious and selected against in the natural environment. Despite this different approach, the overall results of Lynch and O'Hely (2001) are amazingly similar to those of Ford (2002), as described here. In the model of Lynch and O'Hely (2001), the relative fitness of the natural population is largely a function of the percent of time that genes spend in the natural environment versus the hatchery environment, a quantity similar to *PNI*. Lynch and O'Hely (2001) also found that increasing the proportion of a broodstock composed of natural-origin adults (*pNOB*) from 0.5 to 1.0 had only a minor genetic benefit - relative to increasing pNOB from zero to 0.5 - at increasing the overall mean fitness of a natural population, a result again similar to that described here based on the model of Ford (2002). Similarly, Lynch and O'Hely (2001) found that reducing *pHOS* from 0.3 to 0.1 had a much greater effect at reducing the segregation load (or increasing mean fitness) of the natural population than reducing *pHOS* from 0.5 to 0.3. These parallel results reinforce the conclusions resulting from the model described by Ford (2002).

Many fishery biologists have suggested that the intensity of domestication selection in the hatchery environment must be low for anadromous salmonid fishes, particularly for species that spend only a few months in captivity prior to their release as smolts (e.g., "ocean-type" Chinook salmon). However, even for species that spend only a few weeks in freshwater prior to release from hatcheries and outmigration to saltwater (e.g., pink and chum salmon, *O. gorbuscha* and *O. keta,* respectively), natural spawning traits related to reproductive fitness have no natural environmental component for hatchery produced fish. Indeed, these latter traits are exactly the kind of traits specifically modeled by Lynch and O'Hely (2001). Artificial spawning in a hatchery can inadvertently impose unknown selection on hatchery populations, eliminate natural selection on traits essential for natural reproduction, while also reducing the genetic effective number of breeders (Campton 2004, 2005; Quinn 2005). Moreover, "natural selection" in a hatchery pond during the freshwater rearing phase can have a significant effect on smolt-to-adult survivorship during the post-release life history phases. For example, the size of fish at the time of release from a hatchery is positively correlated with post-release survival and adult return rates, suggesting that hatchery fish better adapted to hatchery culture have a post-release selective advantage in the wild (Reisenbichler et al. 2004).

The homing instinct of anadromous salmonid fishes provides an evolutionary genetic mechanism for maximizing fitness and development of local adaptations (Quinn 1993; Kinnison et al. 2001; Quinn et al. 2006). Many studies have further demonstrated a genetic component to homing (Bams 1976; McIsaac and Quinn 1988; Pascual et al. 1995; Candy and Beacham 2000; Stewart et al. 2002; Dukes et al. 2004). In general, based on controlled breeding studies, fish reared and released in their natal streams and watersheds exhibit higher homing fidelity than fish of the same population reared and released outside their natal watersheds. These latter results are consistent with *a priori* expectations that homing confers a higher mean fitness to fish that return to spawn in

areas where their parents reproduced successfully compared to fish that "stray" and spawn randomly elsewhere (Hendry et al. 2000). Many biologists have long recognized that subtle variations in the life histories of anadromous salmonid fishes can be attributed to local adaptations that appear to reflect evolutionary responses to stream specific hydrologies, water temperatures during the incubation phase, and geographic location (Hendry et al. 1998; Brannon et al. 2004; Keefer et al. 2004). These traits include date of reentry to freshwater and spawn date of adult fish, age and size at sexual maturity, fecundity and egg size of female parents, pre-hatch developmental rates of embryos, length of freshwater residence prior to outmigration, and marine migration patterns (e.g., Smoker et al. 1998). In some cases, entire geographic races have evolved in response to geographic location, hydrology, and local water temperatures (Waples et al. 2004).

The general results of the Ford (2002) model presented here, and modeled by the HSRG via *AHA*, assumed that heritabilities and selection intensities in the hatchery and wild environments were equal. In practice, the values of these parameters for some traits may differ substantially between the two environments. Selection intensity, as measured by $1/\omega^2$, is proportional to the *force* of stabilizing selection that resists genetic change and maintains phenotypic means as close as possible to the phenotypic optima for each environment. Similarly, heritability is a measure of the *efficiency* of selection acting on phenotypic variation within a population to effect genetic changes between generations. As selection intensity and heritability of a trait in a particular environment increase, the magnitude of gene flow into that population must also increase to achieve the same genetic and phenotypic outcome. For example, if the heritability of a trait is substantially greater in the hatchery environment than in the natural environment, then *pNOB* would need to exceed *pHOS* to achieve $PNI = 0.5$ because the higher efficiency of selection in the hatchery environment will be able to better resist the genetic effects of gene flow from the natural environment. Similarly, if selection intensity in the hatchery environment is greater than selection intensity in the natural environment for a particular trait, then *pNOB* will also need to exceed *pHOS* to achieve a value of $PNI = 0.5$. On the other hand, if the heritability or selection intensity on a trait are greater in the natural environment than in the hatchery environment, then a value of *pNOB* less than *pHOS* could achieve a value of $PNI = 0.5$. In practice, based on our fundamental understandings of population biology and how selection operates, one might predict – for a large number of traits related to fitness - that heritabilities in the hatchery environment may exceed those in the natural environment, but selection intensities in the natural environment may exceed those in the hatchery environment. The counteracting effects of those two unequal forces in the two environments could lead to the situation where a value of *pNOB* approximately equal to *pHOS* yields a value of $PNI \approx 0.5$ for a large number of traits.[13] The following table summarizes the necessary relationships between *pNOB* and *pHOS* to achieve $PNI = 0.5$ when heritabilities (h^2) and selection intensities ($1/\omega^2$) may not be equal in the two environments. As noted previously, the magnitude of selection intensity within each environment is proportional to $1/\omega^2$ (Fig. 2).

[13] Sensitivity analyses performed by Craig A. Busack, Washington Dept. of Fish and Wildlife, Olympia, Washington, indicate that values of *PNI* are fairly robust to violation of the assumption that heritabilities and selection intensities are equal in the two environments.

Table 1. Relative values of *pNOB* and *pHOS* to achieve *PNI* = 0.5 when heritabilities (h^2) and selection intensities (~1/ω^2) differ between natural (*W*) and hatchery (*H*) environments.

	$\omega_H^2 = \omega_W^2$	$\omega_H^2 < \omega_W^2$	$\omega_H^2 > \omega_W^2$
$h_H^2 = h_W^2$	*pNOB = pHOS*	*pNOB > pHOS*	*pNOB < pHOS*
$h_H^2 > h_W^2$	*pNOB > pHOS*	*pNOB >> pHOS*	*pNOB ≈ pHOS?*[14]
$h_H^2 < h_W^2$	*pNOB < pHOS*	*pNOB ≈ pHOS?*	*pNOB << pHOS*

The HSRG has concluded that all hatchery programs for Pacific salmon and steelhead must be classified as either *integrated* or *segregated* (Mobrand et al. 2005). The HSRG defines these terms as follow:

- A hatchery population is defined as *segregated* if it is propagated as a "closed" population where only hatchery-origin fish are used, or are intended to be used, for broodstock;
- A hatchery population is defined as *integrated* if it systematically - and purposefully - includes natural-origin fish in the broodstock, or the intent of the program is to purposefully include natural-origin fish in the broodstock, with the goal of maintaining genetic continuity and phenotypic similarity with a specific natural population.

The segregated and integrated strategies yield very different broodstock goals and propagation protocols. The segregated strategy creates a genetically-distinct, hatchery-adapted population, whereas the integrated strategy attempts to increase the abundance of fish representing an existing natural population.

Both the integrated and segregated strategies have their strengths and weaknesses. If hatchery fish can be precluded from spawning naturally, then the segregated approach may be favored if the primary purpose of the hatchery program is to produce fish for harvest. The segregated strategy will maximize the fitness of hatchery fish adapted to artificial propagation, and the genetic risks of those hatchery fish to natural populations will be minimal if – but only if - *pHOS* is near zero. However, in most instances, the natural spawning of hatchery fish cannot be precluded, and large numbers of fish from segregated hatchery populations escape harvest and broodstock recapture, thus resulting in relatively high values of *pHOS* (>10%) in many watersheds. As noted previously, the long-term genetic effects of hatchery fish spawning naturally over many generations become significant when *pHOS* approaches and exceeds 5%, particularly when pNOB = 0. One goal of the *integrated* strategy is to reduce those risks by increasing the effective *PNI* for hatchery fish where the natural spawning of those fish cannot be precluded. The

[14] The HSRG suggests heritabilities are likely to be greater in the hatchery environment than in the natural environment, but that selection intensities in the natural environment are likely to be greater in the natural environment than the hatchery environment. Under these circumstances, approximately equal levels of gene flow between the two environments may be sufficient to achieve *PNI* = 0.5.

integrated strategy is also favored for hatchery programs intended to assist with the conservation or recovery of natural populations (e.g., Olson et al. 2005). However, integrated hatchery programs inherently impose their own *demographic risks* to natural populations by "harvesting" wild fish for broodstock under the premise that the recruit-per-spawner ratio (*R/S*) is substantially greater for wild fish spawning in a hatchery than in nature. Moreover, natural populations must be viable and self-sustaining to support a "properly-integrated" hatchery population where *pNOB* - at a minimum - exceeds *pHOS*. In general, reducing *pHOS* is a much more effective and efficient method of increasing *PNI* than increasing *pNOB*. For example, increasing *pNOB* above 0.5 is expected to confer a comparatively minor genetic benefit to a naturally spawning population (Lynch and O'Hely 2001; Ford 2002; this paper) but could substantially increase demographic risks to a natural population depending on the size of the hatchery program and the total number of adult fish collected for broodstock. [15]

Minimizing *risks* of hatchery programs to natural populations of salmon and steelhead is a major goal of hatchery reform in the Pacific Northwest (Mobrand et al. 2005). As a consequence, the HSRG has established management guidelines for *PNI*, *pHOS*, and *pNOB* to minimize genetic risks to naturally spawning populations. These guidelines are based primarily on the relationships illustrated in Figs. 3 through 10.

8.1 Management guidelines for segregated hatchery programs (pNOB ≈ 0)

- **Maintain *pHOS* < 5%.**
- When *pHOS* > 5%, either (a) reduce the size of the hatchery program and/or (b) implement new measures to recapture hatchery-origin fish to reduce pHOS to <5%.

8.2 Management guidelines for integrated hatchery programs (pNOB > 0)

- **Maintain *PNI* > 0.5.** *PNI* must exceed 0.5 in order for the natural environment to have a greater influence than the hatchery environment on the genetic constitution of a naturally-spawning population. In general, this guideline requires *pNOB* > *pHOS*.[16]
- **Maintain *pHOS* < 30%.** The effectiveness and efficiency of *pNOB* for maintaining *PNI* > 0.5 decreases significantly for values of *pHOS* > 30%. Consequently, to achieve a desired *PNI* > 0.5, it is much more efficient – and less risky biologically - to reduce *pHOS* than increase *pNOB*. Increasing *pNOB* for high values of *pHOS*, as opposed to decreasing *pHOS*, imposes additional demographic (and potential genetic) risks to naturally spawning populations with comparatively minor increases in *PNI*.
- **Maintain *PNI* > 0.67 for natural populations considered essential for the recovery or viability of an *Evolutionarily Significant Unit* (ESU) of Pacific**

[15] One exception to this generalization might occur when the natural population is highly imperiled or at risk of demographic extinction. In this situation, the demographic risks to the natural population may outweigh the genetic risks, and a value of pNOB = 1.0 may be desired or necessary to reduce those demographic risks.

[16] This guideline and constraint also require a minimum pNOB > 0.10, even for values of pHOS < 0.10 (Figs. 3 and 4). One goal of an integrated hatchery program is to maintain genetic continuity and phenotypic similarity to a naturally-spawning population, and this goal requires a minimum *pNOB* ≥ 10%.

salmon or *Distinct Population Segment* (DPS) of steelhead, as those terms are defined and designated under the U.S. Endangered Species Act (ESA). The HSRG has adopted the term "primary" for natural populations considered by NOAA Fisheries[17] to be essential for the recovery of an ESU or DPS of Pacific salmon or steelhead, respectively. That designation requires a much more stringent constraint on *PNI*.

The HSRG considers the preceding guidelines as *minimal* requirements for minimizing the genetic risks of hatchery programs to naturally spawning populations. For example, a value of *pHOS* = 6% from a segregated hatchery population should not be viewed as exceeding the pHOS < 5% guideline by only 1%; on the contrary, a value of pHOS = 6% for a segregated hatchery population should be viewed as posing a significant, long-term genetic risk to the viability of a naturally spawning population if that potential level of gene flow continues unabated for many generations. Moreover, the aforementioned guidelines should not be interpreted as "benchmarks" or "goals"; rather, they should be interpreted in the context of their presentation here with respect to Figs. 3 through 10: that is, violation of any of those guidelines on a sustained basis over many generations will pose long-term genetic risks to the future viability of naturally-spawning populations.

8.3 Exceptions to the guidelines

The HSRG recognizes that many natural populations of Pacific salmon and steelhead, particularly in watersheds significantly impacted by hydropower and land use practices (e.g., logging, agriculture), may not be viable or self-sustainable at the present time. The HSRG further recognizes that hatcheries and artificial propagation can play critically-important roles at conserving genetic resources and maintaining naturally-spawning populations in areas where significant habitat impacts have occurred. In some instances, the future survival of a naturally-spawning population may require significant increases in natural productivity and recruit per spawner (R/S) ratios, measured as the mean number of natural-origin adult recruits per natural-origin adult spawner in the preceding generation. Such desired increases may not be possible under current conditions.

Consequently, the HSRG acknowledges that some hatchery programs may be required to perform a "life support" function to prevent *functional extirpation* of a naturally spawning population in particular watersheds or geographic areas. Moreover, the abundance of fish representing a natural population must be sufficiently high to allow selection in the natural environment to be an effective deterministic force towards maximizing mean population fitness in view of stochastic forces. Under these exceptional circumstances, maintaining a naturally-spawning component to a hatchery-sustained population – where the number of hatchery fish spawning naturally exceeds HSRG guidelines - may be desirable for both genetic and demographic reasons. In practice, such situations need to be clearly identified and evaluated carefully on a case-by-case basis. Deliberately allowing "surplus" hatchery fish to spawn naturally under the premise of "increasing natural production" (ISAB 2002; Brannon et al. 2004) is not the same justification as preventing local extirpation of an imperiled population; the former poses significant genetic risks whereas the latter confers conservation benefits.

[17]National Marine Fisheries Service, National Oceanic and Atmospheric Administration (NOAA), U.S. Department of Commerce, Washington, DC, USA.

9 Conclusions

Hatchery-origin fish spawning naturally over many generations pose significant long-term genetic risks to natural populations of Pacific salmon and steelhead. Those risks are primarily a function of the mean proportion of a naturally-spawning population composed of hatchery-origin fish each year. Those risks are also a function of the genetic history of the hatchery broodstock over the preceding generations.

When the genetic risk guidelines presented here are violated, the most expeditious and biologically efficient solution is to reduce the number of hatchery-origin fish spawning naturally. This can be accomplished by a number of methods, the simplest of which is to reduce the size of the hatchery program and the number of hatchery-origin fish that are released, at least until other solutions can be implemented (e.g., construction of a weir at a hatchery, implementation of *mass marking* of hatchery fish coupled with intense selective fisheries on hatchery fish).

Genetically-*integrated* hatchery populations can reduce genetic risks to naturally spawning populations, and they can also provide long-term conservation benefits, but they also impose additional *demographic* risks to naturally spawning populations that are not imposed by *segregated* programs. Consequently, reducing *pHOS* should be considered the first management option of choice – rather than increasing *pNOB* - whenever the genetic risk guidelines presented here are violated.

A careful evaluation of the viability of a naturally spawning population, and its biological capability to adequately support a genetically-integrated hatchery program, will be necessary before a segregated hatchery program is converted to an integrated one under the umbrella of "hatchery reform". In most cases, a *sliding scale* may be necessary to adjust the number of natural-origin fish retained for broodstock each year based on the abundance of natural-origin recruits returning to a watershed (e.g., Olson et al. 2005). In all cases, either *pHOS* needs to be maintained at less than 5% (*segregated* programs) or PNI needs to exceed 0.5 to 0.67 (*integrated* programs) to minimize genetic risks to natural populations.

Violations of the guidelines presented here over many generations may jeopardize the future viability and self-sustainability of a natural population. Ultimately, implementation of the HSRG guidelines may represent trade-offs between maintaining benefits and reducing risks of a hatchery program. If resource managers intentionally do not rectify violations of biological guidelines in order to maintain perceived benefits - regardless of whether those guidelines are genetic guidelines, fish health guidelines or other guidelines intended to protect the viability of a biological resource - then those managers need to justify their actions to the scientific community and the general public. Resource managers need to be accountable for their decisions when they contradict established biological principles.

In the long run, resource managers should follow three principles established by the HSRG for hatchery programs: (1) explicitly state the goals of each hatchery program quantitatively in terms of desired or intended benefits; (2) provide scientific justification for each hatchery program through appropriate benefit-risk analyses, including scientific justification of all the methods and protocols (e.g., spawning protocols, rearing protocols) associated with execution of the program; and (3) monitor and evaluate the program annually to determine whether the intended benefits are realized, and whether biological risks exceed established guidelines. The information obtained from (3) should then be used to adjust the program on a regular basis with the goal of increasing benefits and/or

reducing risks. This three step process is nothing less than the foundation of hatchery reform in the Pacific Northwest.

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11 Appendix: Quantitative genetic foundations: one population, one environment

Ford (2002) described a deterministic model that is based on the foundation principles of quantitative genetics and modern animal breeding (Bulmer 1985; Falconer and MacKay 1996). Under those principles, the phenotypic distribution of a quantitative trait (e.g., spawn date, run timing, female fecundity, etc.) within a population is assumed to be distributed normally ~N(u, σ^2) with an expected value (population mean) = u and variance = σ^2 (Falconer and MacKay 1996). The phenotypic variation among individuals in a population, measured by σ^2, is assumed to be caused by (a) heritable genetic variation among those individuals, commonly referred to as the *additive genetic variance*, (b) non-heritable genetic variation among individuals associated with interaction effects among alleles within and between loci (e.g., *dominance* and *epistasis*), and (c) environmental variation among individuals, including genotype x environment interaction effects. Under this model, genetic variation is assumed to be caused by allelic (Mendelian) variation at a large number of genes that directly affect the trait in question. The "environment" refers to all non-genetic influences experienced by an individual from the time of fertilization (conception) to the time of death. Variation in those "experiences" is the source of environmental variation.

Under the classic genetic model, the phenotypic value (P) of a trait for an individual is assumed to be sum of the genetic (G) and environmental effects on that trait, plus genetic-environment interaction (I) effects (GxE) for that individual (i.e., $P = G + E + I$). GxE effects occur when the relative phenotypic values of different genotypes vary or change among different environments (e.g., genotype "A" grows faster than genotype "B" in environment "C" but genotype "B" grows faster in environment "D"). Consequently, phenotypic values of individuals are not simply an additive function of genetic and environmental effects. Genetic and environmental variation among individuals within a population, plus variation in the *GxE* interaction effects among individuals (i.e., genotypes), results in measurable phenotypic variation (e.g., spawn date) among individuals, and that variation generally follows a "bell-shaped" curve that closely approximates a normal distribution.

The phenotypic variance among individuals in a population (σ^2) can be partitioned into its causal components:

$$\sigma^2 = \sigma_G^2 + \sigma_E^2 + \sigma_I^2 \qquad \text{(A1)}$$

where σ_G^2 = the *additive genetic variance* among individuals that can respond to artificial or natural selection that can result in a change in the mean value of a trait between the parental and offspring generations, σ_E^2 = the *environmental variance* among individuals, and σ_I^2 = variance in non-additive genetic effects and all genetic-environmental interaction effects.[18]

[18] The covariances between genetic effects and between genetic and environmental effects have been ignored in eq. (1). For example, a covariance between genetic and environmental effects occurs when faster growing genotypes are provided more food than slower growing genotypes, thus resulting in a positive covariance between genotype and environment for the population as a whole.

Another important parameter is the *heritability* (h^2) of a trait ($h^2 = \sigma_G^2 / \sigma^2$) which measures the *proportion of the total phenotypic variance among individuals due to additive genetic variation among those individuals* ($h^2 = \sigma_G^2 / \sigma^2$; $0 \leq h^2 \leq 1.0$). In general, heritabilities of most traits related to fitness (e.g., age and size at sexual maturity, spawn date, etc.) range from approximately 0.1 to 0.3 and rarely exceed 0.5 (Carlson and Seamons 2008).

The heritability of a trait is both population-specific and environment-specific because its value is a direct function of the amount of additive genetic variance within a specific population (numerator of h^2) *and* the amount of environmental variance contributing to the phenotypic variance among those individuals within that population (denominator of h^2). Hence, any reduction in the environmental variance experienced by individuals within a population will increase the heritability of a trait because a greater proportion of the observed phenotypic variation will be due to genetic variation among individuals within that population, all other factors remaining equal. In this context, geneticists have hypothesized that many traits related to fitness in Pacific salmon may have higher heritabilities in hatchery-propagated populations than natural populations because of the potentially lower environmental variances associated with hatchery environments versus natural environments. Also, a low heritability does not necessarily mean that phenotypic variation in the trait is not under significant genetic control because high environmental variation could simply be contributing to the majority of the observed phenotypic variation.

The heritability of a trait, estimable from controlled breeding studies or populations that are pedigreed, can be used to predict a one-generation response (R) to selection (natural or artificial) according to the following expression:

$$R = \overline{P}' - \overline{P} = h^2(\overline{P}_S - \overline{P}) \qquad \text{(A2)}$$

where $\overline{P}$ = mean value of the trait for the population in the parental generation , $\overline{P}'$ = mean value of the trait in the offspring generation, $\overline{P}_S$ = mean value of the trait among the *selected* or *surviving* parents that reproduce where each parent is weighted by the number of adult progeny produced, and h^2 = the heritability of the trait. The term "($\overline{P}_S - \overline{P}$)" is called the "selection differential" (SD) of the trait, and the response to selection ($\overline{P}' - \overline{P}$) - which is defined as the change in mean phenotypic value of the trait between offspring and parents – essentially equals the proportion of the parental SD that is transmitted to the progeny generation as determined by the heritability of the trait. These equations, in more complicated forms, have been the foundation for predicting responses to selection in the agriculture and livestock industries for decades.

The selection component of Ford's (2002) model includes a fitness function that measures the relative fitness[19] (f) of an individual in a particular environment as a

[19] *Fitness* is a commonly used term that is rarely defined precisely. *Individual fitness* can generally be subdivided into two components: *viability fitness* and *reproductive fitness*. Viability fitness measures the probability of individual survival from zygote formation to sexual maturity. Reproductive fitness of an individual measures the number of adult progeny resulting from reproduction. Parents and offspring share 50% of their genes in common (i.e., phenotypes of parents and offspring are highly correlated genetically) and, hence, fitness is correlated

function of (a) an individual's specific phenotypic value (P), (b) the parametric *optimum phenotypic value* (θ) that maximizes fitness of individuals within a particular environment, and (c) the strength or intensity of *stabilizing selection* that results in increasingly reduced fitness of individuals with phenotypic values that deviate increasingly from the phenotypic optimum in the specific environment under consideration. This relative fitness (f_i) of the ith individual with phenotype P_i within a population follows a *quasi-normal distribution* (eq. 2 of Ford 2002):

$$f_i = e^{-\frac{1}{2}\frac{(P_i-\theta)^2}{\omega^2}} \qquad \text{(A3)}$$

where "$(P_i - \theta)$" is the deviation of the ith individual's phenotypic value (P_i) from the *optimum phenotypic value* (θ) in the environment under consideration, and ω^2 is the variance of the probability density function that defines relative fitness as a function of phenotypic values (Fig. 2).

The relative mean fitness of the population is given by the following (eq. 3 of Ford 2002):

$$\overline{F} = e^{-\frac{1}{2}\cdot\frac{(\overline{P}-\theta)^2}{(\omega^2+\sigma^2)}} \qquad \text{(A4)}$$

This mode of selection is called "stabilizing" because it *drives* the mean phenotypic value ($\overline{P}$) of a population each generation towards the optimum phenotypic value (θ) for individuals in the specific environment inhabited by that population. Under this model, θ can have different values in different environments. A population would be considered "locally-adapted" when $\overline{P} = \theta$. The model assumes that θ for a particular environment is constant over multiple generations. However, in practice, the optimum for many traits (e.g., age at sexual maturity) most likely varies stochastically among generations due to varying environmental conditions (e.g., decadal oscillations in marine ocean conditions).

The *intensity* of selection is inversely proportional to the variance of the fitness distribution of phenotypes (i.e., selection intensity $\sim 1/\omega^2$; eq. 3). That is, as ω^2 increases, the selection intensity towards the phenotypic optimum decreases (Fig. 2). In other words, the relative fitness of an individual with a particular phenotypic value (P) in a particular environment will increase as ω^2 increases (when $P \neq \theta$) because the intensity of selection decreases (Fig. 1). According to Ford (2002), $\omega^2 = 10\sigma^2$ ($\omega \approx 3\sigma$, or less) is considered "strong selection", whereas $\omega^2 = 100\sigma^2$ ($\omega \approx 10\sigma$, or greater) would be considered "weak selection" (Lande 1976).

If the mean phenotypic value ($\overline{P}$) for individuals in a population does not equal the phenotypic optimum for that population (i.e., $\overline{P} \neq \theta$), then a population response to stabilizing selection is expected each generation for traits with $h^2 > 0$ until $\overline{P} = \theta$. This predicted response to stabilizing selection (R) follows the following relationship (eq. 4 from Ford 2002):

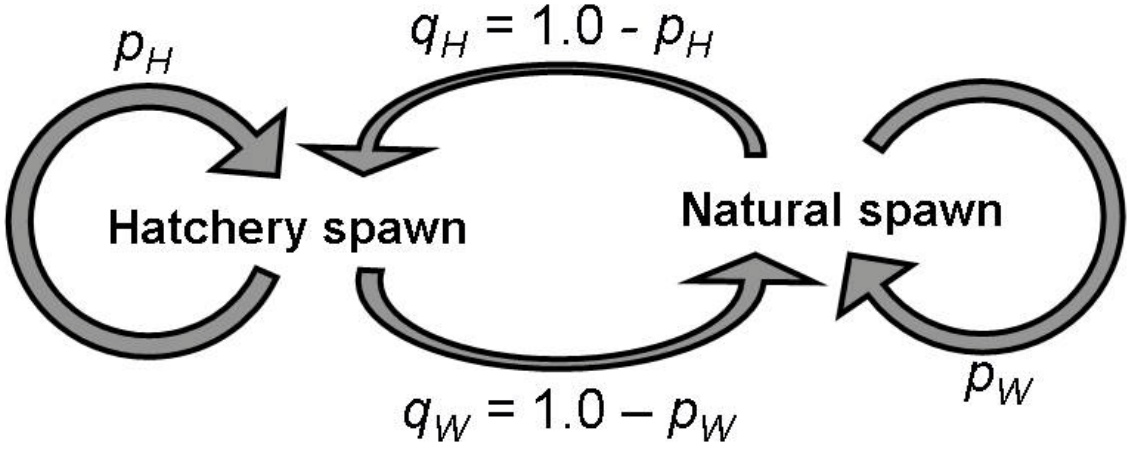
genetically between parents and offspring. For example, increased survival of progeny to sexual maturity (viability fitness) increases the fitness of their parents (reproductive fitness).

$$R = \overline{P}' - \overline{P} = h^2\left[\frac{\overline{P}\omega^2 + \theta\sigma^2}{\omega^2 + \sigma^2} - \overline{P}\right] \quad \text{(A5)}$$

where the quantity in brackets is the selection differential, h^2 is the heritability of the trait, and $\overline{P}'$ is the mean phenotypic value for the population after one generation of selection. The reader should note that the left-hand quantity within brackets is the predicted mean phenotypic value of breeding parents after selection/survival to adulthood (compare eq. A5 to eq. A2). Equation (A5) can be rearranged as a recursive equation which predicts the mean phenotypic value of a population in the offspring generation ($\overline{P}'$) as a function of the mean phenotypic value of the population in the parental generation ($\overline{P}$):

$$\overline{P}' = \overline{P} + h^2\left[\frac{\overline{P}\omega^2 + \theta\sigma^2}{\omega^2 + \sigma^2} - \overline{P}\right] \quad \text{(A6)}$$

These simple relationships are the basis for the two population, selection and gene flow model described by Ford (2002).



p_H = proportional genetic contribution of hatchery-origin adults to hatchery-origin offspring each generation.

p_W = proportional genetic contribution of natural-origin (wild) adults to natural-origin offspring each generation.

Figure 1. Schematic representation of 2-way gene flow between hatchery and wild populations. Each generation, hatchery-origin progeny are composed of a proportion p_H genes from hatchery-origin parents and a proportion 1.0-p_H (= q_H) genes from natural-origin parents. Similarly, natural-origin progeny are composed of a proportion p_W genes from natural-origin parents and a proportion 1.0-p_W (= q_W) genes from hatchery-origin parents. Those proportions are assumed to be constant over time.

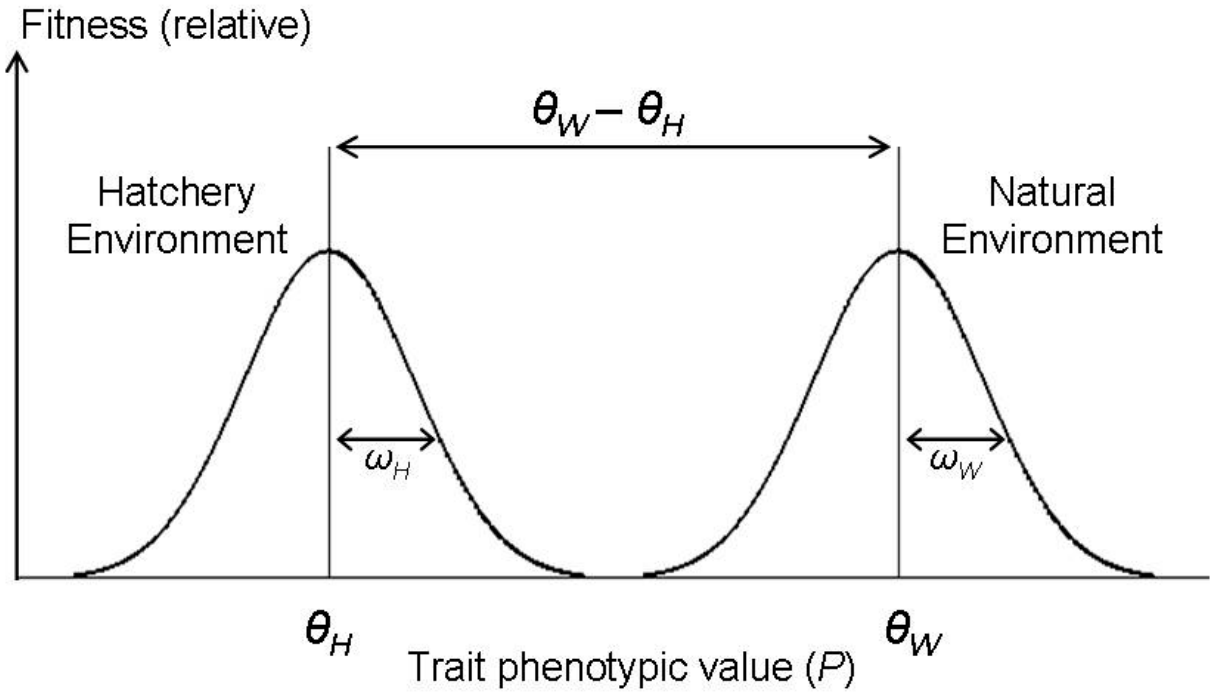


Figure 2. Schematic representation of fitness as a function of phenotypic values for two populations inhabiting different environments where the phenotypic optima (θ_W and θ_H) in the two environments are not equal. Each population is assumed to be under stabilizing selection in each environment. In the absence of gene flow between the two populations, selection in each environment maintains mean phenotypic values for each population equal to the phenotypic optimum for the respective environment. Each distribution is assumed to be distributed as a quasi-normal distribution with mean θ and variance ω^2 where the subscripts "*H*" and "*W*" refer to hatchery and "wild" environments, respectively. The magnitude of the difference $\theta_W - \theta_H$ is a measure of the strength of selective differences between the two environments. For hatchery and wild populations of fish, this difference reflects the strength of domestication selection in the hatchery environment relative to natural selection regimes in the wild environment. The difference in $\theta_W - \theta_H$ depicted here is greater than the actual difference observed for most traits associated with anadromous salmonid fishes. For most traits, the phenotypic distributions for hatchery and wild fish overlap such that a range of phenotypic values have relative fitnesses greater than zero in both environments. (after Ford 2002).

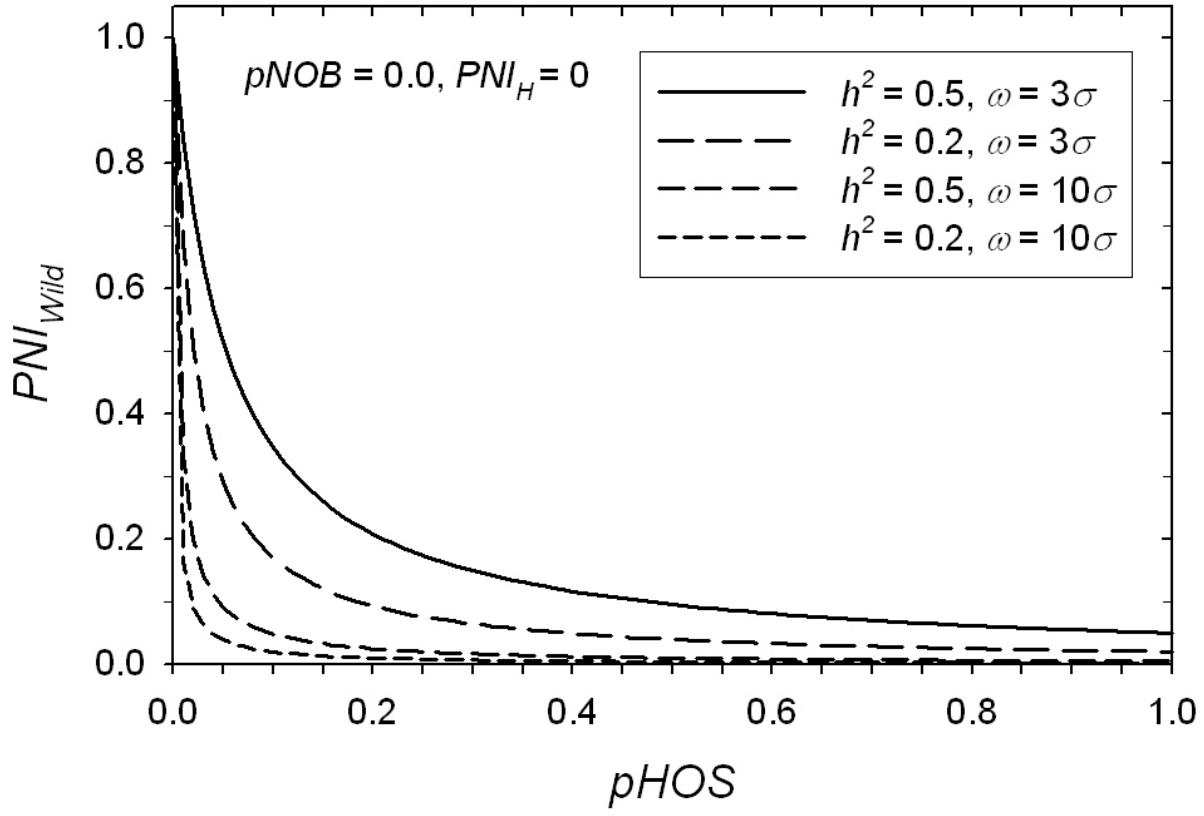


Figure 3. *Proportionate Natural Influence* for wild fish (PNI_{Wild} *or* PNI_W) as a function of the relative genetic contribution of hatchery-origin adults to natural-origin progeny each generation (eq. 9). The proportion of naturally-spawning fish composed of hatchery-origin adults (*pHOS*) is generally used as a management "surrogate" in lieu of empirical estimates of the mean proportional genetic contribution of hatchery-origin fish to a wild population each generation. In this figure, no wild fish are included in the broodstock (*pNOB* =0), thus resulting in $PNI_H = 0$ for hatchery fish (eq. 10). Heritabilities equal to $h^2 = 0.2$ and $h^2 = 0.5$ are considered *moderate* and *high* heritabilities, respectively. Selection intensities equal to $\omega = 3\sigma$ ($\omega^2 = 9\sigma^2$) and $\omega = 10\sigma$ ($\omega^2 = 100\sigma^2$) are considered *strong* and *weak* selection, respectively, where ω^2 = the variance of the distribution function for stabilizing selection about a phenotypic optimum (Fig. 2). Traits are assumed to be normally distributed with optimum values of θ_W =1.0 and θ_H = 0.0 in the wild and hatchery environments, respectively, with standardized phenotypic variances of $\sigma^2 = 1.0$ for both hatchery and wild fish. Heritabilities (h^2) and selection intensities (ω^2) are assumed to be equal in the two environments.

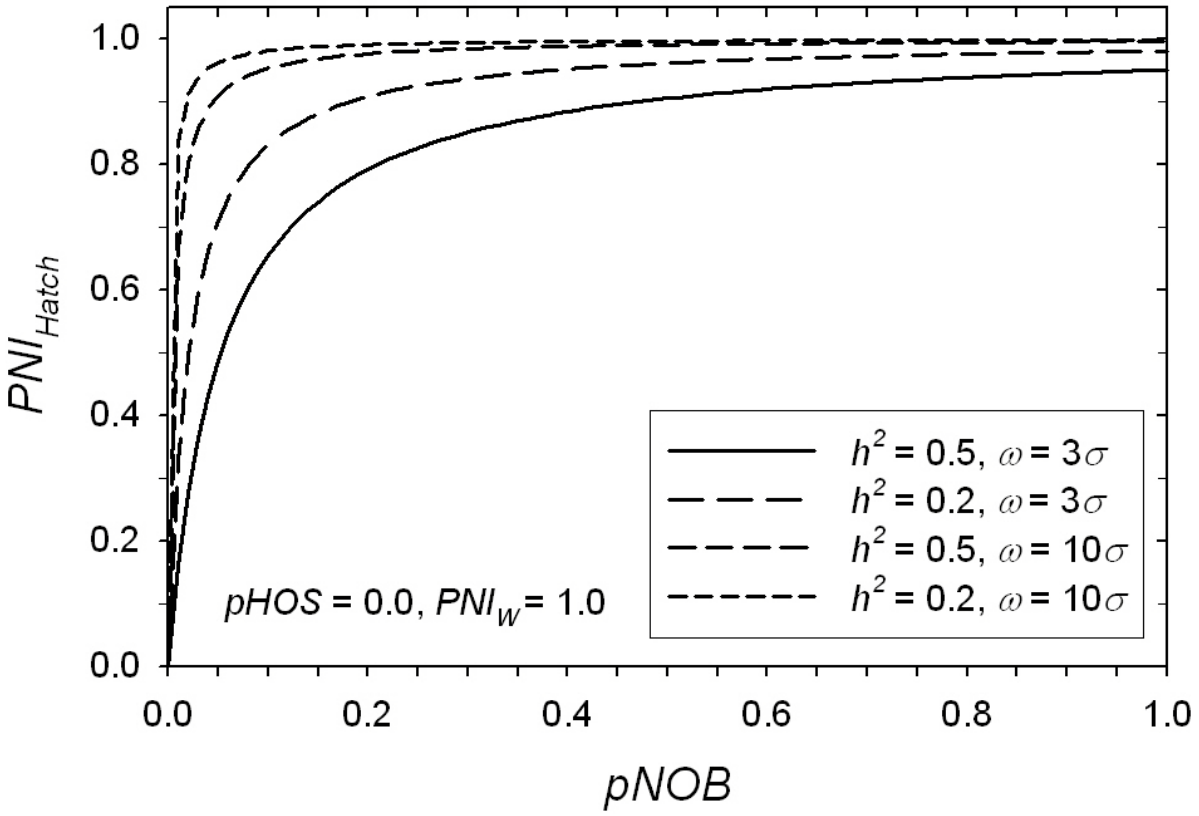


Figure 4. *Proportionate Natural Influence* for hatchery fish (PNI_{Hatch} *or* PNI_H) as a function of the relative genetic contribution of natural-origin adults to hatchery-produced progeny each generation (eq. 10). The proportion of a hatchery broodstock composed of natural-origin adults (*pNOB*) is generally used as a management "surrogate" for the mean proportional genetic contribution of natural-origin fish to hatchery-produced progeny each generation. In this figure, no hatchery fish are allowed to spawn naturally (*pHOS* = 0), thus resulting in $PNI_W = 1.0$ for wild fish (eq. 9).

When pHOS = 0, relatively small amounts of gene flow from the natural environment to the hatchery environment can increase PNI_H substantially. Indeed, when only 20% of a broodstock is composed of wild fish each generation (*pNOB* = 0.2), PNI_H will be greater than 0.75 even under conditions of high heritability and strong selection intensity in the hatchery environment if *pHOS* = 0.

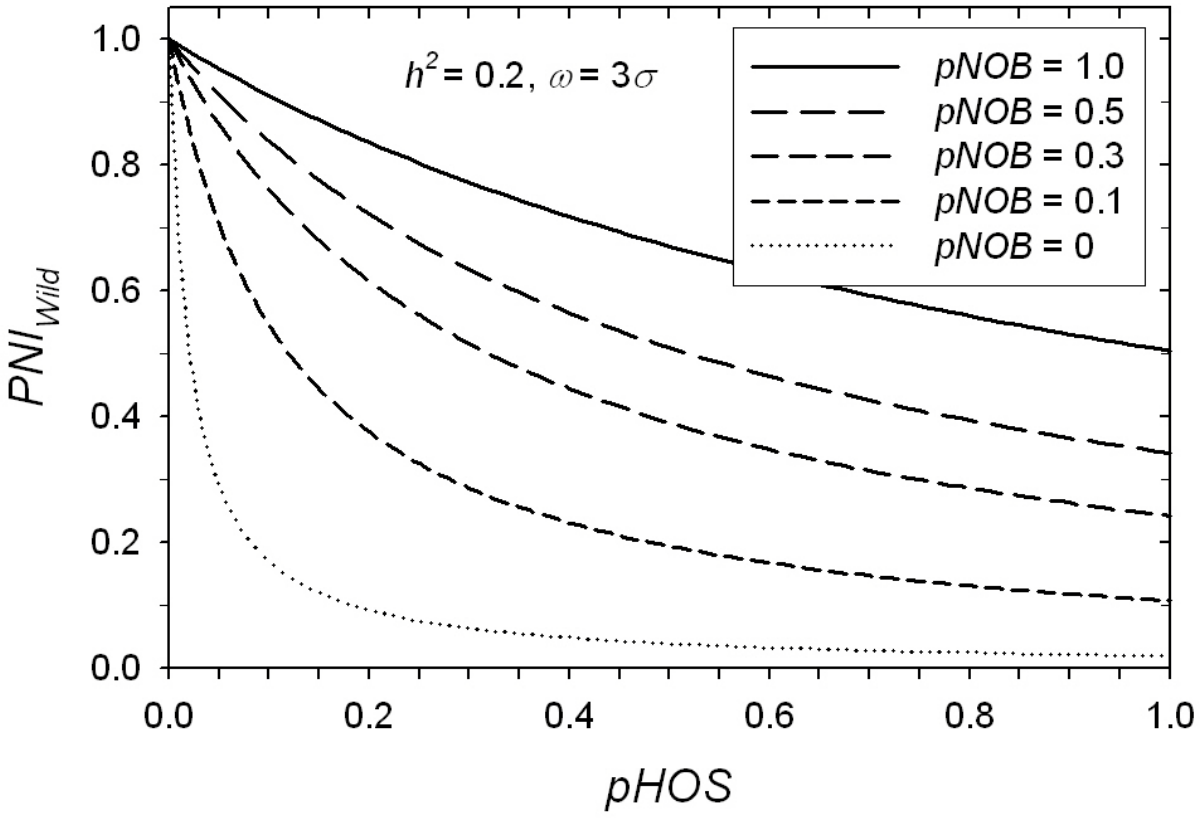


Figure 5. *Proportionate Natural Influence* for wild fish (PNI_W) as a function of the proportion of naturally spawning fish composed of hatchery-origin adults (*pHOS*) for different values of *pNOB*, the mean proportion of the hatchery broodstock composed of natural-origin fish each generation (eq. 9). Heritability and selection intensity in these plots are considered moderate ($h^2 = 0.2$) and strong ($\omega = 3\sigma$), respectively. The variables *pNOB* and *pHOS* are surrogates for the proportional genetic contribution, each generation, of wild fish and hatchery fish to a hatchery broodstock and a naturally spawning population, respectively (see eqs. 5 and 6). Of particular interest here is the long-term genetic effect on PNIW of including wild fish in a hatchery broodstock when pHOS is greater than 0.05.

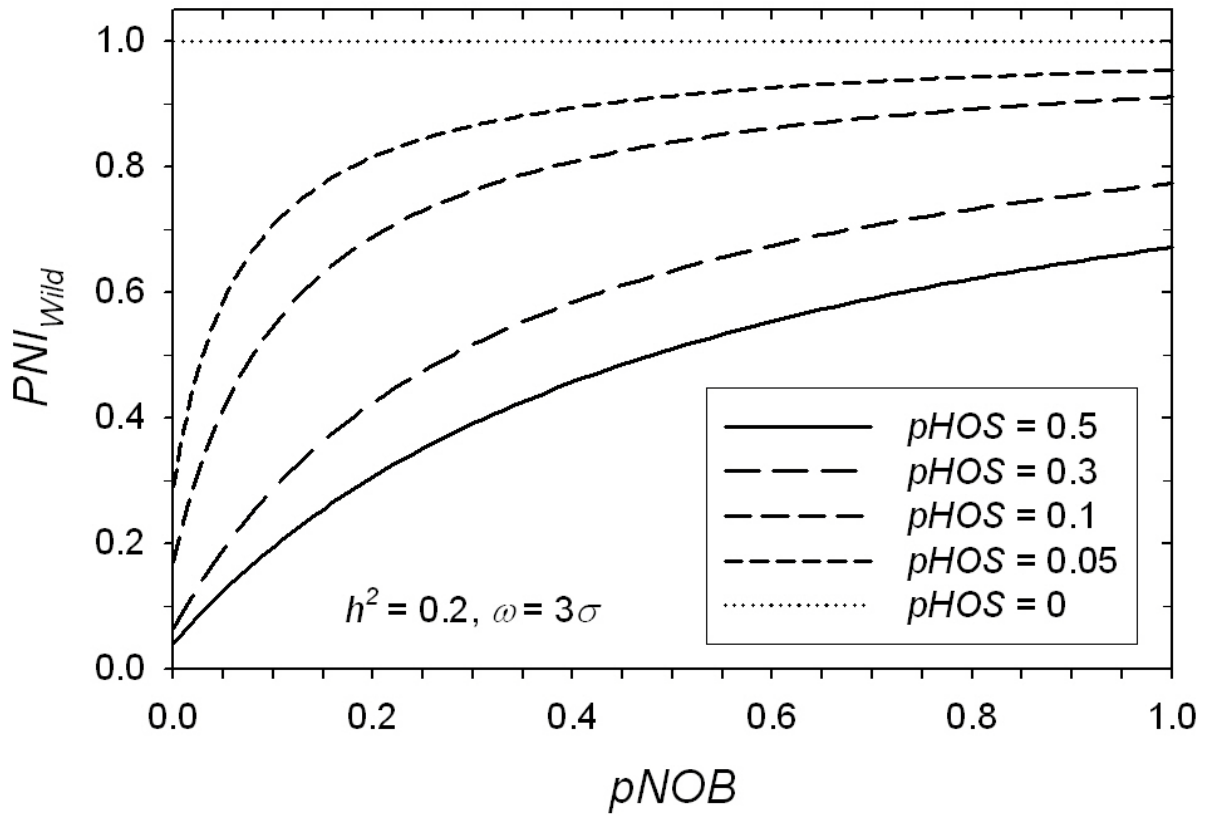


Figure 6. *Proportionate Natural Influence* for wild fish (PNI_W) as a function of the proportion of a hatchery broodstock composed of natural-origin adults (*pNOB*) for different values of *pHOS* (eq. 9). Of particular interest here is the large effect of small amounts of gene flow each generation from the hatchery environment to the natural environment (e.g., *pHOS* = 0.05) when *pNOB* = 0. Increasing *pNOB* = *pHOS* results in $PNI_W \approx$ **0.5 over all values of** *pHOS*. This graph is identical to Fig. 5 (eq. 9) except that PNI_W is plotted as function of *pNOB* instead of *pHOS*.

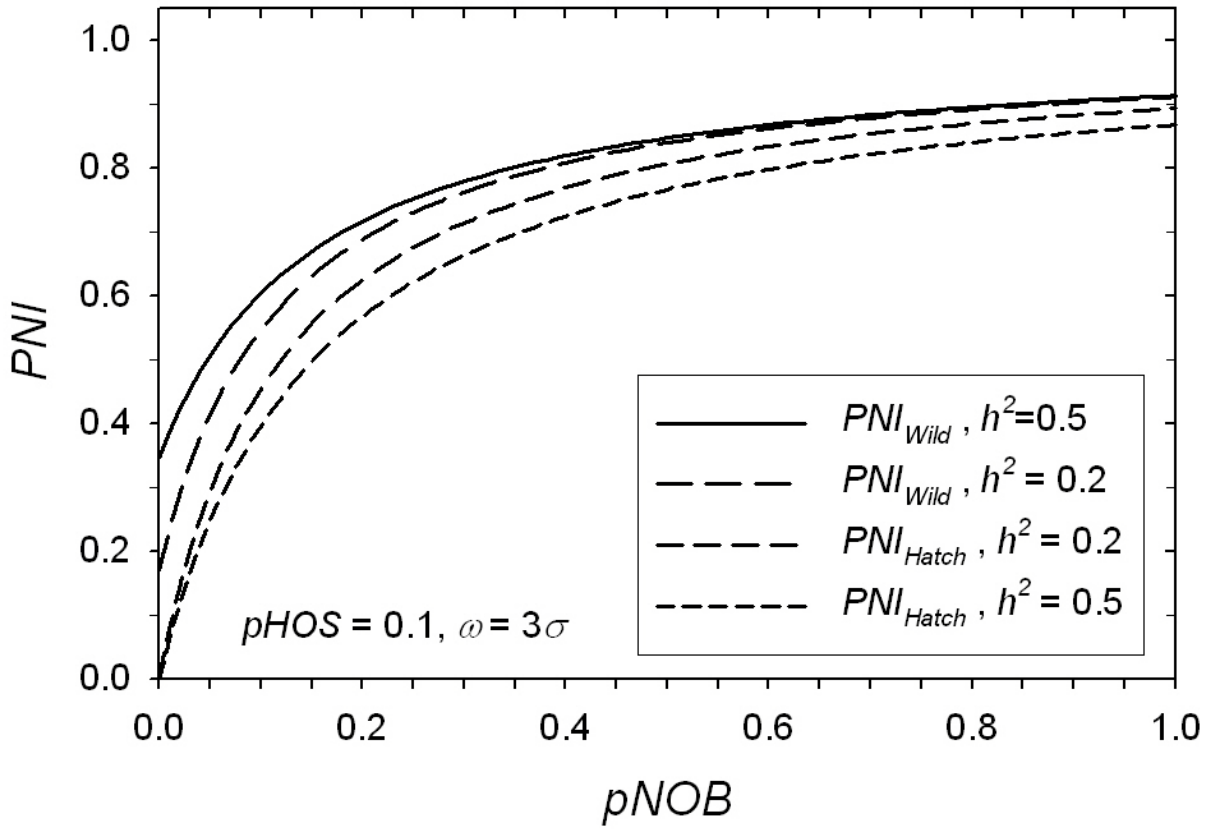


Figure 7. Comparison of *PNI* values for hatchery and wild fish as a function of *pNOB* (eq. 9) when *pHOS* = 0.1, selection intensity is considered strong ($\omega = 3\sigma$), and trait heritabilities are moderate and or high (h^2 = 0.2 and 0.5, respectively). For a given set of parameters, PNI_W will always be greater than PNI_H because wild fish, compared to hatchery fish, represent one extra generation of natural reproduction and selection in the wild environment. Nevertheless, the genetic composition for hatchery and wild fish will be nearly identical when an equilibrium between gene flow and selection is reached (eqs. 5 and 6). The difference between PNI_W and PNI_H increases with increasing heritability, reflecting the increased efficiency of selection and single-generation responses to selection as a function of increasing heritability (eqs. A2 and A4). Conversely, the difference between PNI_W and PNI_H decreases with increasing values of *pNOB*.

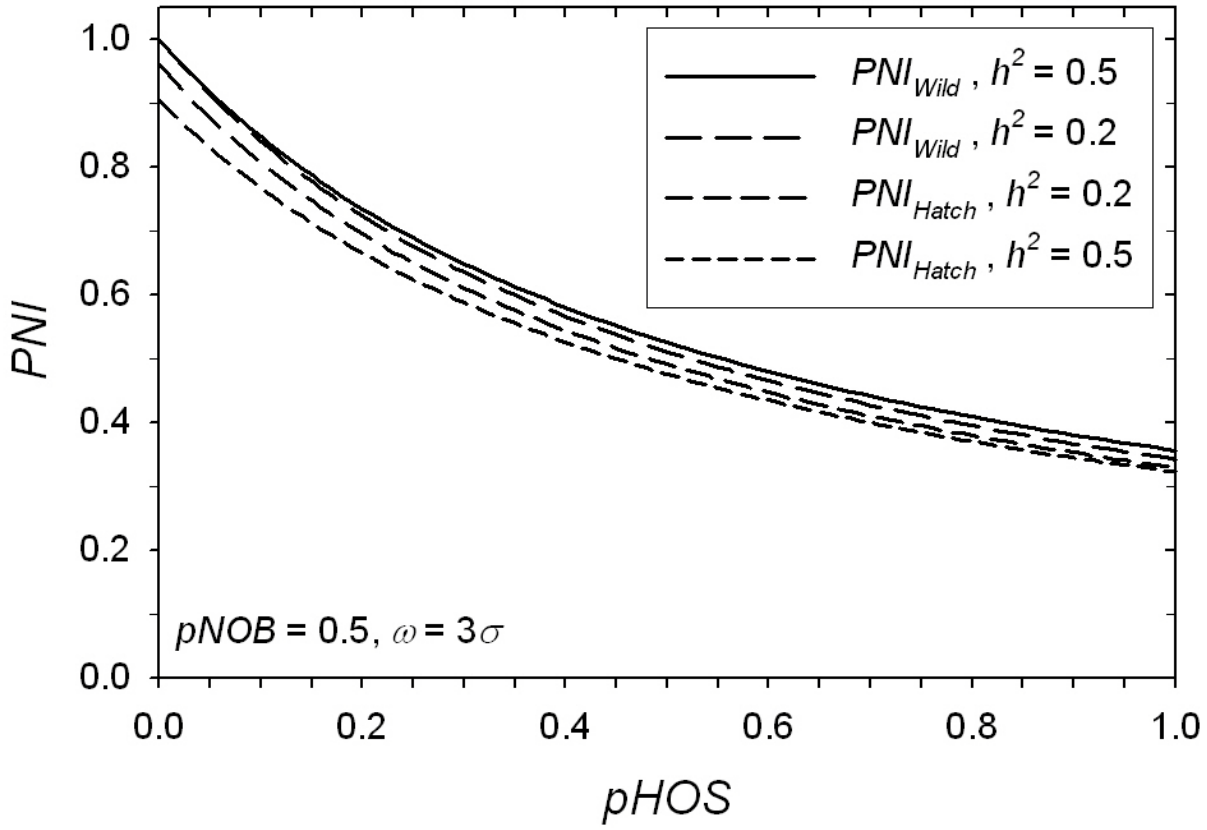


Figure 8. Comparison of *PNI* values for hatchery and wild fish as a function of *pHOS* when 50% of a hatchery broodstock is composed of wild fish each generation (*pNOB* = 0.5) and heritabilities are moderate or high (h^2 = 0.2 or 0.5, respectively). As in Fig. 7, PNI_W will always be greater than PNI_H for a given set of parameter values, although the difference between PNI_W and PNI_H will decrease with increasing values of *pNOB*.

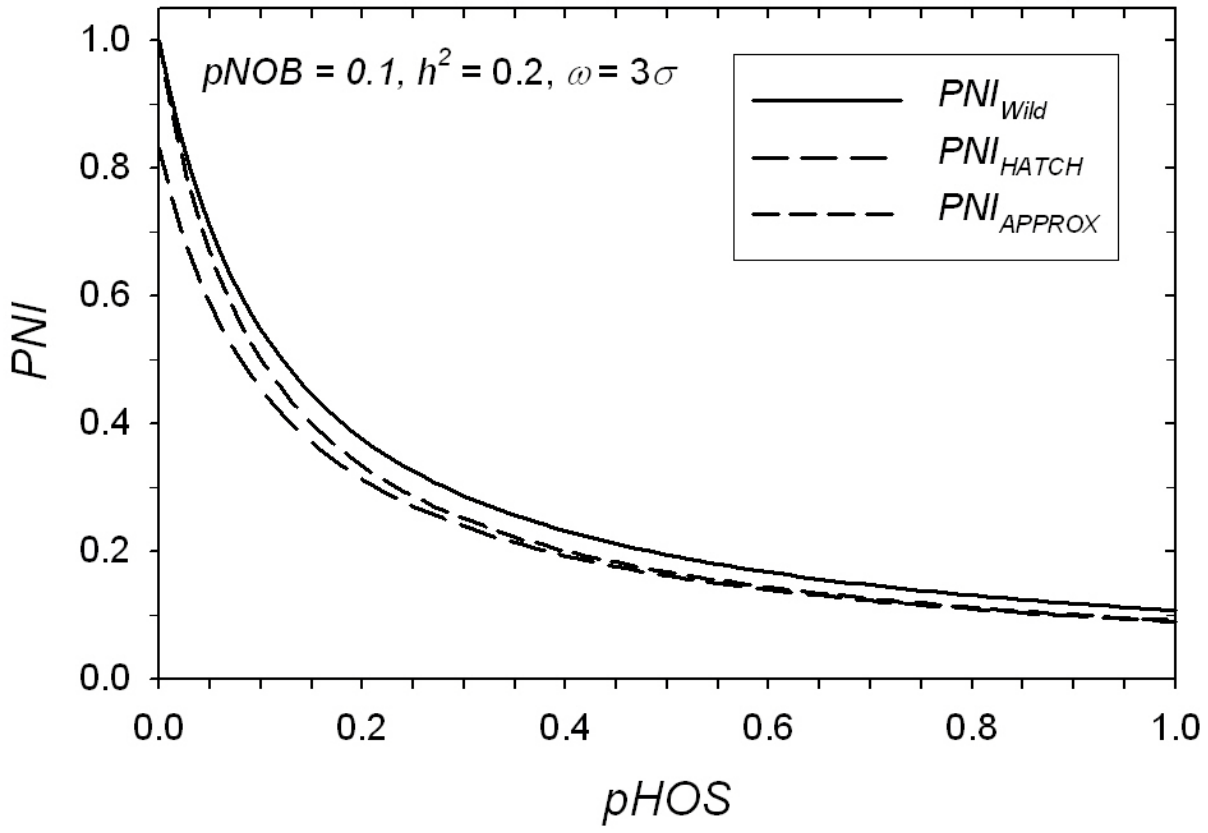


Figure 9. Comparison of the *PNI* index approximation (PNI_{Approx}; eq. 11) to PNI_W (eq. 9) and PNI_H (eq. 10) as a function of *pHOS* when *pNOB* = 0.1 for a trait under strong selection ($\omega = 3\sigma$) with moderate heritability ($h^2 = 0.2$). When *pNOB* is greater than zero, the approximation is very close to the derived value of PNI_W (eq. 9). However, when *pNOB* = 0, which is true for a large number of hatchery broodstocks where only hatchery-origin fish are spawned, then eq. (9) should be used to estimate PNI_W for natural-origin fish. In this latter situation, a range of possible PNI_W values can be generated via eq. (9) assuming heritabilities and selection intensities for traits that are likely to be of greatest concern: that is, traits that can respond quickly to selection over a small number of generations because they are under moderate to high selection intensities ($\omega = 6\sigma$ to $\omega = 3\sigma$)[20] and/or because they have moderate to high heritabilities (h^2=0.2 to h^2 = 0.5, respectively).

[20] Equation (9) assumes that the phenotypic variance of the trait has been standardized to $\sigma^2 = 1.0$.

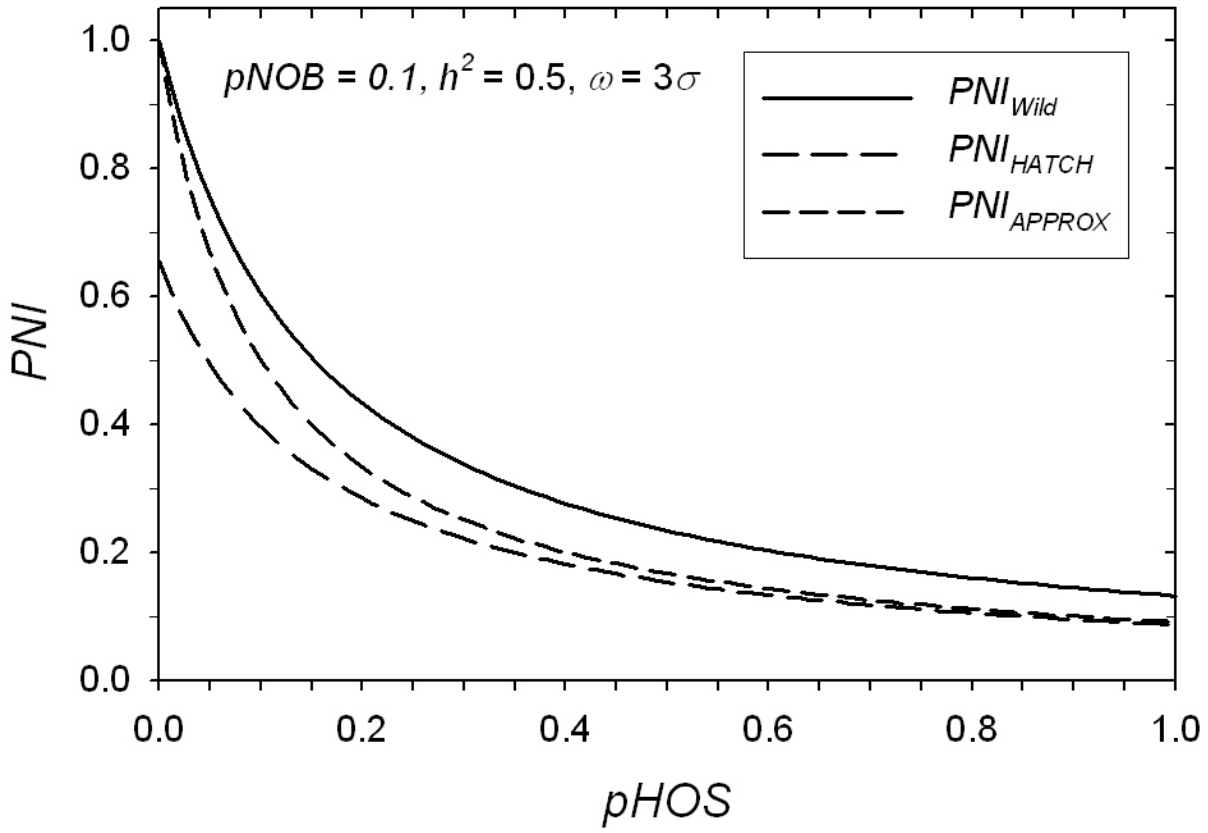


Figure 10. Comparison of the *PNI* index approximation (PNI_{Approx}; eq. 11) to PNI_W (eq. 9) and PNI_H (eq. 10) as a function of *pHOS* when *pNOB* = 0.1 for a trait under strong selection ($\boldsymbol{\omega} = 3\boldsymbol{\sigma}$) with high heritability (h^2 = 0.5; compare graph above to Fig. 9 where h^2 = 0.2). For a trait with high heritability, an extra generation of selection in the respective environments can result a comparatively large difference in the values of PNIW and PNIH at low values of pHOS; however, PNI_{Approx} more closely tracks PNI_W which is the index of greater concern from a natural population perspective. As noted in the caption of Fig. 9, equation (9) should be used to calculate PNI_W, not equation (11), whenever *pNOB* equals zero.

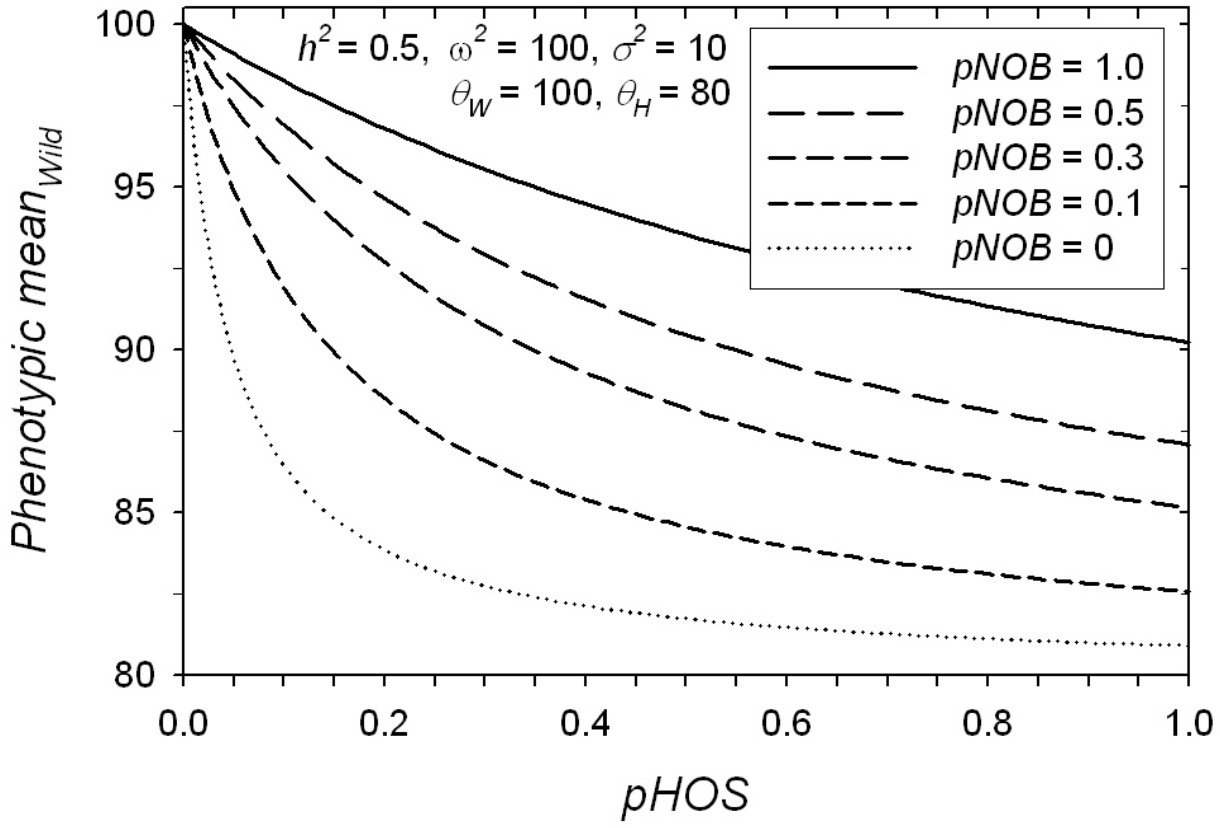


Figure 11. Phenotypic mean of wild fish at equilibrium after many generations of gene flow between hatchery and wild populations as a function of *pHOS*, the mean proportion of a naturally spawning population composed of hatchery-origin fish each generation (eq. 3). The hypothesized trait is assumed to have a heritability (h^2) and phenotypic variance (σ^2) equal to 0.5 and 10, respectively, in both environments. The variance of the fitness function (ω^2) is assumed to be equal to $10 \cdot \sigma^2$ in both environments, which is considered "strong" selection. The trait is further assumed to have phenotypic optima of θ_H = 80 and θ_W = 100 in the hatchery and natural environments, respectively. The values of h^2, ω^2, σ^2, θ_H and θ_W presented here are the same values used by the HSRG in the *AII-H Analyzer* (*AHA*) model to simulate the population dynamics of hatchery and wild fish in the Columbia River Basin. The reader should note that the shapes of the graphs presented here are nearly identical to those presented in Figure 5; slight differences in the shape of the two sets of curves are due primarily to the high heritability (h^2 = 0.5) used here (and in *AHA*) versus the moderate heritability (h^2 = 0.2) used to generate Figure 5. As noted in the text, the shapes of the equilibrium curves for the phenotypic means of wild and hatchery fish ($\hat{P}_W$ and $\hat{P}_H$, eqs. 3 and 4, respectively) are largely independent of specific values of θ_H, θ_W, and σ^2; only the scale of the vertical axis changes as a function different values for the phenotypic optima in each environment. These latter results further warrant the use of equations (9), (10), and (11) to assess the genetic risks of hatchery programs to naturally spawning populations.

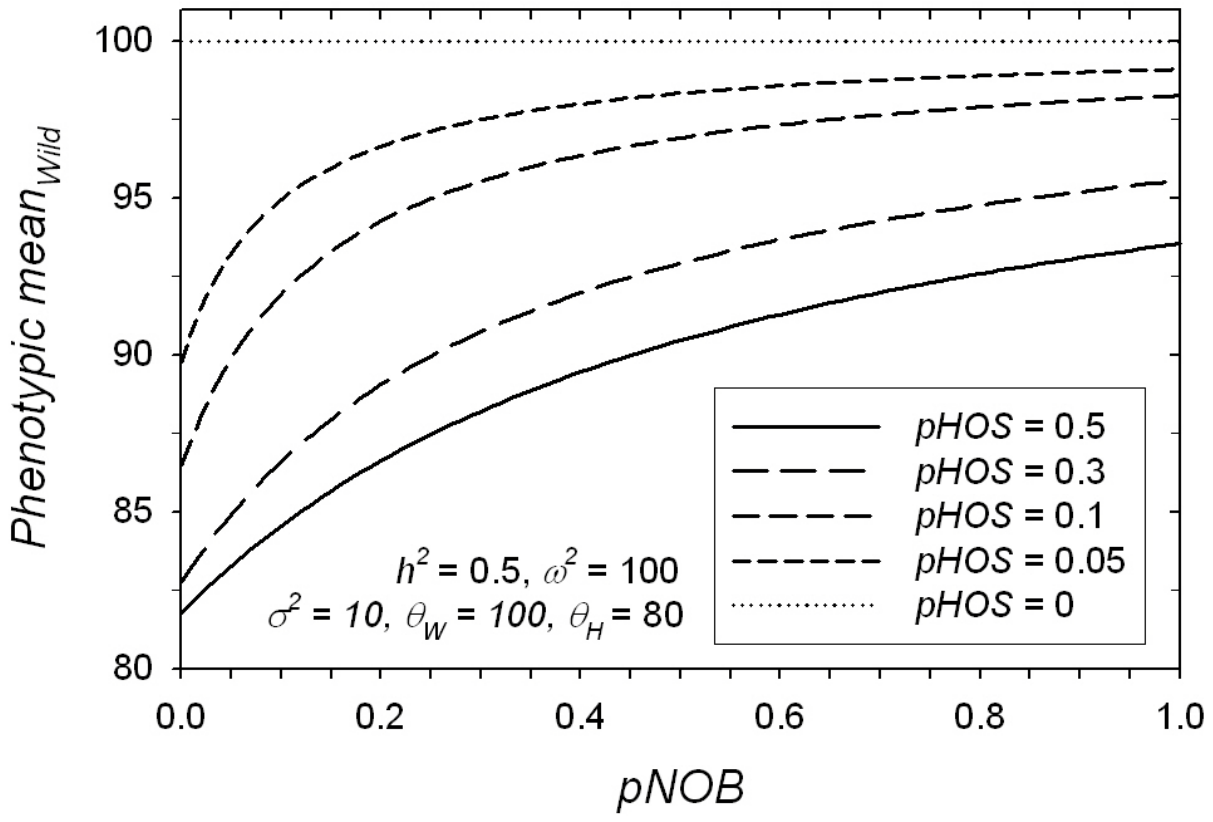


Figure 12. Phenotypic mean of wild fish at equilibrium after many generations of gene flow between hatchery and wild populations as a function of *pNOB*, the mean proportion of a hatchery broodstock composed of natural-origin fish each generation (eq. 3). Parameter values presented here are the same as those described in Figure 11. The reader should note the close similarity between this figure and Figure 6. As noted in Figure 11, the shapes of the curves are largely independent of the specific values of θ_H, θ_W, and σ^2. Variation in the values of θ_H and θ_W only affects the scale of the relationship (vertical axis) without affecting the relative phenotypic values of hatchery and wild fish relative to their optima within each environment.