QUANTITATIVE GENETIC CONSEQUENCES OF CAPTIVE BROODSTOCK PROGRAMS FOR ANADROMOUS PACIFIC SALMON (*ONCORHYNCHUS* SPP.)[1]

by

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Introduction

Quantitative genetics is one of the oldest fields in genetics, its origins predating even the discovery of Mendel's work at the turn of the century (Provine 1971). The quest to reconcile inheritance of quantitative characters (i.e., those with phenotypes that do not clearly fall into discrete classes, but instead are more or less continuously distributed) with Mendelian genetics (Yule 1902, Fisher 1918) had a profound influence on the early development of both genetics and biometrics.

The debate that arose after the turn of the century between the Mendelian and Biometrician schools of genetics over the genetic basis of phenotypic variation produced a number of important analytical tools, such as correlation and regression (Galton 1889, Pearson 1920) and the analysis of variance (Fisher 1918), as well as genetic techniques such as the estimation of the "effective" number of genes contributing to quantitative characters (Wright, in Castle 1921) and the characterization of mutational effects (Haldane 1927).

Curiously, however, the influence of quantitative genetics on evolutionary biology has been sporadic over most of the last 60 years, a situation that was not helped by the breakthroughs of modern molecular genetics in the 1960s. Quantitative genetics evolved during much of this century in the realm of animal and plant breeders primarily, and during this time the field progressed somewhat independently of other areas in biology, including evolutionary genetics (Lande 1988a). As a result, many developments in quantitative genetics resulted from applied research aimed at measuring and predicting responses to selection in domesticated plants and animals (Lush 1945).

In the last 15-20 years, interest in quantitative genetics as a tool in evolutionary biology has been revived. This interest has grown out of theoretical efforts to understand the complex evolutionary behavior of quantitative characters under mutation, migration, genetic drift, and selection (e.g., Wright 1978; Lande 1976, 1980, 1982a; Turelli 1984; Lynch and Hill 1986; Clark 1987; Charlesworth 1990). Efforts to explain differences between short-term and long-term responses to selection have also been a major focus of investigation in quantitative genetics (Falconer 1989).

In the face of important developments in molecular genetics in the last 30 years, and especially during the last decade, quantitative genetics has remained the mainstay of analysis for quantitative traits. Many of these traits bear on issues in fish biology and conservation, such as adaptation of natural populations to environmental variability, selection in cultured populations, and the genetic and phenotypic divergence of natural and cultured populations sharing recent ancestry.

Quantitative genetics in fishery biology has been reviewed by Kirpichnikov (1981), Gjedrem (1983), Kinghorn (1983), and Tave (1993). These reviews emphasized the role of quantitative genetics in selective breeding programs for economically important fish species.

Although many of these programs have involved salmonids, the focus of these reviews has been primarily on the use of quantitative genetic methods to increase aquacultural production or contribution to fisheries, both traditional purposes of artificial propagation.

The purpose of this review is to summarize current knowledge in the area of quantitative genetics related to the genetic consequences of captive culture programs for Pacific salmon (*Oncorhynchus* spp.), especially captive broodstock programs, and to identify issues in this area in need of further research.

Although the focus of this review is on the quantitative genetic management of Pacific salmon populations, most of the empirical work in quantitative genetics has involved plants and animals with short generation times or of widespread economic importance, primarily in agriculture. Consequently, the literature reviewed here encompasses a wide range of organisms. Nevertheless, these studies--as well as the relatively few studies involving salmonids that do exist--provide a foundation for developing future quantitative genetic research on Pacific salmon.

For this report, we outline prominent quantitative genetic risks associated with captive broodstock programs, discuss the importance of genetic monitoring for quantitative characters, and introduce some basic approaches to address these issues. The quantitative genetic consequences of these programs can be grouped into three main categories: 1) loss of genetic variability within a population resulting from the establishment of a captive broodstock, and the inbreeding depression that may result; 2) genetic change that may result from natural selection (domestication) on captive fish in protective culture; and 3) genetic divergence of the captive fish from their natural source population, and the consequences of genetic interactions between these groups.

Quantitative Genetic Approaches to Genetic Inference

The approach to genetic analysis of variation in quantitative characters is affected by three mmon aspects of these characters: 1) their phenotypic expression is typically sensitive to vironmental variation (Falconer 1989), 2) in general, they do not fall into discrete phenotypic asses but instead exhibit continuous phenotypic distributions (for "threshold" characters, such as sease resistance or migratory tendency, they are thought to have an underlying distribution of enetic effects that is approximately continuous; Falconer 1965, 1989), and 3) they usually appear) be under the control of several genes of generally unknown effect on the character (Wright 968, Lande 1981).

Quantitative genetic investigations use statistical analyses of these characters to describe he composite behavior of the underlying genes. These analyses generally require an assumption)f Mendelian inheritance at constituent loci and small, independent effects of many genes on the characters of interest (Bulmer 1985). With this assumption, these analyses use the phenotypic resemblance of individuals of known average relationship to permit inferences about the inheritance and evolution of quantitative characters, processes that are controlled by hidden variation in gene frequencies and effects (Falconer 1989). Through often elaborate statistical analyses, observed patterns of means, variances, and covariances in a population, when combined with appropriate breeding designs and statistical techniques, can be used to estimate the genetic parameters that determine the population's response to selection.

Unlike most molecular genetic analyses, which focus on traits controlled by a single gene or a few major genes, quantitative genetic analyses are generally incapable of detecting the effects of individual genes on the expression of quantitative traits. Despite this limitation, it is significant that genetic variation not detectable with molecular genetic analyses may be revealed with the appropriate quantitative genetic tool. Thus, molecular and quantitative genetic techniques detect different facets of the genome, a point worth remembering when considering different techniques for applications to problems in conservation biology.

The quantitative genetic approach is fundamentally one of partitioning observed variation into its genetic and environmental components. Because it does not focus on genes themselves, but rather on composite genetic and environmental effects on the phenotype, the power of this approach to resolve the details of genetic architecture is limited. Quantitative genetic approaches are therefore more synoptic and less mechanistic than molecular genetic approaches. Nevertheless, this quality of quantitative genetic analysis contributes to its suitability for investigating patterns of adaptive evolution and comparing them in populations that occur over broad environmental or geographic gradients. Such analyses are especially appropriate for investigating variation in life-history traits, which are known to be influenced strongly by both genetic and environmental variation.

The rapid development of molecular genetic techniques such as the analysis of restriction fragment length polymorphisms (RFLPs) might appear to offer an alternative to conventional

quantitative genetics for assessing genetic variation, but these methods can at best provide only part of the picture. For example, molecular analyses like these are not designed to detect the effects of environmental variation, or its interaction with genotypic variation, on the phenotype. Interpreting molecular genetic variation in terms of adaptive evolution is always problematic because the direct relationship between molecular variants and fitness differences is obscure (Eanes 1987, Houle 1989, Lewontin 1991, Avise 1994).

Quantitative genetic techniques also differ from molecular ones in that they are prospective rather than retrospective (Ewens 1979): quantitative genetic approaches generally focus on the potential evolutionary consequences of particular genetic states rather than on describing the genetic states that have resulted from past evolution. Thus, quantitative genetics can be useful for generating hypotheses for what the consequences of genetic change will be, at least in the short term.

Quantitative genetic approaches are not without potential problems. Although it is relatively straightforward simply to determine whether or not a character has a genetic basis (Lawrence 1984, Crow 1986, Falconer 1989), the estimation and interpretation of genetic parameters can be difficult and have some limitations. Most parameters that are estimated are specific to the population and environment in which they are measured. They are also sensitive to the influence of migration, mutation, selection, non-random mating, level of inbreeding, genotype by environment correlation or interaction, and ecological or social factors (Barker and Thomas 1987). In addition, quantitative genetic estimates are based on an assumption that the underlying genes are unlinked structural genes, not modifiers.

Despite these limitations, quantitative genetic methods are the tools of choice to analyze the mechanisms of adaptive evolution. These methods are designed to measure the inheritance of life-history characters, to estimate their responses to evolutionary forces (mutation, gene flow, genetic drift, and selection), and to identify the consequences of these responses for the adaptation and divergence of populations. There are several of these methods available to the experimental quantitative geneticist, and they can be organized into a few general categories defined by their respective objectives: 1) determine the inheritance of quantitative characters and the genetic basis of their phenotypic variation, 2) identify the mode of gene action affecting phenotypic expression within and among populations, 3) estimate the "effective" number of underlying genes, and 4) assess and predict the response to selection (and limits to this response). All of these objectives and their corresponding techniques have well-developed theoretical foundations and are based on analyzing patterns of observed variation within or among groups of individuals of known relatedness (Barton and Turelli 1989).

Thus, quantitative genetics has applications to a wide variety of genetic problems. The primary objectives of animal and plant breeders are to maximize a population's response to selection in particular environments and to estimate "breeding values" (Falconer 1989) to predict and enhance this response. Evolutionary quantitative geneticists analyze phenotypic variation within and among natural populations, both to understand its genetic basis and describe the

evolutionary mechanisms that produced its observed variation. Quantitative genetic methods have not yet been widely applied to the supplementation and conservation of natural populations, but much of what is known from evolutionary genetics and from applied breeding may be useful in increasing the prospects for success in these efforts.

The Significance of Quantitative Genetic Variation

Evolutionary geneticists recognize four primary agents that affect gene frequencies and, hence, genetic variation: mutation, genetic drift, gene flow, and selection (Futuyma 1986, Hartl and Clark 1989). Because the distribution and maintenance of genetic variation within and among natural populations is a fundamental problem in evolutionary genetics, the effects of these agents on patterns of genetic variation have received a great deal of theoretical and empirical attention (reviewed by Barton and Turelli 1989, Falconer 1989, Avise 1994). Traditional approaches to characterizing these patterns of variation have concentrated on single-locus polymorphism (Lewontin 1974, 1991) and have often neglected quantitative genetic variation. Quantitative genetic variation differs from single-locus polymorphism in that a *distribution* of genotypic values potentially exists at each locus; these values are usually assumed to be normally distributed (Kimura 1965, Lande 1976). Major changes in observed variation that affect adaptation are thought to result largely from polygenic variation rather than variation at single loci (Wright 1968, Lande 1981). For this reason, Lande and Barrowclough (1987) argued that quantitative characters and their inheritance should be considered distinct from single-locus characters (but see Orr and Coyne 1992 for a counterargument).

A common explanation for the observed maintenance of quantitative genetic variation in populations is a balance between the "forces" of mutation and selection. A variety of models have been advanced to describe the mechanism for this maintenance. Although these models take into account the incidence of mutation, strength of selection, and distribution of alleles at each locus, and assume polygenic inheritance (e.g., Lande 1976, Lynch 1984, Turelli 1984, Houle 1989), the true situation is probably more complicated. For example, other factors affecting genetic variation include phenotypic plasticity (Via and Lande 1985), frequency-dependent selection, spatial variation, and gene effects expressed through multiple characters (i.e., pleiotropy; Rose 1982). In addition, theoretical (Griffing 1960; Goodnight 1987, 1988; Lynch 1988; Gimelfarb 1989) and empirical (Wade and McCauley 1984, Bryant et al. 1986, Carson and Wisotzkey 1989, Cohan et al. 1989, Bryant and Meffert 1992, Hard et al. 1993) studies suggest that genetic variation may be redistributed into additive genetic variation under certain conditions (e.g., sharp population bottlenecks).

The dynamics of genetic variation are potentially much more complex for quantitative characters under selection because quantitative characters are affected by many more genes than single-locus characters, whose dynamics are thought to be affected primarily by genetic drift. The consequences of neglecting quantitative genetics in the management or conservation of natural populations are not clear, but Bentsen (1991) has claimed that one possibility is that monitoring

only the "qualitative" genetics of a population may cause undesirable genetic change. For example, if populations are differentiated only by allele frequencies and not by the distribution of unique alleles, as is often the case among anadromous salmonid populations, the use of a low number of breeders thought to represent a population's "unique" genetic architecture may lead to a reduction in quantitative genetic variability, potentially eroding local adaptation.

Bentsen (1991) believed that the erosion of local adaptation in several Atlantic salmon (*Salmo salar*) populations could have resulted from relying solely on electrophoretic surveys for monitoring genetic variability in these populations. Other evidence suggests that it may be easier to maintain quantitative genetic variation than single-locus variation (Lande and Barrowclough 1987, Bryant and Meffert 1992). Unfortunately, little explicit guidance is available for monitoring salmon populations to reduce genetic problems associated with interaction between hatchery and wild fish (Lande and Barrowclough 1987, Hindar et al. 1991; but see Hard, in press).

On the whole, fishery genetic researchers are not taking advantage of approaches being developed by evolutionary quantitative geneticists. Most fishery genetic work published to date has dealt with estimating levels of genetic variation within and among populations. While this objective is important, it ultimately prompts the question, What maintains or erodes this variation? Attempts to answer this question are not only of basic evolutionary interest, but impinge on issues essential to informed management and conservation.

The Components of Quantitative Variation

As explained above, quantitative genetics is used to address a variety of issues associated with inheritance and evolution. These include the distribution of genetic variation within and among populations and the consequences of mutation, gene flow, genetic drift, and selection for this distribution. In order to familiarize the reader with concepts and terms used in our review of empirical quantitative genetics, this section outlines a fundamental objective of quantitative genetics: characterizing the elements of phenotypic variation for quantitative traits. At its simplest level, this objective requires partitioning phenotypic variation into genetic and environmental components (Falconer 1989). For any quantitative trait, the relationship among these components can be expressed in terms of variances as follows:

$$V_P = V_G + V_E \quad (1)$$

where V_P is the trait's phenotypic variance, V_G is its genotypic variance, and V_E is its environmental variance. Thus, this relationship can be interpreted as the phenotypic variation (i.e., measured or observed variation) in a trait that results from variation in both genotypes and environmental factors.

Based on what is known about each of these components, they can be further subdivided to yield a more realistic equation. The genotypic variance can be dissected into three parts that reflect three general types of gene expression:

$$V_G = V_A + V_D + V_I \quad (2)$$

where V_A is the additive genetic variance, or variance due solely to the composite independent effects of genes exclusive of net directional dominance and other effects; V_D is the dominance genetic variance, or that due to dominance effects within loci; and V_I is the interaction (or epistatic) genetic variance, or that due to interactions among loci. This partitioning of genotypic variance reflects the influences that these different categories of allelic or genic variation may have on phenotypic expression.

Another term is usually added to Equation 1 to indicate the fact that genotypic and environmental effects on the phenotype are often not independent. This term, V_{GE}, reflects the genotype-environment interaction that can contribute to local adaptation, and renders a more realistic equation:

$$V_P = V_A + V_D + V_I + V_E + V_{GE} \quad (3)$$

This equation is widely used in quantitative genetics and is applicable as long as genotypic and environmental differences are uncorrelated (Falconer 1989).

Because most quantitative characters may be under the control of many genes with small phenotypic effects, it is typically impractical to identify either the location or the effect of individual genes. Quantitative genetic techniques attempt to characterize the composite behavior of a quantitative trait's underlying genes by analyzing two pieces of information: the statistical behavior of the observed trait, and the relationship between the individuals examined. The elegance of quantitative genetics is that the inheritance and evolution (in the very short term, at least) of such traits can be understood and, in principle, predicted with this information alone.

The basic metric used in quantitative genetics to depict the relative contribution of genetic and environmental variation to a trait's phenotype in a population is heritability. The origin of this term is unknown, but probably arose before Mendel (Bell 1977). It is currently used in one of two ways: broad-sense or narrow-sense. Broad-sense heritability (designated H^2) is used to estimate the ratio of all "genetic" sources of variance (V_G) to V_P. These sources include dominance, epistasis, and some specific environmental effects such as maternal and cytoplasmic effects (see Falconer 1989). Narrow-sense heritability (designated h^2) is used to estimate the ratio of V_A to V_P. Both are ratios with scales from 0 (no "genetic" variance) to 1 (no "environmental" variance) (Figure 1). Narrow-sense heritability is a more reliable predictor of a trait's short-term evolutionary response than broad-sense heritability and, consequently, is more widely estimated in breeding programs. However, the broad-sense heritability is more appropriate when dealing with asexually reproducing organisms, in which additive and non-additive sources of genetic variance are not separable, or in circumstances where it is not feasible to implement breeding designs to estimate h^2.

A variety of methods exist to estimate h^2 and its analog for determining the genetic relationship between two traits, the additive genetic correlation, r_A (Falconer 1989). Three basic techniques are commonly used: sib analysis, offspring-parent regression, and response to selection. The first two techniques use the relationship among relatives. When estimated from this relationship, the h^2 of a trait and the r_A between two traits are ratios of covariances and variances. For example, using a conventional half-sib analysis (see Falconer 1989), h^2 of trait x is estimated as four times the ratio of the phenotypic covariance (cov) among half sibs (HS) to the phenotypic variance (var):

$$h^2 = \frac{4\ cov_{HS}(x)}{var\,(x)} \qquad (4)$$

The coefficient of 4 indicates the fact that half sibs have an average coefficient of relationship of 1/4 (i.e., share 1/4 of their genes).

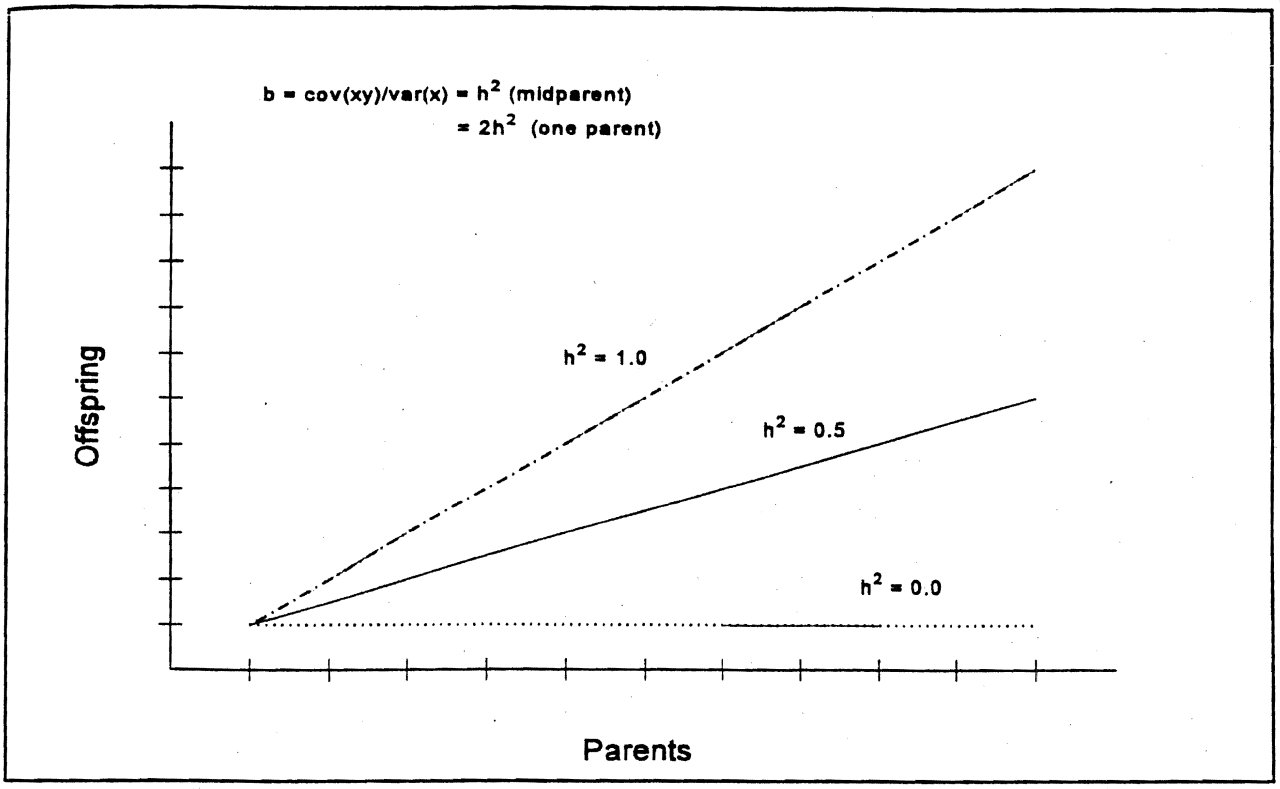

Figure 1. Graphical representation of estimating the heritability (h^2) of a quantitative trait by regressing offspring phenotypes on those of their parents. The relationship of the least-squares linear regression coefficient b to h^2 depends on which parents are used in the regression (Falconer 1989). The estimate of h^2 ranges from 0 (no genetic variance) to 1 (no environmental variance).

The r_A between traits x and y is the ratio of their covariance to the square root of the product of their variances:

$$r_A = \frac{cov_{HS}(xy)}{\sqrt{var_{HS}(x)\ var_{HS}(y)}} \qquad (5)$$

With offspring-parent regression, h^2 can be estimated directly from the slope of the regression line relating the trait in parents and offspring. If the regression is of offspring on midparent (parental mean), the slope estimates h^2; if the regression is of offspring on one parent, the slope estimates $1/2(h^2)$.

In terms of variances and covariances, the equations for h^2 and r_A using offspring-parent regression are

$$h^2 = \frac{cov_{OP}(x)}{var_P(x)} \tag{6}$$

and

$$r_A = \frac{cov_{OP}(xy)}{\sqrt{cov_P(xy)\ cov_O(xy)}} \tag{7}$$

It should be noted that if the phenotypic variances in the two sexes are unequal, some adjustments to these equations are necessary (Falconer 1989).

The other primary method of estimating h^2 and r_A relies on phenotypic responses to an applied amount of selection on a trait. This method is derived from the fact that the response of a trait to a precise amount of selection, when expressed as a ratio, estimates its heritability. The "realized" h^2 of trait x under selection is estimated from the breeder's equation (Falconer 1989):

$$h^2 = \frac{R}{S} \tag{8}$$

where R is the change in the phenotypic mean of x after selection (the response) and S is the difference in the mean of x between the unselected and selected groups (the selection differential). Figure 2 depicts graphically how realized h^2 is related to R, S, and trait means.

The genetic correlation between traits x and y, when selection is imposed directly on trait x, can be estimated from the equation

$$r_A = \frac{CR(y)\ var(x)}{S(x)\ var(y)\ \sqrt{h^2(x)h^2(y)}} \tag{9}$$

where CR(y) is the correlated phenotypic response in trait y to selection on trait x, and var (x) and var (y) are the phenotypic variances of the two traits (Falconer 1989).

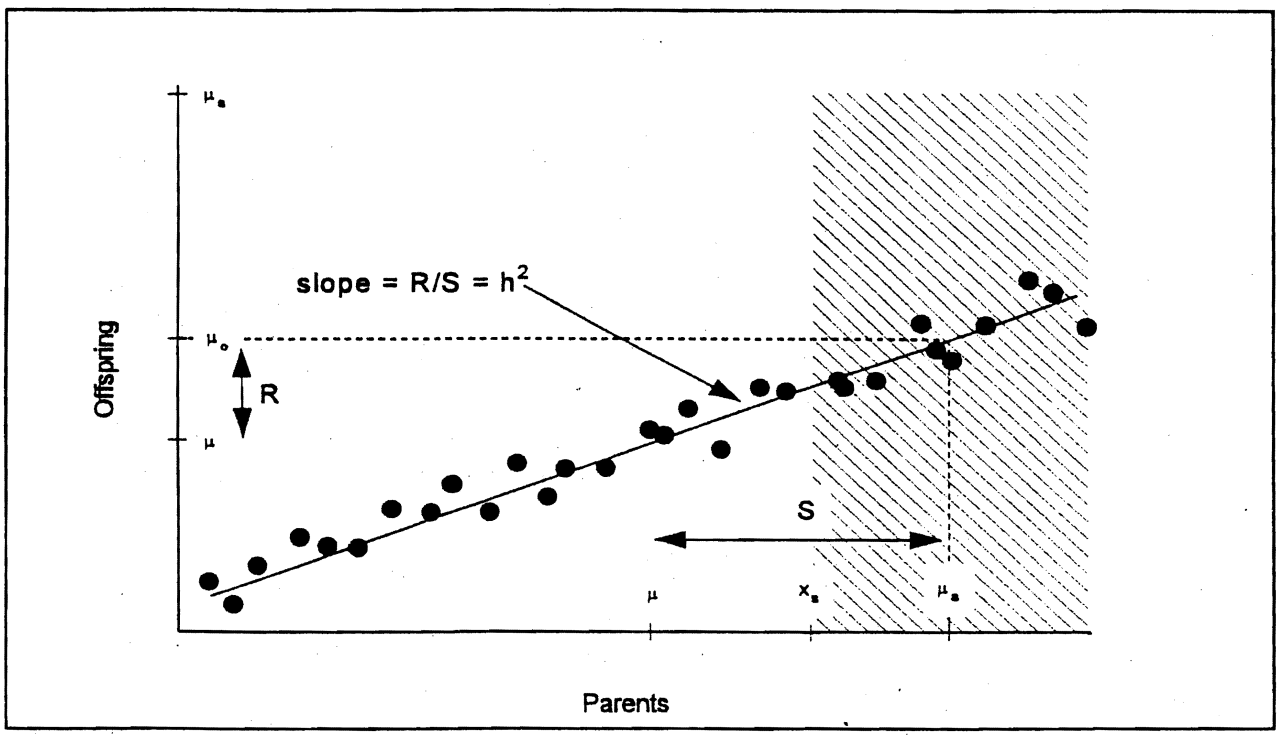

Figure 2. Graphical representation of truncation selection on a quantitative character and its phenotypic response. μ and μ_o are the respective trait means in parents and offspring before selection, x_s is the threshold for selection (the cross-hatched area represents parental phenotypes allowed to contribute to the next generation, and μ_s is the phenotypic mean of the selected parents). The selection differential is $S = \mu_s - \mu$. μ_o is the phenotypic mean of the offspring of the selected parents, and the response to selection (R) is $\mu_o - \mu$. The realized h^2 of the trait is estimated from the slope of the regression line, which for this one-generation case is R/S. Note that as h^2 increases, μ approaches μ_s.

The choice of technique depends on several factors, including the types of relatives that can be analyzed, the number of individuals available for analysis, and practical limitations on the design of the analysis. Each technique has peculiar strengths and limitations. More comprehensive discussions of these and other methods are presented by Becker (1984) and Falconer (1989). An introduction to some of the multivariate forms of these equations is presented by Lande (1982b, 1988b).

Heritabilities and genetic correlations are in principle simple to estimate, but they generally have such large sampling errors that the experimental requirements necessary to quantify them with reasonable precision can exceed the capacity of many facilities. Readers interested in experimental designs necessary to estimate these genetic parameters and their precision should refer to Kempthorne (1957), Klein et al. (1973), Klein (1974), Becker (1984), and Falconer (1989).

Estimates of genetic parameters depend on gene frequencies as well as the environment in which they are measured, and, therefore, any particular estimate is accurate only for the population and environment from which it is estimated. Additionally, selection over even just a few generations can alter heritabilities (Hill 1972, Sheridan 1988, Falconer 1989). Nevertheless, despite the difficulties associated with estimating these genetic parameters and the limitations of inference based upon them, such estimates are the only means to quantify with reasonable precision genetic and environmental sources of variation and covariation in traits under selection in captive broodstock programs.

Breeding Programs

Specific phenotypic and genetic objectives--Given the importance of quantitative traits and their analyses to plant and animal breeding programs, it is not surprising that most genetic research applied to fish has been directed toward aquacultural production. The major objective in conducting aquaculturally oriented investigations has been to develop populations of fish that express phenotypes or phenotypic traits leading to increased production efficiency and marketability. To accomplish this, 1) one to several traits are chosen such that their improvement will yield the maximum increase in production or marketability, 2) genetic parameters for these traits are estimated, and, based on the results, and 3) selection and breeding designs are implemented. While the details of such programs are often complex (Shultz 1985), the overall aim is to utilize available genetic variability to develop a population of fish with traits that enhance aquacultural production.

Genetic and environmental components of trait variation--***Growth and size.*** With increased production as the basis for the research conducted, several salmonid traits that contribute to aquacultural production have received major attention. Among the most prominent of these are growth and size. Growth and body size are important to successful commercial aquaculture and are relatively easy to measure; consequently, the emphasis on these traits is not surprising. In general, analyses have shown that the genetic component defining size characteristics (typically, length or weight) is of a magnitude that a reasonable response to selection can be anticipated.

Many of these analyses have been summarized by Gjedrem (1983, Table III) and Tave (1993, Tables 4.1 and 4.5). Estimated heritability estimates (h^2) for weight or length range from about 0.10 to about 0.90, depending on the species and population from which the fish were derived. In most cases, h^2 for size measured early in the life cycle is fairly large (0.50-0.70) and decreases as the fish ages and approaches maturity. This could suggest that the genetic determination of size during the early part of the life cycle is different than that for later in the life cycle. On the other hand, some results may be due to breeding designs that do not permit separation of maternal or common environmental effects from additive genetic effects. Research has shown that maternal effects on size are diminished at about 150 days after hatching in salmonids (Kincaid 1972), although in some species, environmentally induced size differences

expressed early in life can be maintained throughout the life cycle, at least in captive culture (Wohlfarth and Moav 1972).

Reproduction. The next area of quantitative genetic research that has received major emphasis in salmonids is reproduction. Research on salmonid reproduction can be divided into two groups of traits: maturation and egg production.

A phenomenon that is relatively common among fish species is precocious maturation. In Pacific salmon (*Oncorhynchus* spp.), precocious maturation is usually expressed in males, often referred to as "jacks." With the onset of maturation, growth is halted, and consequently, precocious maturation leads to the production of adult fish that are smaller (often dramatically) than those maturing at older ages. Because of the small size and the deterioration in flesh quality (i.e., decrease in red coloration and softening of texture) brought on by maturation, precociously maturing fish are generally unmarketable and, from an aquaculture-production standpoint, result in a loss of investment and resources. On the other hand, precocious male maturation may be a valuable life-history trait for the viability of fish populations in the natural environment (Gross 1991). Thus, numerous studies have been conducted to attempt to identify the genetic and environmental components responsible for this phenomenon, especially in salmonids.

Much of the research on sexual precocity has been focused on captive populations, especially of Atlantic salmon, and the results have implicated both genetic and environmental influences (Piggins 1974, Bailey et al. 1980, Glebe et al. 1980, Saunders 1986, Rowe and Thorpe 1990, Herbinger and Friars 1992). Although information for Pacific salmon is less complete, results from captive populations have also shown that genetic or environmental factors or both may be involved in determining the incidence of precocious male maturation (Garrison 1971, Childs and Law 1972, Hager and Noble 1976, Bilton 1978, Hard et al. 1985, Heath et al. 1994). In both Atlantic salmon (Thorpe et al. 1983) and coho salmon, (*O. kisutch*) (Iwamoto et al. 1984), the incidence of male precocity in progeny sired by precocious males was 4 to 5 times higher than that shown in progeny sired by non-precocious males. Yet other studies to estimate the heritability of this trait have yielded low values for rainbow trout, (*O. mykiss*) (h^2 = 0.04-0.06), coho salmon (h^2 = 0.05), and Atlantic salmon (h^2 = 0.07-0.25) (Gjerde 1986, Silverstein and Hershberger 1992). Consequently, lowering the incidence of precocious males in these species will require exacting selection and breeding procedures, which may be slow to yield results. On the other hand, recent work with chinook salmon (*O. tshawytscha*), which generally mature at any of a wider range of ages, has yielded h^2 estimates of 0.30 to 0.50 for the same trait (Heath et al. 1994). Response to selection in this species should be somewhat more rapid, at least in some populations.

In addition to genetic factors, some environmental factors have been shown to produce precocious maturation in such diverse species as brook trout (*Salvelinus fontinalis*), Arctic charr (*S. alpinus*), brown trout (*Salmo trutta*), rainbow trout (*O. mykiss*), and kokanee (*O. nerka*). These factors include high feeding rate (Bagenal 1969, Kato 1975, McCormick and Naiman 1984) and high temperature (Saunders et al. 1983, Papst and Hopky 1984, Crandell and Gall 1993a).

However, results from other studies are not in agreement with these conclusions. Iwamoto et al. (1984) showed that high temperature, while increasing the growth rate of coho salmon, did not produce a higher percentage of early maturing fish. Glebe et al. (1980) reported little difference in the proportions of precocious male Atlantic salmon raised at ambient and high temperatures. Nævdal (1983) reported faster growth of rainbow in full-strength seawater than in brackish water, but noted little difference in the maturity schedule of male fish. Similarly, Siitonen (1986) found more rapid growth of rainbow trout in brackish water than in fresh water, but a lower proportion of mature fish in brackish water. It would appear that fish of different strains have different capabilities for early maturation (Nævdal 1983) and that environmental factors will influence those fish in which sensitivity to external factors is high.

In addition to precocious maturation, the genetic component for "normal" age at maturity has been estimated for rainbow trout, Atlantic salmon, and chinook salmon. In rainbow trout and Atlantic salmon, Gjerde (1986) reported relatively high h^2 estimates for age at maturity (0.20 to 0.37), based on data transformed to a continuous scale. The estimates reported for chinook salmon were somewhat higher (0.4 to 0.6), although these results were obtained from selection response calculations (Hankin et al. 1993), which can differ substantially from estimates obtained from sib analyses or parent-offspring regression.

Within-season variation in spawning time has also been analyzed genetically in several species of fish, and this trait has been found to be highly heritable ($h^2 > 0.50$) (Campton and Gall 1988, Gall et al. 1988, Siitonen and Gall 1989, Crandell and Gall 1993a, Silverstein 1993). Thus, it would appear that the age at spawning and the timing of spawning within a season could both change rather rapidly with selection.

Obtaining large numbers of high-quality eggs at the right time of year is important for programming aquaculture production. In addition, egg size has been correlated with hatching percentage in rainbow trout (Gall 1974), with growth between 1-4 months of age in that species (Chevassus and Blanc 1979, Springate and Bromage 1985), and with survival and growth in chinook salmon (Fowler 1972). Heritabilities for egg weight, egg volume and egg number have been estimated for rainbow trout and Atlantic salmon (Gall 1975, Gall and Gross 1978, Haus 1984, Halseth 1984). The values are all fairly high ($h^2 = 0.19$-0.52) and suggest these traits could be altered relatively easily by selection. In fact, Gall and Huang (1988) developed a selection approach utilizing these traits for enhancing reproductive performance in rainbow trout.

Disease resistance. Another suite of traits important to aquaculture, although it has received relatively little attention, represents those involved in the inheritance of disease resistance. One of the earliest reports of successful selection with salmonid species was that of Embody and Hayford (1925), in which they demonstrated increased resistance to furunculosis (*Aeromonas salmonicida*) in brook trout. Subsequent genetic work on disease resistance has involved a variety of approaches (Chevassus and Dorson 1990), although relatively little research has emphasized the estimation of h^2 and the potential for response to selection. In fact, the only

other reported selection program to improve disease resistance in fish has involved infectious dropsy (*A. hydrophila*) in common carp, (*Cyprinus carpio*) (Schaperclaus 1962, Ilyassov 1987).

Considerable data on salmonids have demonstrated differences between populations in susceptibility to bacterial (Cipriano 1983; Wolf 1953; Ehlinger 1964, 1977; Snieszko et al. 1959; Suzumoto et al. 1977; Gjedrem and Aulstad 1974; Refstie 1982; Beacham and Evelyn 1992a), viral (Amend and Nelson 1977, McIntyre and Amend 1978, Silim et al. 1982, Okamoto et al. 1987) and parasitic (Zinn et al. 1977) infections. Published estimates of genetic influence on disease resistance range widely in magnitude. The heritability of resistance to bacterial kidney disease, caused by *Renibacterium salmoninarum* infection, was estimated at 0.00-0.38 in chinook salmon by Beacham and Evelyn (1992a).

However, Beacham and Evelyn (1992b) found that the heritability of mortality and time to death in chinook salmon challenged with *R. salmoninarum* was not different from zero. Heritabilities of these traits in coho and chum salmon (*O. keta*) were also low (Beacham and Evelyn 1992b). The heritability of resistance to *Vibrio anguillarum* infection (vibriosis) was estimated at 0.11 in Atlantic salmon (Gjedrem and Aulstad 1974) and 0.00-0.13 in chinook salmon (Beacham and Evelyn 1992a,b), whereas the heritability of the same trait in chum salmon was estimated at 0.5 (Smoker 1981).

On the other hand, the heritability of susceptibility to the causative agent for furunculosis in Atlantic salmon was estimated at 0.48 (Gjedrem et al. 1991) and from 0.00 to 0.34 in chinook salmon (Beacham and Evelyn 1992a,b). Measurement of resistance to viral hemorrhagic septicemia (VHS) in rainbow trout yielded h^2 estimates of -0.10 to 0.30 (Kaastrup et al. 1991). Assessment of tolerance to infectious hematopoietic necrosis (IHN) virus in sockeye salmon (*O. nerka*) produced h^2 estimates of 0.27 to 0.38 (McIntyre and Amend 1978).

There are a number of reasons why the genetic basis of disease resistance and susceptibility has not received more attention (Chevassus and Dorson 1990). A major reason is lack of understanding of the mechanisms fish employ to resist infection, the interactions among these mechanisms, and their relationship to fish mortality. For example, a pathogen may not invade the fish because of a barrier or destructive effect presented by mucous, gastric, or intestinal secretions. Alternatively, a pathogen may penetrate but be inactivated by spontaneous mechanisms such as bactericidal serum effect; macrophage, complement, or killer cells; or induced mechanisms such as interferon or antibody production.

These agents represent numerous means through which genetic variability could impact disease resistance in fish. Refstie (1982) studied the development of serum antibodies to *V. anguillarum* in rainbow trout families after immunization and calculated heritabilities of 0.15 to 0.25. However, low antibody titers and the consequent inability to identify large variations make these estimates suspect. Genetic analysis of serum hemolytic activity in rainbow trout as a measure of natural immunological defense revealed high h^2 estimates of 0.34-0.96 (Røed et al. 1990). However, the relationship of this trait to disease resistance has not been clearly defined.

Recent quantitative genetic work on susceptibility of rainbow trout to the myxosporean parasite *Ceratomyxa shasta* revealed several significant genetic components involved in the expression of susceptibility (Ibarra et al. 1994). Further, it appeared that the nature of the genetic causal components differed with time of exposure to the parasite.

As a consequence of this lack of basic information, there has been little progress on the use of genetics for improving disease resistance in fish. Instead, reliance on the use of antibiotic treatment and immunization to address disease problems continues.

Body composition and flesh quality. A final set of traits that has direct application to salmonid aquaculture is that dealing with body composition and flesh quality. A number of studies have been conducted to estimate the genetic determination of the quantity of various biochemical compounds (e.g., proteins and lipids) in the flesh. Estimates based on proximate analyses of proteins, lipids, and moisture in the flesh of rainbow trout and coho salmon have yielded relatively low heritabilities (< 0.10) for percent protein and somewhat higher values (0.14-0.47) for percent lipid and percent moisture (Gjerde and Schaeffer 1989, Iwamoto et al. 1990).

Another quality that is important to the marketability of cultured salmon and trout is flesh color. Genetic analyses of flesh color in rainbow trout and coho salmon have yielded h^2 estimates that would suggest a relatively rapid response to selection (0.27 and 0.30, respectively; Gjerde and Schaeffer 1989, Iwamoto et al. 1990). Work with chinook salmon suggested the possibility of a rather simple genetic system defining whether or not the flesh contained red coloration (Withler 1986). Hard (1986) observed both strong genetic and environmental effects on flesh pigment coloration in chinook salmon reared in seawater netpens. The genetic basis of pigmentation intensity in this species is unknown but is probably polygenic. Research on Atlantic salmon yielded low h^2 estimates (0.01) for meat color scores (Gjerde and Gjedrem 1984), suggesting that little change in the flesh coloration could be achieved in this species through selection.

Life history. In addition to work on performance traits that are of direct consequence to aquaculture production, a number of studies have been conducted to estimate genetic parameters on traits that could be termed "adaptive" (Table 4.1, Tave 1993). These include such characteristics as incubation performance, early development traits, survival, and behavior. Without going into detail on each of these traits, there are two generalizations that can be made from the data obtained from studies on salmonids.

First, traits associated with incubation, hatching, and early development exhibit a wide range of genetic underpinnings. Analyses of hatching rate (McIntyre and Blanc 1973, Beacham 1988, Sato and Morikawa 1982) and hatching time (Sato 1980, Sato and Morikawa 1982) have yielded relatively high heritabilities. On the other hand, most traits associated with survival during early development exhibit rather low heritabilities (< 0.20) (Kanis et al. 1976, Robison and Luempert 1984, Withler et al. 1987, Sato and Morikawa 1982, Beacham 1988). In addition, most

alevin and fry body traits (e.g., yolk weight, alevin weight and length, and fry weight and length) exhibit relatively low heritabilities (Withler et al. 1987, Beacham 1988). Since the early survival and physical characteristics of alevins and fry have been shown to be strongly influenced by the physical characteristics of the eggs (Fowler 1972, Gall 1974, Chevassus and Blanc 1979, Springate and Bromage 1985), a large amount of additive genetic variability (expressed by a high h^2) should not be expected for traits measured during early development.

In fact, Beacham (1988) concluded from his results that increased survival rates for the embryos and alevins would be more readily obtained by providing suitable environments than by selection or crossbreeding. By contrast, Withler et al. (1987) detected genetic variability in early developmental traits among three strains of chinook salmon; for example, h^2 estimates for survival of uneyed eggs varied from 0.0 to 0.21 among the strains. Additional investigations are needed to better define the contribution of genetics to phenotypic expression during early development.

Second, where genetic analyses have been conducted over different environmental conditions there appeared to be a fairly strong interaction between genotype and environment. Beacham (1988) identified the genetic determinants of early developmental traits in pink (*O. gorbuscha*) and chum salmon under three different temperature regimes (3°, 8°, and 16°C) and found that heritabilities varied in different ways with temperature. For example, the h^2 estimate for alevin tissue weight varied from 0.06 at 3°C to 0.45 at 16°C, while the h^2 estimate for hatching time was highest at 3°C (0.52) and lowest at 8°C (0.30). Studies of hatching time in steelhead (*O. mykiss*) demonstrated that the h^2 estimate differed depending on the type of container in which eggs were incubated (McIntyre and Blanc 1973). In addition, although the results vary, there may be greater additive genetic variance (i.e., higher h^2) for traits associated with survival when salmonids are raised under adverse conditions. Assessment of brown trout egg survival in acidic water showed h^2 estimates of 0.27 to 0.33 (Edwards and Gjedrem 1979). Estimates of h^2 for survival of Atlantic salmon in acidic water varied with the length of exposure, but were relatively high (0.29 to 0.72; Schom 1986).

Inbreeding and inbreeding depression. Inbreeding and inbreeding depression have been topics of considerable concern to fishery geneticists. Inbreeding can be defined as the mating of individuals more closely related to each other than to individuals chosen at random from the population (Wright 1978, Gall 1987). Related individuals have one or more common ancestors and, therefore, may have received one or more identical genes. The measure of inbreeding, designated the inbreeding coefficient (F), is the probability that two alleles at a common locus are identical copies of an ancestral allele (Falconer 1989). Inbreeding can occur in populations either as a directed or a random process, and it can occur in large or small groups of animals. The basic impact of inbreeding on the genetic constitution of a population is an altered distribution of genotypes toward fewer heterozygotes and more homozygotes. This distribution leads to the "unmasking" of deleterious recessive alleles and reduces the frequency of genotypes expressing codominance and overdominance.

Often, the result of increased inbreeding is a decrease in mean phenotypic value of one or more traits with respect to fitness, a phenomenon known as inbreeding depression. Inbreeding depression is commonly measured by comparing the average phenotypic values between an inbred population and the base population from which it was derived (Gall 1987). The traits often most strongly affected by inbreeding are those connected with reproductive capacity (e.g., fecundity, egg size, hatchability) or physiological efficiency (e.g., growth rate, feed conversion efficiency, survival) (Falconer 1989).

In general, inbreeding depression tends to increase in proportion to the inbreeding coefficient during the early stages of inbreeding. However, as Kincaid (1983) pointed out, when the impacts of inbreeding on viability and survival traits become severe enough to result in the actual loss of inbred lines, the relationship between inbreeding depression and the inbreeding coefficient is often unpredictable.

Because the exact level of inbreeding in a population is usually impossible to measure, inbreeding in practice is measured relative to the initial level in the source population (or assumed to be initially zero; Lande and Barrowclough 1987). A major problem when investigating inbreeding and its effects in populations of fish is that information on the breeding history is generally inadequate to determine even the initial level of inbreeding. Genealogies are rarely known for fish populations. This precludes the use of pedigrees for calculation of inbreeding coefficients (Gall 1987), which requires definition of the relationship of individuals over generations and the ability to follow and analyze a pathway of inheritance (Wright 1969). Consequently, estimates of inbreeding are most often based on the number of breeding individuals in the population. The generalized formula for making this calculation with a random mating population is

$$\Delta F = \frac{1}{2N} \qquad (10)$$

where ΔF is the expected increase in the inbreeding coefficient per generation and N is the number of individuals that mate to produce the next generation.

Under non-ideal conditions (e.g., non-random mating, different numbers of each sex, or greater than binomial variation in family size) the "effective" number of individuals contributing to subsequent generations can be substantially lower than the census number. This effective number of breeding individuals (N_e) can be estimated by several methods (Falconer 1989) and is substituted for N in the above equation to determine the rate of increase in inbreeding. The major problem with this approach to calculation of the inbreeding coefficient is that the estimates of inbreeding are highly sensitive to departure from non-ideal conditions, especially variation in family size. Consequently, values estimated by this procedure yield overestimates of the actual inbreeding rate after the first generation, and the overestimation increases as the effective population size decreases.

Additional factors that complicate accurate estimation of inbreeding include overlapping generations and nonrandom mating (as well as mutation, gene flow, and selection). Also, estimates using population size (N or N_e) yield an average rate of inbreeding over the population and do not provide information on individual fish. Finally, these estimates depend on the assumptions that population size is stable between generations and that mating is random.

Nevertheless, in the absence of records on breeding practices and pedigrees, this method of approximation provides a valuable estimate of the rate that inbreeding accumulates in a population. The calculated ΔF value for each generation can be added to the inbreeding coefficient of the previous generation to obtain an estimate of the current level of inbreeding (Falconer 1989). Falconer (1989) discusses this topic in detail and provides alternative formulae for estimating levels of inbreeding when population size or family size vary or when generations are not discrete.

Results from studies on the effects of inbreeding in fish have shown that increasing the level of inbreeding in a population yields reduced performance in a variety of traits. The equivalent of one generation of brother-sister mating (i.e., full-sib mating; $\Delta F = 0.25$) led to an increase in the occurrence of fry deformities in rainbow trout (Aulstad and Kittelson 1971). Results from this same level of inbreeding in brook trout demonstrated a decrease in body weight of 27.7% after 7 months and 34.4% after 19 months (Cooper 1961).

Longer-term studies have permitted estimates of changes in traits with increased inbreeding. Bridges (1973) reported depression estimates of 5.1% in fish weight and 0.4% in formalin tolerance at 150 days of age in rainbow trout with each 10% increase in inbreeding. Gjerde et al. (1983) reported that three generations of inbreeding in rainbow trout led to increases of 2.5% in eyed egg mortality, 1.9% in alevin mortality, and 3.2% in fry mortality. They also found decreases of 3.0% in fingerling growth and 5.1% in growth to 18 months in seawater per 10% increase in inbreeding. One problem with these results is that increases in inbreeding were achieved by use of sequential generations, which necessitates comparing data on trait performance over environmental conditions that are not exactly comparable. This method interjects error in the estimates of inbreeding depression that is difficult to quantify.

To address this problem, Kincaid (1976a,b) designed an experiment that allowed comparisons between rainbow trout families with different levels of inbreeding, as well as comparisons between related but outbred families, in a single year. Analyses of the phenotypic values for a number of traits expressed with the equivalent of one and two generations of full-sib matings ($\Delta F = 0.25$ and 0.375, respectively) revealed that inbreeding depression increased with the level of inbreeding. At inbreeding coefficients of 0.25 and 0.375, respectively, fry deformities increased 37.6% and 191% over outbred levels. Also, feed conversion efficiency decreased by 5.6% and 14.9%, fry survival declined by 19.0% and 29.7%, and fish weight at 147 days and 364 days of age decreased by 11.0% and 13.4% ($\Delta F = 0.25$) and 23.2% and 33.5% ($\Delta F = 0.375$).

Subsequent work using this same design to investigate field performance and later growth and reproductive traits revealed lower fishery recovery with inbred groups, resulting from lower rates of survival and growth (Kincaid 1983). Additionally, the total weight of eggs produced decreased with increased levels of inbreeding, probably resulting from the smaller size of inbred females. Lower performance in the natural environment was also shown in Atlantic salmon by lower recapture frequencies for members of inbred families (Ryman 1970). Thus, inbreeding can impact all phases of the life cycle and the deleterious effects appear to be, to a large degree, linear with increases in the level of inbreeding.

There are several precautions that need to be considered when dealing with inbreeding and its phenotypic effect, especially on animals with little or no history of domestication. First, accurate prediction of the response of a particular trait to increased levels of inbreeding may not be possible. For most quantitative traits, little is known about the number of genes that define the traits, the frequencies of these genes in the population, or the relationships between the structure (e.g., linked vs. independently segregating) and expression (e.g., additivity vs. dominance) of these genes. Since inbreeding affects the genotypic distribution within the population, the frequency, arrangement, association, and interaction of genes determining a trait are all critical to any change in phenotypic expression. In addition, environmental factors play a major role in expression of quantitative traits and may act to "buffer" otherwise negative changes in genotypic distribution.

Second, fish populations with little history of domestication could exhibit stronger responses to increased levels of inbreeding (i.e., inbreeding depression) than those with some history of controlled breeding. One predictable result from increased levels of inbreeding is the unmasking of deleterious recessive alleles via increased homozygosity and the potential for elimination of these alleles from the population. The unmasking and elimination of these alleles may have fewer opportunities to occur under natural circumstances and, thus, inbreeding depression levels may be somewhat higher as an initial response to an increase in inbreeding.

Finally, changes in the phenotypic expression of a trait with increased levels of inbreeding are a consequence of a number of genetic factors. Perhaps the most important to consider is selection, although other elements (e.g., migration, mutation, drift) may play a role. Although natural selection acts to eliminate the less fit genotypes produced by inbreeding, it may not be sufficient to offset inbreeding depression. However, a change in selection pressures, such as might be expected in the initial phases of domestication, can counteract the effects of inbreeding and result in weaker inbreeding depression. There is some evidence for this effect in domesticated animals (Falconer 1989). These considerations lead to the conclusion that assessment of the effects of inbreeding must be approached very carefully and with an appropriate experimental design (Gall 1987).

Utility and Reliability of Genetic Estimates

In attempting to interpret these data, it must be remembered that heritability is calculated as a proportion of the total phenotypic variance and that its magnitude for a specific trait in a specific population will be strongly influenced by nongenetic variation. Allendorf et al. (1987) pointed out that while fish species demonstrate a large amount of phenotypic variation (as measured by coefficient of variation), comparison of heritability estimates for similar traits with other animal species reveals smaller values in general. This could be explained by greater fish susceptibility or responsiveness to environmental factors mediated by 1) poikilothermy, 2) indeterminate growth, and 3) flexibility of age and size at sexual maturation.

Estimation of environmental influence on quantitative traits can be achieved from results of studies analyzing the magnitude of genotype-environment interactions. These interactions can be defined as the consistent variation in phenotypic expression of specific genotypes under different environments. These interactions are usually estimated by analyzing the phenotypic response of organisms with the same or similar genotype(s) under different environmental conditions.

For example, Donald and Anderson (1982) found that 72% of the total variation in weight of 2-year-old rainbow trout from a strain stocked in mountain lakes could be attributed to stocking density and overall productivity of food organisms, i.e., environmental variation. In another study, Ayles and Baker (1983) determined that growth differences among rainbow trout strains stocked in central Canadian lakes were primarily due to lake-to-lake variability. Under more controlled culture conditions, Atlantic salmon strain (genetic) differences accounted for only 6.4% and 7.0% of the total phenotypic variance in body weight and body length, respectively (Gjerde and Gjedrem 1984).

In addition, Gjerde (1986) found large variation in percent immature fish in comparisons between fish from the same full-sib families (families of fish sharing the same parents) reared on different fish farms, fish from different year classes reared on the same farm, and full-sib fish reared in different cages on the same farm. Further, in analyses of results from rainbow trout grown under various combinations of density and feeding regimes, environmental factors accounted for more than 40% of the total variance in weight (Iwamoto et al. 1986). Consequently, it is critical that environmental factors be estimated when attempting to interpret quantitative genetic influence on phenotypic variation.

How satisfactorily h^2 estimates explain the genetic component of phenotypic variation for a trait can be determined only through a directed selection program that assesses the magnitude of actual response. There have been few reports on directed selection programs of sufficient length to test the predictions of quantitative genetic analyses with fish (Hershberger 1993). Those that have been conducted beyond one or two generations have demonstrated the efficacy of selection programs to develop strains of fish with improved aquaculture characteristics.

Probably the most extensive program with this purpose is in Norway, where stocks of Atlantic salmon are being developed for aquaculture in marine net pens (Gjedrem et al. 1987). There are also programs in Israel and Hungary for the development of carp stocks for pond rearing (Moav and Wohlfarth 1966, Bakos 1976). In the United States, several programs have been conducted with salmonids that have applications to various sectors of the aquaculture industry (Donaldson and Olson 1957, Gall and Gross 1978, Hershberger et al. 1990). Results in all of these studies have demonstrated that selection can have major effects on phenotypic traits in fish, although not all studies were based on prior quantitative genetic analyses or had appropriate experimental designs for estimation of genetic influence.

Where selection programs with fish have been conducted based on a priori genetic heritability estimates, the realized estimates have generally corroborated estimates made before initiating selection (see Sheridan 1988 for counterexamples from other domesticated organisms). For example, programs conducted to increase size-related traits in salmonids have shown that h^2 estimates are adequate predictors of the gains that can be realized (Gjedrem et al. 1987, Hershberger et al. 1990). Not surprisingly, however, work with other fish has demonstrated that the results are dependent on the species and strain being analyzed and tested.

Study of the common carp has demonstrated an asymmetrical response to selection (Moav and Wohlfarth 1976): no response was realized in selection for larger fish, but a response was apparent when selecting for smaller fish. Also, a positive response was reported for divergent selection on body weight in the Tifton strain of blue tilapia, *Tilapia aurea* (Bondari et al. 1983), whereas little response to selection on this trait was realized in either the Ivory Coast strain of *T. aurea* (Tave and Smitherman 1980) or the Ghana strain of Nile tilapia, *Oreochromis niloticus* (Hulata et al. 1986).

These differences have been attributed to a lack of genetic variability in these latter strains, due to either a long history of selection or a small founding population size or both. Although many other explanations can be cited for either an asymmetrical response to selection or a lack of response (Falconer 1989), the exact mechanism will be a function of the genetic composition of the population of fish being investigated.

A final area where breeding programs have been valuable in understanding quantitative genetic variation is in characterizing genetic relationships among traits. These relationships are estimated by analyses of correlations between traits and, depending on the experimental design, can be defined on a phenotypic or genetic basis (Falconer 1989). A correlation between traits, whether phenotypic or genetic, is a measure of the proportion of observed covariance between them, scaled by the square root of the product of the trait variances. It is typically calculated from variance and covariance components in an analysis of variance.

Estimation of the genetic correlation differs from that of the phenotypic correlation in that its computation requires knowledge of the genetic relationship of the individuals that the variance and covariance components are estimated from (see Equation 5). Clearly, given the number of

traits that have been analyzed in fish, delving into the variety of combinations of traits that may exhibit genetic or phenotypic correlations is a complex undertaking (see Table 4.5, Tave 1993). Nevertheless, there are several generalizations apparent from the results that have been obtained.

First, in most investigations with fish it has been difficult to obtain accurate estimates of relationships between traits other than phenotypic correlations between measurements of the same trait made at different points in the life cycle. Until recently (with the development of the Passive Integrated Transponder (PIT) tag) it has not been possible to ensure that measurements at different stages of the life cycle were conducted on the same animal. Without this capability there is an unknown quantity of environmental influence on the results, and thus potential bias in genetic correlation estimates.

Second, correlations between measurements made on the same body-size trait at different times decrease with age. For example, in rainbow trout the phenotypic correlation between length at 6 months and length at 12 months was 0.81, whereas length at 6 months and length at 30 months exhibited a correlation of 0.06 (Møller et al. 1979). Estimations of correlations between lengths at intermediate points in the life cycle were between these two values. Consequently, it would seem that either different genes affecting size are expressed at particular ages or environmental factors are exerting cumulative influence over time.

A third general observation is that genetic correlations between traits associated with body size (e.g., weight and length) and between these traits and other morphological measurements at a particular life cycle stage can be rather high (> 0.80). For example, the genetic correlation between length and weight in coho salmon at 84 days post swim-up was reported to be 0.95 ± 0.04 (Iwamoto et al. 1982). In Atlantic salmon, the genetic correlation between these same two traits at two years of age was reported to be 0.99 ± 0.01, and the genetic correlation between body length and pectoral fin length at 1 year was 0.95 (Riddell et al. 1981). Thus, it appears that there is a strong pleiotropic effect of genes associated with morphological size.

Finally, it appears that body-size traits are fairly strongly correlated with some prominent life-history traits. Gall (1975) and Huang and Gall (1990) obtained some relatively high genetic correlations between female body weight and egg volume (0.37-0.47); these and other data suggested that body size has evolutionary implications for reproductive performance. For pink salmon, Beacham and Murray (1988) obtained genetic correlations in excess of 0.90 for the relation between 315- and 500-day body weight and the gonadosomatic index in males and females. In coho salmon, Saxton et al. (1984) found that the best predictor of successful transfer to saltwater was body weight at transfer. For rapidly growing coho salmon raised in freshwater, moderate phenotypic correlations were estimated between precocious maturation and body length (0.45) and weight (0.47) (Silverstein and Hershberger 1992). Consequently, salmonids appear to exhibit a fairly strong genetic and phenotypic relationship between body size and life-history traits, at least in an aquacultural setting.

This brief consideration of results from breeding programs indicates that information on genetic variation in quantitative traits in salmonids has been directed primarily at traits affecting aquacultural production. For example, our understanding of the quantitative genetic basis of life-history variation is meager. However, new developments in technology, such as molecular genetic markers and PIT tags or other nonlethal tags that identify individuals, will undoubtedly allow increases in the type and number of traits analyzed and in the breadth of experimental applications.

Mixed-Stock Management

Quantitative genetic analyses as a source of information for assisting stock identification of salmon in mixed-stock fisheries are problematic because unequivocal identification of component populations is a management requirement. Given the synoptic nature of the statistical analyses used for quantitative traits and the interplay of environmental influences and genetics in the expression of phenotypic differences, the results are usually not definitive enough for effective mixed-stock management. The integration of molecular and quantitative genetic methods may be useful in providing a means to identify the genes responsible for the determination of quantitative traits affecting performance or fitness (Paterson et al. 1988, Lander and Botstein 1989, Andersson et al. 1994). However, this approach has limited ability to explain the genetic basis of quantitative traits because it relies heavily on the analysis of single loci with large effects on the character.

Nevertheless, there are at least two important applications of quantitative genetic analyses to management of mixed-stock fisheries. First, identification of quantitative genetic differences between groups of animals can provide a basis for separate management of groups that had been considered genetically homogeneous. For example, Gharrett and Smoker (1993) identified pink salmon subpopulations within a stream by their temporal segregation in adult return timing. Other measures of genetic differentiation (e.g., allozyme electrophoresis) did not separate these two groups, and genetic variability in return timing apparently was the factor responsible for their divergence. These results are consistent with a hypothesis of finer population differentiation at the level of life history (quantitative trait loci) than at neutral genetic markers (single loci) (Utter et al. 1993).

The second application is the identification of changes that may result from the selective effects of management activities such as fishing regulations. Many of the restrictions intended to control harvest are selective on fish populations by the way they are practiced. For example, Burgner (1963) demonstrated that the selective effects of the gillnet fishery on Bristol Bay sockeye salmon could decrease the size of a population and skew the sex ratio. Alexandersdottir (1987) suggested that the run timing of southeastern Alaskan pink salmon was altered as a result of directed fishing.

There have been few analyses of such effects on anadromous salmon that utilize a quantitative genetic approach. Ricker (1981) analyzed a large data set on body size collected

over a number of years from different types of fisheries to estimate a realized heritability for this trait on the basis of realized response to selection. Results from analysis of body size (weight) data on pink salmon yielded a heritability estimate that was very close to similar estimates for other fish species (h^2 = 0.22-0.33). His interpretation of this result was that selection imposed by gillnet, troll and seine fisheries was a major cause for the observed decrease in body size of pink salmon in British Columbia. Hankin et al. (1993) used a similar approach to estimate, after a single generation, the realized h^2 of age at maturity (h^2 = 0.4-0.6) in hatchery chinook salmon harvested in troll fisheries. Their result indicates a strong potential for this character to decline under exploitation by size-selective (and, hence, age-selective) fisheries. Since many life-history traits in fish populations are quantitative in nature, it seems prudent to emphasize the quantitative genetic analysis of these traits.

Genetic Conservation

Quantitative traits are the major genetic determinants of the adaptive capability of a population, and their genetic architecture constitutes a fundamental constraint on the population's evolutionary pathway. Consequently, the monitoring of these traits should be an integral part of a genetic conservation program. However, while much evidence suggests substantial genetic determination of adaptive traits, there have been few attempts to estimate the magnitude and type of genetic determinants underlying these traits. The value of these estimates would reside in 1) better understanding of the genetic and environmental basis of variation in adaptive traits and 2) prediction of the evolutionary response of these traits to selection or other factors. One problem of growing interest to salmon geneticists and managers that such estimates could provide a better understanding of is the genetic consequences of artificial propagation for hatchery and natural populations.

Genetic concerns in artificial propagation--There is evidence that quantitative traits affecting adaptation in salmonids can change under human influences, including harvest practices, habitat alteration, and artificial propagation (as in conventional hatcheries or in captive broodstock programs). Until recently, the effects of artificial propagation on adaptation of salmonids in the wild were not widely appreciated. In the last 10-15 years, however, many authors have pointed to genetic problems that can arise during salmon artificial propagation (Krueger et al. 1981, Reisenbichler and McIntyre 1986, Allendorf et al. 1987, Lichatowich and McIntyre 1987, Nelson and Soulé 1987, Lannan et al. 1989, Hindar et al. 1991, Waples 1991a). Much of this attention has been sparked by declines in productivity (returns per spawner) of hatchery populations.

To address some of these problems, a number of authors have drafted documents in an attempt to guide hatchery managers in certain areas of genetic management; these authors include Hershberger and Iwamoto (1981), Hynes et al. (1981), Krueger et al. (1981), Kincaid (1983), Davis et al. (1985), Allendorf and Ryman (1987), Kapuscinski and Jacobsen (1987), and Simon (1991). Almost without exception, the detailed guidelines developed and recorded by these

workers focus heavily on the maintenance of a large effective breeding population size (N_e), the monitoring and control of sex ratio, and broodstock sampling.

The immediate genetic concerns of artificial propagation for conservation may differ substantially from those of artificial propagation for enhancement and mitigation. For example, minimizing the genetic differentiation of hatchery fish and the natural fish they are intended to supplement can be of equal concern to maximizing genetic variability in the hatchery population in a conservation program. However, few of the publications that address the genetic management of threatened or endangered populations of fishes, including papers by Meffe (1986), Nelson and Soulé (1987), Kapuscinski and Phillip (1988), Johnson and Jensen (1991), and Ryman (1991), have explicitly made this point. Much of this work has also failed to stress the importance of minimizing unintentional selection (Tave 1993) during captive culture.

Lichatowich and Cramer (1979) surveyed natural variation in a number of life-history characters in several populations of Pacific salmon. They argued that investigators interested in tracking natural variation should monitor traits that have a strong influence on survival rather than survival or abundance directly, because natural variation in survival and abundance may be large enough to preclude detecting effects of hatchery practices on these traits in natural populations, at least over the short term. Lichatowich and Cramer (1979) based their recommendation on the greater statistical power to detect changes in traits such as growth rate, age and size at outmigration and return, migration timing, and spawn timing. These are precisely the kinds of traits that are amenable to quantitative genetic analysis.

Interest in the use of artificial propagation as a tool to assist in the recovery of threatened or endangered populations has grown dramatically in the past few years. Reviews of the salmon supplementation literature (Bakke 1987, Miller et al. 1990, Steward and Bjornn 1990, Cuenco 1991, Cuenco et al. 1993) have identified several areas where deficiencies make it difficult to determine the genetic consequences of supplementation for natural populations. These deficiencies generally fall into two categories: information on levels of genetic variation for traits important to reproductive success in the wild, and knowledge of the effects of supplementation on this genetic variation.

Concern about the uncertainties associated with supplementation of natural salmon populations with artificially propagated fish has produced a flurry of recent research intended to guide the use of this technique (e.g., Busack 1990, Kapuscinski et al. 1991, Hard et al. 1992a, RASP 1992, Kapuscinski and Miller 1993, Lichatowich and Watson 1993). Recommendations of this research have focused on ways to reduce the genetic risks associated with supplementation.

Unfortunately, while these guidelines have often provided specific information on methods to minimize the *random* loss of genetic variation, they have generally failed to recommend specific ways to minimize genetic change that may arise during captive culture through the *directional* process of selection. Such guidelines must often be constructed from information in the quantitative genetics and animal breeding literature, which may not be easily accessible to fisheries

biologists with little or no genetics training. The texts by Kempthorne (1957), Turner and Young (1969), Mather and Jinks (1982), Becker (1984), and Falconer (1989) are prominent storehouses of this information.

Inbreeding and loss of genetic variability during supplementation--Recently, Ryman and Laikre (1991) pointed out that, in a supplementation program, the effective size of the hatchery-wild system as a whole is a more important consideration than the effective size of either component separately. They showed that differentially enhancing only part of the gene pool of a population through artificial culture (as might easily occur in a captive broodstock program) can lead to higher levels of inbreeding and loss of genetic diversity in the overall population. However, Ryman and Laikre's study assumed discrete generations and considered only a single generation of enhancement.

In a study conducted as part of a genetic monitoring and evaluation program for Snake River chinook salmon and steelhead (BPA Project 89-096), Waples and Do (in press) evaluated more fully the Ryman and Laikre effect in age-structured Pacific salmon populations. Waples and Do used computer simulations to model the level of inbreeding in the hatchery-wild system as a whole and how this level is affected by various types of captive broodstock programs. Three scenarios were considered in the simulations: control (no supplementation), increase (population increases through supplementation and remains large), and crash (population temporarily increases but declines after supplementation ends).

Results were summarized in terms of the parameter ΔIBD, which represents the change in level of inbreeding in the postsupplementation population compared to the control. Waples and Do (in press) found that: 1) The single most important factor affecting ΔIBD was whether the increase in population size was sustained. Compared to the control, higher levels of inbreeding were found under the crash scenario and lower levels were found under the increase scenario. 2) The absolute number of wild adults taken for broodstock had a stronger influence on ΔIBD than did the proportion of the population sampled. 3) In both the crash and increase scenarios, over 99% of genes in the postsupplementation population could be traced to hatchery fish. In the crash (but not the increase) scenario, broodstock taken in later years of the program dominated the final genetic makeup of the population. 4) ΔIBD was higher if broodstock collection lasted longer than 1-2 generations, but the increase was not linear and there were some exceptions to the trend. 5) Marking hatchery fish so that they could be avoided in subsequent broodstock collections postponed further increases in ΔIBD but did not prevent them altogether. Furthermore, appreciable reductions in ΔIBD occurred only when the proportion marked was nearly 100%. 6) Captive breeding and rearing strategies such as sib-avoidance mating and equalizing progeny number generally had little effect on ΔIBD.

These results should be useful in evaluating genetic risks of captive broodstock programs and in developing programs to minimize these risks. However, although Waples and Do (in press) attempted to provide a comprehensive evaluation of the Ryman-Laikre effect on captive broodstock programs for Pacific salmon, it was not possible to consider every possible

combination of parameter values. Similarly, there were a number of factors not considered in their study (e.g., selective changes due to the culture environment and fitness consequences of particular values of ΔlBD) that should also be evaluated in deciding whether or how to implement captive broodstock programs.

Quantitative genetic issues in supplementation--Research to guide the use of artificial propagation for supplementation of natural salmon populations has generally provided few details about how supplementation should be monitored and about which traits should be monitored closely. The lack of guidance on how to detect, monitor, and respond to the effects of selection in hatchery fish undoubtedly has resulted largely from uncertainties about how adaptation operates in novel environments. This process is a topic of active research in evolutionary genetics (Bryant et al. 1990, Holloway et al. 1990, Allendorf 1993, Frankham et al. 1993).

As the previous review of quantitative genetic studies indicates, most quantitative genetic research on salmonids has been applied primarily to estimate levels of genetic variation and covariation in traits important to hatchery production or market demand. Genetic variation and covariation in life-history traits important to adaptation in the wild are, with few exceptions, unknown for most Pacific salmon populations. These traits include age at juvenile outmigration and adult maturity, juvenile outmigration and adult run timing, fecundity, and habitat preference (Riggs 1990, Kapuscinski and Miller 1993). To this list could be added stage-specific survival rates, sex ratio, egg size, development and growth rates, food conversion, temperature and pH tolerance, body morphometry and composition, migration tendency, stamina and burst swimming speed, stress and disease resistance, seawater tolerance, agonistic behavior and competitive ability, and homing ability (Steward and Bjornn 1990, RASP 1992).

Estimation of genetic variation and covariation is essential to the implementation of quantitative genetics in the management of natural populations, but other genetic problems also warrant investigation. There are a number of quantitative genetic issues relevant to salmon supplementation efforts, including captive broodstock programs, that remain largely unexplored. One of these issues is inbreeding depression expressed during short-term captive propagation of small populations composed of close relatives. Another is domestication selection, or directional genetic change during captive propagation that results from adaptation to the protective culture environment. A third issue is outbreeding depression resulting from interbreeding between cultured and wild individuals.

Inbreeding depression. Geneticists generally consider inbreeding depression to be the one of the most serious threats to the viability of small captive populations (Ralls and Ballou 1983, Lande and Barrowclough 1987, Ralls et al. 1988, Simberloff 1988, Hedrick 1992, Hedrick and Miller 1992). As described above, inbreeding depression is the reduction in fitness resulting from mating between close relatives that occurs by chance in small populations, or from assortative mating in large populations. Inbreeding depression is a consequence of the expression of deleterious recessive alleles as homozygosity increases; therefore, it depends largely on dominance, or interactions between alleles within loci (Falconer 1989, Lynch 1991). Inbreeding

depression is an important concern in captive broodstock programs for threatened or endangered species because it addresses the additional risk of extinction that results when related individuals are mated. In populations that have existed at low numbers for any appreciable length of time, this risk can be high (e.g., Templeton and Read 1984).

Inbreeding depression has been documented repeatedly in many plant and animal taxa (Hedrick et al. 1986, Charlesworth and Charlesworth 1987, Hedrick 1992). To our knowledge, however, in Pacific salmon inbreeding depression has not been documented quantitatively in hatchery populations, and its prevalence in wild salmon populations is unknown (Allendorf and Ryman 1987, Gall 1987). Inbreeding depression is often a laborious quantitative genetic problem to investigate because, unlike estimation of simple inbreeding, estimation of inbreeding depression requires the assessment of some measure of fitness as well as knowledge of the parentage of the individuals under consideration. Such assessment requires a level of manipulation that is not typically practiced (and, indeed, would be difficult to practice) in most production situations. Nevertheless, further research is needed to characterize the relationship between inbreeding and fitness in salmon populations.

In addition to examining the extent of inbreeding and inbreeding depression in both hatchery and natural populations, three research topics are particularly worthy of attention: 1) effective mating schemes to minimize further inbreeding, 2) estimation of critical levels of inbreeding that lead to substantial reductions in fitness, and 3) conditions necessary to avoid or recover from inbreeding depression (i.e., amount and type of outbreeding) (Waples, in press).

Some experimental studies have shown that even though population bottlenecks increase the opportunity for inbreeding depression to occur, these events sometimes increase the genetic variance in quantitative traits (Bryant et al. 1986, Carson and Wisotzkey 1989, Cohan et al. 1989, see also Hard et al. 1993). This expression of "hidden" variance is thought to result from a redistribution of genetic variance under strong genetic drift (Robertson 1952; Griffing 1960; Cockerham 1984; Goodnight 1987, 1988; Gimmelfarb 1989; Willis and Orr 1993). Nevertheless, strong bottlenecks pose considerable risk to populations. Bryant et al. (1990) found that fitness dropped sharply after a single bottleneck (of 1, 4, or 16 mating pairs) in *Musca domestica*, although fitness in surviving lines began to rise by the third successive bottleneck. In *Drosophila melanogaster*, Frankham et al. (1993) found that selection for high fitness in inbred lines substantially reduced the loss in fitness due to inbreeding depression; however, fitness was still significantly lower than in an outbred, unselected control line. Thus, while bottlenecks may provide some adaptive opportunities for populations in unstable environments, the additional risk of extinction posed by bottlenecks should be avoided if possible (Willis and Orr 1993).

Domestication selection. In captive broodstock programs intended to supplement natural populations, the genetic objective must be to minimize genetic and phenotypic divergence of cultured fish from the natural fish they are intended to supplement. The opportunity for selection to produce this divergence in a captive broodstock program is large because fish are cultured entirely in captivity for one or more generations. The greater potential control of mortality in

captive broodstocks may limit the effects of natural selection during captivity--but only if the genetic divergence that results is sufficiently low that it entails only nominal fitness costs once the fish are released to the wild.

Genetic change in a quantitative character depends on three basic parameters: the amount of genetic variation for that character, the amount of selection exacted by factors in the environment, and the number of generations exposed to the environment. Only recently has it been explicitly recognized that the hatchery environment may be sufficiently different from the natural environment that, even if fish are held for a few generations for only a small portion of their lives in captivity, substantial quantitative genetic divergence of hatchery and wild fish might occur.

Genetic divergence can result from selection or genetic drift (as well as mutation and migration), and it can be difficult to distinguish between the effects of these agents if populations are at low effective population sizes (Lynch 1988). Yet this is precisely the condition that exists when establishing most captive broodstock programs. The effects of drift, because they are determined by the effective population size, are to a large degree determined by the time a captive broodstock is established and are typically random in nature. By contrast, genetic change through selection is directional (i.e., through adaptation to the protective culture environment) and can be more difficult to control.

Artificial selection is not required in order to domesticate animals, a process that can occur through "inadvertent" selection (Doyle 1983, Kohane and Parsons 1988). In supplementation schemes such as captive broodstock programs, which are designed for reintroduction of animals to the wild, domestication is not desirable. Domestication is a process of adaptation to a novel, usually controlled environment, and this process is only partially independent of experimental design. The environment may be artificial, but the adaptive process is a natural one. The bulk of evidence for domestication in wild animals comes from animal breeding experience (Spurway 1952, Hale 1969, Price and King 1969, Lande 1983, Price 1984, Fredeen 1986, Kohane and Parsons 1988) and from experimental work on other organisms, primarily invertebrates (Doyle and Hunte 1981a,b; Frankham et al. 1986; Parsons 1986; Holloway et al. 1990; Briscoe et al. 1992).

Because domestication is a form of adaptation, it is thought to involve primarily quantitative characters that affect fitness. Evolutionary theory predicts that the number of genes affecting trait expression should increase with the trait's correlation with fitness. Indeed, many quantitative characters (especially life-history traits) which are known to affect fitness show evidence of polygenic control (Wright 1968, Lande 1981). Williams (1966), Gadgil and Bossert (1970), Rose (1982), and Clark (1987) have argued on theoretical grounds that many of these constituent genes have pleiotropic effects on different characters, which are manifested as genetic correlations between these traits.

Adaptation should drive allelic combinations with positive genetic correlations with respect to fitness toward higher frequencies, with the result that, when near selective equilibrium, the correlations that remain will be predominantly negative. These negative correlations mean that selection to increase one character with respect to fitness will tend to decrease correlated characters with respect to fitness. As a result, genetic variation for fitness may be low, but genetic variation for its components can remain high. The major prediction from this theory is that populations that have adapted to a particular range of environments should exhibit negative correlations among characters affecting fitness, a result supported by empirical evidence (Rose 1984).

A population apparently adapted to one environment, upon exposure to a novel environment with a different selective regime, can face a formidable evolutionary challenge. Different selective pressures will favor different allelic combinations; the process of adaptation to new environmental characteristics should result in rapid changes in allele frequencies corresponding to the newly favored alleles. During this process, prior to selective equilibrium, genetic correlations between fitness characters should become more positive, and the additive genetic variance for these characters should increase (Service and Rose 1985, Holloway et al. 1990). The expected result is a rapid reorganization in the genetic architecture of life history, including a phase characterized by positive genetic correlations among life-history traits.

Empirical results generally appear to support the notion of rapid adaptation to novel environments. For example, Doyle and Hunte (1981a,b) found a substantially higher population growth rate in a laboratory environment in a domesticated population (25 generations in the laboratory) of the estuarine amphipod *Gammarus lawrencianus*, relative to a wild population. Lande (1983) concluded from a survey of the literature that major adaptive changes occur in domesticated and artificially disturbed populations due to mutations with large phenotypic effects.

The evidence for such effects comes primarily from resistance to toxins, pathogens, and predation. Service and Rose (1985) showed that rapid adaptation to a novel environment (as might result if wild fish are cultured in a hatchery) altered the genetic covariance structure of life history in *Drosophila melanogaster* transferred after 80 generations from an environment of banana-agar-corn syrup medium, 25°C, and constant light to one of Instant Drosophila Medium Blue medium, 15.5°C, and constant darkness. Holloway et al. (1990) showed similar results when they introduced rice weevil (*Sitophilus oryzae*) that had been cultured for over 50 generations on wheat (*Triticum aestivum*) and transferred to yellow split-pea (*Pisum sativum*). These results were consistent with the above predictions.

Several studies have failed to detect negative genetic correlations among life-history traits (e.g., Giesel and Zettler 1980; Giesel et al. 1982; Murphy et al. 1983; Bell 1984a,b). However, Rose (1984) and Service and Rose (1985) argued that these correlations were measured in populations that either 1) were partially inbred--i.e., exhibited some degree of inbreeding depression--which can bias patterns of genetic correlation (as in the case of the studies led by Giesel) or 2) had been subjected to the laboratory environment for only a few generations--i.e., were not near selective equilibrium (as in the case of the other studies).

On balance, experimental evidence supports a prominent role for antagonistic pleiotropy in life-history structure and the ability of rapid adaptation in novel environments to disrupt this structure. However, more work is needed on fishes and other organisms to confirm the generality of these results. Research on the genetic consequences of hatchery culture for salmonid life history is required to fully evaluate the genetic risks of captive broodstock programs for these fish.

The large apparent differences in rearing environment between traditional salmon hatcheries and natural rearing habitats suggest that the types of selection experienced by fish developing in these environments may differ substantially (Hynes et al. 1981, Doyle 1983). Waples (1991a) argued that sharp differences exist in mortality profiles between hatchery and natural salmon, and that these differences transcend metamorphosis (smoltification) and preclude almost any chance that genetic differentiation between these two groups can be avoided.

The empirical evidence for domestication in artificially propagated salmonids that are not subjected to artificial selection is equivocal; most of it is based on reduced performance of hatchery fish in the wild, including reduced survival (Schuck 1948, Reisenbichler and McIntyre 1977, Leider et al. 1990), reduced stamina (Green 1964, Leon 1986), altered behavior (Vincent 1960, Moyle 1969, Swain and Riddell 1990, Riddell and Swain 1991, Fleming and Gross 1992), and reduced reproductive success (Fleming and Gross 1993). Skaala et al. (1990, Table 1) summarized genetic changes connected with salmon culture, although their survey clearly included other genetic problems such as inbreeding and inbreeding depression.

Indirect evidence for domestication selection comes from changes in quantitative genetic parameters in artificial selection experiments that are difficult to account for on the basis of the applied selection differential alone. Gjedrem (1979) observed a larger response to selection for increased growth rate in Atlantic salmon than he could ascribe to the amount of artificial selection that he applied. Kinghorn (1988) presented evidence from farmed Norwegian Atlantic salmon compared to wild fish that was consistent with a response within four generations to domestication selection for growth. In an experiment to determine response to selection for high 8-month weight in coho salmon, Hershberger et al. (1990) detected a weight increase in unselected (control) populations reared initially in a hatchery and then transferred to marine netpens.

It should be recognized, however, that the first three conditions are not very restrictive and that the last condition is superfluous if no artificial selection is being practiced. The evolutionary genetic literature contains several examples of genetic change occurring over a few generations in quantitative traits with relatively low heritabilities (but potentially high additive genetic variances) that are difficult to attribute to genetic drift. The point is not that genetic change is difficult to produce, but rather that its direction and magnitude are difficult to predict (e.g., Sheridan 1988).

Table 1. Fitness consequences of crossbreeding between conspecific populations, exclusive of salmonids (updated from Endler 1977, Table 4.6). The symbols +, –, and = refer to the character's value in the crossbred offspring relative to the parental mean; a period (.) indicates no data. "Overall" refers to the general result of crossbreeding for the character across studies. See Endler (1977) for further notes and qualifications.

		Mean		Variance		
Character	Species	F_1	F_2	F_1	F_2	References
Viability	*Drosophila melanogaster*	+	–	–	+=	Wallace (1955)
		=	=	–	+	King (1955)
	Drosophila pseudoobscura	+	–	+=	=	Vetukhiv (1953, 1955)
		+	–=	.	.	Brncic (1954)
		+	–	.	.	Wallace and Vetukhiv (1955)
		.	.	.	+	Spassky et al. (1958)
		+=	–=	–=	+=	Vetukhiv and Beardmore (1959)
	Drosophila willistoni	+	–	+	+	Vetukhiv (1954)
		+	–	.	.	Wallace and Vetukhiv (1955)
	Drosophila paulistorum	+	–	+=	+	Vetukhiv (1954)
		+	–	.	.	Wallace and Vetukhiv (1955)
	Drosophila pavani	.	–	.	.	Brncic (1961)
	Drosophila persimilis	.	–	.	.	Spiess (1959)
	Drosophila prosaltans	.	–	.	.	Dobzhansky et al. (1959)
	Phyciodes tharos	=	–	+=	+	Oliver (1972)
	Boloria toddi	+=	–	.	.	Oliver (1972)
	Cisseps fulvicollis	–	–	+	+	Oliver (1972)
	Hyperia postica	–	–	.	.	Blickenstaff (1965)
	Rana pipiens	–	.	.	.	Ruibal (1955), Fowler (1964)
		–=	.	.	.	Moore (1950, 1967), Volpe (1957), Cuellar (1971)
Viability	*Triturus critatus*	+	–	.	.	Callan and Spurway (1951), Spurway (1953, 1954)
	Streptanthus glandulosus	+	.	.	.	Kruckeberg (1957)
	Mimulus luteus	–=	.	.	.	Hughes and Vickery (1974)
	Mimulus tigrinus	=	.	.	.	Hughes and Vickery (1974)
	Mimulus cupreus	–	.	.	.	Hughes and Vickery (1974)
	Drosophila mojavensis	=	=	.	.	Etges (1989)
	Delphinium nelsoni	+=	.	.	.	Price and Waser (1979)
	Chamaecrista fasciculata	+=	.	.	.	Fenster (1991)
	Amphicarpaea bracteata	–	.	.	.	Parker (1992)
	Tigriopus californicus	.	–	.	.	Burton (1986, 1990)
	Capra ibex subspp.	–	.	.	.	Grieg (1979)
	Micropterus salmoides	=	.	.	.	Philipp and Whitt (1991)
	Ipomopsis aggregata	–=	.	.	.	Waser and Price (1989)
	OVERALL	+=	–	+=	+	

Table 1. Continued.

Character	Species	Mean F_1	Mean F_2	Variance F_1	Variance F_2	References
Fecundity	*Drosophila pseudoobscura*	+	–	.	.	Wallace and Vetukhiv (1955)
		+=	–	+=	+=	Vetukhiv (1956)
		+=	–=	=	=	Vetukhiv and Beardmore (1959)
	Drosophila willistoni	+	–	.	.	Wallace and Vetukhiv (1955)
	Drosophila paulistorum	+	–	.	.	Wallace and Vetukhiv (1955)
	Phyciodes tharos	+	.	+	.	Oliver (1972)
	Hyperia postica	+	–	.	.	Blickenstaff (1965)
	Zea mays	+	=	.	.	Moll et al. (1965)
	Delphinium nelsoni	+=	.	.	.	Price and Waser (1979)
	Chamaecrista fasciculata	+=	.	.	.	Fenster (1991)
	Tigriopus californicus	+=	.	.	.	Brown (1991)
	Polemonium visosum	=	.	.	.	Newport (1989)
Fecundity	*Ipomopsis aggregata*	+=	.	.	.	Waser and Price (1989)
	OVERALL	+=	–=	+=	+=	
Fertility	*Phyciodes tharos*	–	–	+	+	Oliver (1972)
	Boloria toddi	–=	–=	.	.	Oliver (1972)
	Cisseps fulvicollis	–	–	+=	+	Oliver (1972)
	Triturus cristatus	–	–	.	.	Callan and Spurway (1951), Spurway (1953, 1954)
	Apodemus sylvaticus	–=	–=	.	.	Jewell and Fullagar (1965)
	Hyperia postica	–	–	.	.	Blickenstaff (1965)
	Mimulus guttatus	–	.	.	.	Vickery (1967)
	Streptanthus glandulosus	–	.	.	.	Kruckeberg1 (1957)
	OVERALL	–	–	+=	+	
Growth or body size	*Drosophila pseudoobscura*	=	–	=	+	Anderson (1968)
	Drosophila subobscura	–=	–=	.	.	McFarquhar and Robertson (1963)
	Micropterus salmoides	=	.	.	.	Philipp and Whitt (1991)
	OVERALL	=	–=	.	.	
Adult longevity	*Drosophila pseudoobscura*	+	–	+=	+	Vetukhiv (1957)
Development time	*Drosophila subobscura*	–=	=	.	.	McFarquhar and Robertson (1963)
	Phyciodes tharos	+=	.	+	.	Oliver (1972)
	Cisseps fulvicollis	+	.	=	.	Oliver (1972)
	Rana pipiens	–	.	.	.	Moore (1946)
	Drosophila mojavensis	=	=	.	.	Etges (1989)
	Tigriopus californicus	–=	+	=	+	Burton (1987, 1990)
	Zea mays	–	–=	.	.	Moll et al. (1965)
	OVERALL	=	=	+=	+	

Table 1. Continued.

Character	Species	Mean F_1	Mean F_2	Variance F_1	Variance F_2	References
Diapause	*Wyeomyia smithii*	–=	–	–	+=	Hard et al. (1992b, 1993)
Sex ratio	*Phyciodes tharos*	+	.	.	.	Oliver (1972)
	Cisseps fulvicollis	+	.	.	.	Oliver (1972)
	Hyperia postica	+	+	.	.	Blickenstaff (1965)
	OVERALL	+	.	.	.	
Pest resistance	*Populus* spp.[a]	–	.	.	.	Whitham (1989)
Relative fitness	*Geospiza* spp.[a]	+=	.	.	.	Grant and Grant (1992)
Morphometry	*Tinca tinca*					
Fluctuating assymetry	*Enneacanthus* spp.[a]	+=	.	.	.	Graham and Felley (1985)

[a] Naturally occurring interspecific hybrids

Population differentiation and outbreeding depression. The genetic mechanisms and consequences of population differentiation are closely tied to the issue of genetic change occurring in captive broodstock programs. Population differentiation may occur through random (mutation, genetic drift) or deterministic (selection, migration) means. Its consequences are important because they determine whether a captive broodstock program used to rebuild a declining wild population has maintained the genetic integrity of that population *in traits that are important to local adaptation* (i.e., life-history traits). Thus, change in the life-history structure of a supplemented population is a direct measure of the genetic success of captive broodstock or other supplementation programs. Assessment of adaptive differentiation requires quantitative genetic methods: controlled breeding, phenotypic evaluation, and tests of quantitative genetic models. Other genetic techniques simply do not address adaptation and life-history variation directly.

Outbreeding depression is the reduction in fitness that results from mating between unrelated or distantly related individuals. Outbreeding depression may result from loss of local adaptation (Templeton 1986) or from the breakup of favorable gene combinations (Dobzhansky 1948). Therefore, like inbreeding depression, it often results from nonadditive expression of constituent genes (Lynch 1991).

Generally, outbreeding depression that results from the breakup of "coadapted gene complexes" is expected to manifest itself after segregation (i.e., in the F_2 or later generations) through reduced trait means and increased trait variances with respect to fitness. When two interbreeding populations are so distantly related that their genomes have diverged considerably,

the resulting genic interactions may be so strong that outbreeding depression is expressed in the F_1. However, outbreeding depression may also be expressed in the F_1 if hybrid offspring are poorly adapted to the habitat they occupy, regardless of the mode of gene interaction.

Although current interest in outbreeding depression is high, its extent and consequences in natural salmon populations or between hatchery and wild populations is unknown. Evidence exists for outbreeding depression in other organisms, but the quality of this evidence varies and it relies largely on a few, primarily invertebrate, species. The contentious nature of this issue for salmon supplementation warrants a detailed examination of the evidence. This evidence is summarized in Tables 1 and 2.

The results surveyed in Table 1 indicate a frequent tendency for outbreeding to yield heterosis with respect to correlates of fitness in the F_1, followed by reduced fitness in the F_2. Where variances in these hybrids have been examined, the trend is for increased variance in both generations. However, phenotypic variance is expected to increase in the F_2 due to segregation. Thus, it is not clear from these results to what extent the observed increases in F_2 variance exceed the amount resulting from segregation (potentially by disruption of coadapted gene complexes).

One way to test observed versus expected increases in trait variances in second-generation hybrids (F_2 and first backcrosses) is by comparing observed means and variances with the expectations of an additive genetic model (Cockerham 1986). Hard et al. (1992b, 1993) tested the means and variances of six geographic populations of the pitcher-plant mosquito (*Wyeomyia smithii*) and their F_1, F_2, and first-generation backcrosses against the additive genetic expectation to show that the lower F_1 variances and higher F_2 variances they observed cannot be attributed to additive effects alone. Large increases in F_1 or F_2 variance with respect to fitness may be consistent with outbreeding depression, but they do not necessarily reflect it.

The evolutionary consequences of outbreeding among salmonid populations are not clear. Virtually all the studies surveyed in Table 2 have examined only first-generation hybrids, and, consequently, most of these studies were not designed to detect outbreeding depression. Nevertheless, relatively few studies have found evidence for heterosis in first-generation hybrids, suggesting little directional dominance (or epistasis involving directional dominance) for fitness among populations. Some crosses have shown reductions in fitness in the F_1, which could portend severe outbreeding depression in subsequent generations through the breakup of coadapted gene complexes if epistasis has contributed to population divergence.

In the only empirical study designed specifically to detect outbreeding depression in salmonids beyond first-generation hybrids, Gharrett and Smoker (1991) examined marine survival, return date, body size, and bilateral assymetry in two generations of crosses between even- and odd-year populations of Auke Creek (Alaska) pink salmon. These workers observed substantially lower survival and increased assymetry in the F_2 but not the F_1 hybrids, a result consistent with outbreeding depression by breakdown of coadapted genes. However, it is important to recognize that even- and odd-year populations of pink salmon from the same stream may have been

reproductively isolated for potentially thousands of generations. Indeed, such populations are genetically more distinct from each other than from populations spawning in the same year in different streams (Aspinwall 1974, Beacham et al. 1988, Shaklee et al. 1991). Consequently, the results found by Gharrett and Smoker (1991) may not be representative of those expected between hatchery and natural populations with a greater natural opportunity for gene flow.

Table 2. Fitness consequences of crossbreeding between conspecific salmonid populations. The symbols are as in Table 1.

		Mean		Variance		
Character	Species	F_1	F_2	F_1	F_2	References
Viability	*Oncorhynchus gorbuscha*	=	–	.	.	Gharrett and Smoker (1991)
	Oncorhynchus mykiss	–	.	.	.	Reisenbichler and McIntyre (1977)
		=+	.	.	.	Ayles and Baker (1983)
		+=	.	.	.	Hörstgen–Schwark et al. (1986)
	Oncorhynchus nerka	=	.	.	.	Wood and Foote (1990)
	Oncorhynchus tshawytscha	=	.	.	.	Cheng et al. (1987)
	Salvelinus fontinalis	–=	.	.	.	Mason et al. (1967)
		+	.	.	.	Webster and Flick (1981)
		=	.	.	.	Fraser (1989)
		=+	.	.	.	Lachance and Magnan (1990a)
	Salmo salar	=	.	.	.	Gjerde and Refstie (1984)
	Salmo spp.	–=+	.	.	.	McGowan and Davidson (1992)
	OVERALL	=	.	.	.	
Seawater adaptability	*Oncorhynchus nerka*	=	.	.	.	Foote et al. (1992)
	Oncorhynchus tshawytscha	–	.	=	.	Clarke et al. (1992)
	OVERALL	–=	.	=	.	
Growth rate	Oncorhynchus mykiss	=	.	.	.	Reisenbichler and McIntyre (1977)
	Oncorhynchus gorbuscha	+=	=	+	.	Gharrett and Smoker (1991)
		=+	.	.	.	Ayles and Baker (1983)
		–=+	.	.	.	Ferguson et al. (1985)
		=	.	.	.	Hörstgen–Schwark et al. (1986)
Growth rate	*Oncorhynchus mykiss*	=	.	.	.	Wangila and Dick (1987)
		–=	.	.	.	Johnsson et al. (1993)
	Oncorhynchus tshawytscha	–=+	.	.	.	Cheng et al. (1987)
		+=	.	=	.	Clarke et al. (1992)
	Salvelinus fontinalis	=+	.	.	.	Mason et al. (1967)
		=+	.	.	.	Keller and Plosila (1981)
		+	.	.	.	Webster and Flick (1981)
		=	.	.	.	Fraser (1989)
		=+	.	.	.	Lachance and Magnan (1990a)
	Oncorhynchus nerka	=	.	+	.	Wood and Foote (1990)
	Salmo trutta	–	.	.	.	Maisse et al. (1983)
	OVERALL	=	.	+=	.	

Table 2. Continued.

Character	Species	Mean F_1	Mean F_2	Variance F_1	Variance F_2	References
Catchability	*Oncorhynchus mykiss*	=+	.	.	.	Pawson and Purdom (1987)
	Salvelinus fontinalis	+	.	.	.	Keller and Plosila (1981)
Hatching time	*Salmo clarki*	–=+	.	.	.	Ferguson et al. (1988)
Parasite resistance	*Oncorhynchus kisutch*	–=+	.	.	.	Hemmingsen et al. (1986)
Homing rate	*Oncorhynchus gorbuscha*	+	.	.	.	Bams (1976)
Maturation/ spawn timing/ GST[a]	*Oncorhynchus mykiss*	=	.	.	.	Marrocco (1982)
	Salmo salar	=	.	.	.	Sutterlin and MacLean (1984)
	Salvelinus fontinalis	=+	.	.	.	Lachance and Magnan (1990b)
Rheotaxis	*Oncorhynchus mykiss*	=	.	.	.	Kelso and Northcote (1981)
Meristics/ fluctuating assymetry	*Oncorhynchus gorbuscha*	=	–	.	.	Gharrett and Smoker (1991)
	Oncorhynchus mykiss	+=	.	.	.	Ferguson and Danzmann (1987)
	Salvelinus spp.[b]	+	.	.	.	Leary et al. (1985)
	Oncorhynchus spp.[b]	+	.	.	.	Leary et al. (1985)
	Oncorhynchus mykiss	–	.	.	.	Ferguson (1986)
	Salmo clarki	+	.	.	.	Ferguson et al. (1988)
		=	.	.	.	Forbes and Allendorf (1991)
	Salmo trutta	=	.	.	.	Ielli and Duchi (1990)
	OVERALL	+=	.	.	.	

[a] GST = Gonadosomatic index

[b] Naturally occurring interspecific hybrids

In the following section, we outline prominent quantitative genetic risks associated with captive broodstock programs, discuss the importance of genetic monitoring for quantitative characters, and suggest some basic approaches to address these issues.

A Quantitative Genetic Approach to Salmon Captive Broodstock Programs

Busack (1990) and Riggs (1990) identified four genetic risks posed by artificial propagation: extinction, reduction in variability within populations, reduction in variability among populations (loss of population identity), and change through domestication. One problem with this framework is that domestication has had different interpretations (RASP 1992, Kapuscinski and Miller 1993, Kapuscinski et al. 1993, Lichatowich and Watson 1993). For the purposes of captive broodstock programs, we suggest three categories of genetic risk: reduction of genetic variability within a population, loss of population identity, and genetic change. We consider extinction to be the complete loss of population identity. The loss of population identity can occur through either gene flow (introgression) or genetic change (through selection or genetic drift). The former mechanism should occur only rarely once a captive broodstock program has been initiated. Genetic change is likely to be of greater concern.

Genetic concerns can surface at any of several points in a captive breeding program. These concerns are not the exclusive domain of such programs, as they are also relevant to more traditional forms of artificial propagation, but they can become even more prominent when fish are cultured for their entire life cycle. The major "control" points in a captive broodstock program that determine the quantitative genetic consequences of supplementing a natural population are outlined in Table 3. These control points indicate the ample opportunity that exists for genetic risks to arise in captive broodstock programs.

The relative importance of these risks depends on the program's scope. For example, if the natural population is very small and all individuals are used for captive broodstock (a situation that already exists for endangered Snake River sockeye salmon; Waples et al. 1991c), then loss of genetic variability within the population (and its enhanced risk of extinction) becomes the most prominent genetic concern. Other risks, such as genetic change, are relevant in that they affect the success of reintroduction, but the highest priority should be given to protection against catastrophic loss or excessive mortality (Hard et al. 1992a).

In many cases, however, not all individuals are used to establish captive broodstock; some are allowed to reproduce in the wild. In this case the risk of genetic change becomes a greater concern. If care is taken to select wild broodstock only from the population to be supplemented, the loss of genetic variability among populations can effectively be ignored. Attention should then be focused on genetic change and the loss of genetic variability within the population.

Quantitative Genetic Monitoring of Captive Broodstock Programs

The genetic risks of captive broodstock programs for natural populations indicate that genetic monitoring should be an integral part of a well-designed program. A properly implemented monitoring scheme should allow for detection of genetic problems before they become large enough to pose serious risk. Few genetic monitoring plans currently exist for

Table 3. Major control points and possible genetic consequences of activities associated with salmonid captive broodstock programs.

Control point	Genetic consequences
Selection of broodstock	Founder event (genetic change)
Collection of broodstock	Founder event (genetic change) Bottleneck (reduced genetic variation)
Mating of broodstock	Genetic drift (reduced genetic variation) Inbreeding depression (reduced fitness) Domestication selection (genetic change)
Rearing of first-generation descendants to maturity in captivity	Bottleneck/genetic drift (reduced genetic variation) Domestication selection (genetic change)
Sampling of descendants for broodstock	Bottleneck/genetic drift (reduced genetic variation) Inbreeding depression (reduced fitness) Founder event/domestication selection (genetic change)
Rearing of second-generation descendants in captivity	Bottleneck/genetic drift (reduced genetic variation) Domestication selection (genetic change)
Release of second-generation descendants to the wild	Bottleneck (reduced genetic variation) Domestication selection (genetic change) Genetic introgression (genetic change)

Pacific salmon (e.g., Waples et al. 1993) and, to our knowledge, none of these monitor quantitative characters. It is important to consider monitoring quantitative characters to fully evaluate the effects of captive propagation on adaptation in the wild.

Monitoring quantitative characters involves one consideration that is not at issue when monitoring single-locus characters: discrimination between environmental and genetic sources of observed variation. Determining the genetic basis of phenotypic variation is the raison d'être of quantitative genetics. Two main issues should be considered in monitoring quantitative genetic variation: the first is genetic, the second statistical (Hard, in press).

The genetic problem arises from the fact that observed variation in quantitative characters has an environmental as well as a genetic component. A portion of this variation often reflects

interaction between these components, so that the phenotypic variation expressed by particular genotypes depends on the environment in which they exist. For captive broodstock programs, the primary significance of environmental influence on phenotypic expression is that phenotypic change during the course of the program is likely to reflect both genetic and environmental change. Determining the amount of genetic change associated with an observed phenotypic change is essential to evaluating the genetic consequences of captive propagation for the population, and this determination requires quantitative genetic methods.

However, to achieve this objective it is probably not necessary to estimate genetic parameters such as heritability with the precision usually employed in most quantitative genetic research or in selective breeding programs. Because these estimates depend on the population's environment as well as its gene frequencies, estimates for captive populations are often higher than corresponding estimates for the same traits in populations developing under naturally more variable conditions (Prout 1958, Coyne and Beecham 1987). Furthermore, the reliability of such estimates in predicting phenotypic response to selection has been called into question (Sheridan 1988).

Thus, it may be sufficient to determine only whether additive genetic variance exists for a character, as this parameter is what determines a population's potential for genetic change (Hard, in press). If significant additive genetic variance exists for a quantitative character in a captively propagated population, and if appreciable phenotypic change has occurred during propagation, concern should arise that genetic change has resulted during the captive broodstock program.

The statistical problem arises directly from the objective to minimize the genetic and phenotypic differentiation of captive and natural fish during supplementation. This objective generates a different approach to hypothesis testing than other goals might. For a captive broodstock program with this objective, an appropriate null hypothesis is that captive and natural fish do not differ for the trait in question.

There are two statistical errors that can result in testing this hypothesis, and only one of these has been widely appreciated by fisheries biologists. This error, known as type I error (Winer 1971, Sokal and Rohlf 1981), is in the present context the probability of concluding that these two groups differ when in fact they do not. Fortunately, the level of type I error (designated α), can be controlled a priori by the investigator by setting the significance level for the test.

The other error, known as type II error (Dixon and Massey 1957, Winer 1971), is the probability of concluding that these groups do not differ when in fact they do. Unfortunately, type II error cannot be controlled by the investigator except indirectly through sample size and "effect size" (the direction and magnitude of the effect the investigator wishes to detect), and it tends to rise with more stringent control of type I error (Cohen 1988).

The consequences of type I error have received a great deal of attention by empirical biologists because it has generally been presumed that this error is the most serious of the two

types (Cohen 1988; Peterman 1990). However, in many studies in environmental toxicology, industrial and chemical safety, and conservation biology, type II error may be more serious than type I error. In general, the risks of these errors should be weighed in any evaluation to minimize unanticipated problems and guide experimental design.

A comprehensive treatment of the statistical risks in hypothesis testing and an explanation of experimental power is beyond the scope of this report. Peterman (1990) gives an exemplary introduction to this issue in fisheries applications; statistical texts by Dixon and Massey (1957) and Sokal and Rohlf (1981) provide additional detail for the interested reader.

For the purposes of captive broodstock programs, the following point should be remembered: The genetic consequences of concluding that appreciable genetic change has not occurred during protective culture, when such change has actually occurred (type II error), are arguably more serious than the consequences of falsely concluding that such genetic change has occurred (type I error).

The former conclusion has irreversible biological consequences; the second has serious (but reversible), primarily economic, consequences. The risk of type II error is inversely related to the intensity of genetic monitoring (i.e., sample size) and the direction and magnitude of genetic divergence considered acceptable (the effect size). These risks, in turn, depend on character variability and how divergence is measured. Hard (in press) discusses this issue in further detail and illustrates difficulties in avoiding type II error that could arise during genetic monitoring of quantitative characters.

The success of supplementation efforts that involve captive broodstock programs can be enhanced if quantitative characters are monitored because these characters are sensitive to changes in environment, and consequently, the potential for domestication. The desirable features of such programs include 1) quantitative genetic monitoring as an integral part of the supplementation process, involving differential marking of released individuals and adequate sampling of life-history characters on captive and natural individuals; 2) identifying the amount of genetic differentiation allowed to occur in the characters that are assessed, this amount to be determined by consideration of information on character variation; and 3) determining appropriate responses to genetic problems that surface in the program.

Future Research Priorities

As the discussion above indicates, there are several potential genetic consequences of salmon captive broodstock programs, and little research has been done on any of them. A considerable amount of work, firmly grounded in quantitative genetics, will be required to determine the likelihood and extent of these consequences, which can be grouped into three main categories: 1) loss of genetic variability within a salmon population resulting from the establishment of a captive broodstock program, and the inbreeding depression that may result

from small population size and patterns of mating; 2) genetic change that may result from adaptation (domestication) in captive fish to the protective culture environment; and 3) genetic divergence of the captive fish from their natural source population and the subsequent consequences of genetic interactions between these groups.

The experimental requirements of studies designed to address these issues are formidable. These studies require large numbers of available spawners and at least one, and generally two or more, fish generations to address the experimental objectives. Previous studies have attempted to address three main categories of quantitative genetic risk in captive broodstock programs:

1) To relate the degree of inbreeding to the degree of inbreeding depression by determining the extent of inbreeding depression that results from various levels of inbreeding, and compare the levels of inbreeding incurred by different mating schemes. The simplest approach is to subject a population to several generations of full-sib mating (Kincaid 1976, 1983) and compare its response to that of other mating techniques.

2) To determine whether natural selection ("domestication") that acts on captive populations during protective culture differs qualitatively from selection that acts on salmon in nature. The approach used could examine variation in family size as an indicator of domestication selection, or could use the relative inter-generational variation in a trait(s) between a selected and control (i.e., unselected) captive population to characterize the direction and general magnitude of natural selection acting on that trait during protective culture (Kinghorn 1988, Hershberger et al. 1990).

3) To determine the genetic consequences of interbreeding between captive broodstock and natural fish. The approach involves making crosses between cultured and natural fish, establishing their first- and second-generation hybrids, measuring the means and sampling variances of life-history traits in each derivative line, and testing the goodness of fit of the variation in these traits among cross derivatives to various simple models of gene expression.

Such an approach can allow one to determine not only whether adaptive divergence between captive and natural fish has occurred during a captive broodstock program, but also whether the most commonly accepted mechanism for this "outbreeding" depression (i.e., the breakup of "coadapted gene complexes"; Dobzhansky 1948) is likely to be responsible for the depression. At a 1994 American Fisheries Society Symposium on the "Uses and Effects of Cultured Fishes in Aquatic Ecosystems" in Albuquerque, New Mexico, outbreeding depression was identified as a primary concern among fishery geneticists.

Conclusions

Quantitative genetics has applications to a wide variety of problems. However, most fishery work published to date has dealt with estimating levels of genetic variation within and among populations. This objective is important, but it raises questions about what maintains or erodes this variation. Attempts to address this issue are not only of basic evolutionary interest, but can contribute to informed management and conservation.

The majority of research in quantitative genetics has emphasized determining the genetic and environmental components of variation in traits important to aquacultural production. These traits include growth and size, reproduction (especially maturation and egg production), disease resistance, and body composition and flesh quality. In general, the genetic basis for most of these characters is often large enough that a reasonable response to selection can be realized, but individual responses depend strongly on the stock and environment involved.

A number of studies have examined the quantitative genetic basis of life-history characters and their covariation with morphological and other characters. Life-history characters examined include incubation performance, early development traits, survival, age at maturity, and behavior. Four general observations emerge from these studies.

First, while life-history characters tend to exhibit a wide range of genetic underpinnings, the reliability of genetic parameter estimates such as heritabilities and genetic correlations is questionable because of inadequate experimental designs or sampling techniques. Second, where analyses have been conducted over different environmental conditions, there often appears to be substantial interaction between genetic and environmental effects on the phenotype. Third, some traits appear to evolve in close association with other traits, often precluding selection on them independently. Finally, genetic relationships among traits are often (but not always) qualitatively similar to their phenotypic relationships.

The genetic basis of adaptation has received little attention in fishery quantitative genetics. Topics in particular need of attention include inbreeding depression, selection, and population differentiation and outbreeding depression. Until these topics receive empirical attention, fishery geneticists will find it difficult to predict the evolutionary consequences of small population size, intensive hatchery culture, or interbreeding between hatchery and wild fish. Consequently, we believe it is crucial to integrate investigation of these topics into genetic conservation research.

Evidence is growing that quantitative traits that contribute to adaptation in salmonids can change under human influences, such as harvest, habitat alteration, and artificial propagation (as in conventional hatcheries or in captive broodstock programs). Many authors have recently pointed out potential genetic problems that can arise during artificial propagation as a result of these influences. since the immediate genetic concerns for conservation may differ substantively from those for enhancement and mitigation. For example, minimizing genetic differentiation between hatchery fish and the natural fish they are intended to supplement may be as important a concern

as maintaining genetic variability within the hatchery population. Lack of guidance on how to detect, monitor, and respond to the effects of selection in hatchery fish undoubtedly has resulted from uncertainties about how adaptation operates in novel environments. Although these and other quantitative genetic issues should be addressed empirically before artificial propagation is applied to salmon conservation on a large scale, there are several reasons why they have not yet been adequately addressed. Two of these issues stand out as particularly significant.

First, it is only recently that possible adverse genetic effects of hatchery culture have been widely appreciated. Supplementation of wild salmon populations and the concept of captive broodstocks are recent developments associated with acknowledged declines in wild populations. Traditional hatchery programs were developed primarily to enhance fisheries or to mitigate for lost abundance of naturally reproducing fish due to harvest or habitat loss or degradation. In such programs, issues such as inbreeding depression, selection, and the consequences of hatchery-wild stock interactions have not received the attention they deserve because hatchery production has generally been considered to be independent of wild production (Larkin 1974, Riddell 1993, Lichatowich and Watson 1993) or even a replacement for it (e.g., Netboy 1974).

Assessment of stock performance has been gauged in terms of fishery contributions and adult returns to the hatchery, not in terms of reproductive success and genetic and phenotypic change. In addition, transfers of fish within and among major river basins removed many constraints on broodstock development. Growing evidence that hatchery production may have adverse effects on wild production has forced a reevaluation of these attitudes (Hindar et al. 1991, Riddell 1991, Waples 1991a).

Second, most quantitative genetic issues are difficult to address experimentally with salmon. Most empirical work done on inbreeding depression, selection, and population differentiation has involved invertebrates and plants that have short generation times and can be cultured in large numbers of closely related groups. Pacific salmon satisfy neither of these criteria very well. Few salmon biologists have adequate training in quantitative genetics, and this limits their ability to design breeding programs that evaluate the evolutionary basis of phenotypic variation. These factors have proven to be a major obstacle to research on the quantitative genetics of Pacific salmon.

The use of artificial propagation techniques such as captive broodstock programs to help reverse declines in wild salmon production should be accompanied by aggressive research to understand the genetic basis of population differentiation and adaptation. This research must entail quantitative genetic approaches. These approaches are often costly, logistically difficult to implement, and protracted, but they are necessary to directly address genetic issues of primary importance to fishery managers and salmon producers.

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