Fisheries and Oceans
Canada

Commissioner

Aquaculture
Development

Pêches et Océans
Canada

Commissaire

Développement
de l'aquaculture

Potential Genetic Interaction Between Wild and Farm Salmon of the Same Species

Prepared for:

The Office of the Commissioner for Aquaculture Development
Fisheries and Oceans, Canada

By: R.G. Peterson, Ph.D.

September 1999

Suite 1610
344 Slater
Ottawa ON K1A 0E6

Canada

Potential Genetic Interaction Between Wild and Farm Salmon of the Same Species

Prepared for:

The Office of the Commissioner for Aquaculture Development
Fisheries and Oceans, Canada
344 Slater St., Suite 1610
Ottawa, Ontario K1A 0E6

By:

R.G. Peterson, Ph.D.
4291 West 16th Ave.
Vancouver, BC V6R 3E5

September 1999

Table of Contents

Summary

The impact of genetic intrusion of escaped farm fish on wild salmon stocks is complex and depends upon the fitness of the wild stock, the genetic diversity of the farm stock and the number of genes that are added to the wild genepool.

Wild and farm stocks are expected to have rather different genepools because wild stocks are subjected to natural selection, migrations from other stocks and random drift. Farm stocks came from one or more wild populations and, in a sense, start from the same genetic base as wild fish. However, they have been subjected to selective breeding programs (SBP) and automatic selection for domestic fitness.

Perhaps one misconception colours the discussion of genetic interaction between wild and farm fish more than any other – the idea that natural selection has transformed wild salmon stocks into the optimum "genetic solution" for their ecological niche.

This idea is, in general, not true. Natural selection, in a stable environment, will increase fitness to an equilibrium point. But, this local maximum is only one of many possible solutions and is not likely the "best" solution.

The key to maintaining a healthy wild stock is to allow or provide for gene flow between stocks. This will maximize the genetic diversity of the stocks and in turn conserve the genetic diversity of populations and ultimately the species.

Natural selection acts on fitness, which is the relative contribution of a spawning fish to the next generation. Fitness of a stock is a measure of its ability to compete for the resources in its ecosystem. A "healthy" wild stock, with respect to fitness, expands to the limits of the ecological niche and would not really be stable but would increase and decrease in size, depending on the resources in the environment. Such a stock will likely have adequate additive genetic variance to respond to changes in the environment. Stocks with little or no additive genetic variation for fitness are at risk of being replaced or eliminated since natural selection can not adapt these stocks to even small changes in their ecological niche.

Genetic diversity of wild stocks is normally maintained by migrations of fish between populations.

Natural selection and random drift in a reproductively isolated stock leads to loss of genetic variation for fitness. When this variance is exhausted, natural selection becomes totally ineffective. Small changes in the environment may easily cause the population to become extinct. Isolation of salmon stocks should be avoided. Obviously, migration between related stocks is less efficient in transferring "new" fitness genes than exchanges between unrelated stocks.

Small escapements of farm fish into large healthy wild stocks are expected to have a small negative effect on fitness, in the short-term. However, these intrusions will likely have long-term benefits.

Genetic variation for fitness introduced by farm fish will allow natural selection to increase the fitness of the local wild stock. Large intrusions of farm genes into wild genepools are expected to cause severe declines in fitness in the short-term. Recovery is likely, but several generations would be required and the stock may not survive the initial flood. These statements follow from considering the genetic nature of fitness and how traits change under natural selection in wild populations and under SBP in farm broodstocks.

Farm stocks are expected to conserve most of the genetic variance for fitness that they obtained from their founding wild stock(s).

Farm fish and the initial crosses between farm and wild fish are not expected to be as fit as fish from an adjacent wild stock. This is because the balance in expression of fitness sub-traits is altered by the SBP. However, since fitness sub-traits are complex polygenic systems they will retain most of the genetic diversity from the founding wild stock(s). The usual situation is that founding and adjacent wild stocks are not related. This means the farm fish introduce “new” genetic variation for fitness to the wild stock.

Farm stocks were not created equal with respect to fitness.

Farm stocks created from a single wild stock will have far less genetic variation than a synthetic that was created from several unrelated wild stocks. Many of the fitness loci of individual stocks will be fixed (homozygous) because of natural selection and reproductive isolation. For this reason stocks created from a single wild stock will have less genetic variation for fitness than stocks derived from a wider genetic base. A diverse synthetic farm stock is likely a better source of fitness variance than most wild stocks. However, a synthetic created from many wild stocks and not subjected to SBP would be the best medium to introduce “new” fitness variation into a wild population.

This paper is not a recommendation for regular releases of farm fish into wild populations.

The objective was to focus on the real genetic issues involved in preserving genetic diversity in wild salmon and on this basis to assess the likely impact of farm salmon. Small escapements of farm fish do not pose a threat to healthy wild stocks. “Small” and “large” are vague terms and defining these with some confidence will require extensive and well-designed field research and simulation studies.

1. Introduction

Escaped farm fish have been found among wild stocks in areas close to the farm of origin. Given the right set of conditions, these fish will mate with other farm or wild fish and contribute to the wild genepool. The question that concerns all stakeholders interested in wild fish populations is ***- Does the incursion of farm fish into a wild stock threaten the viability of the wild stock, or cause a loss of genetic diversity?***

This paper will only deal with the genetic aspects of this question, which is limited to the interaction between wild and farm fish of the same species.

Does the incursion impair the wild stocks' ability to compete in its ecological niche or its ability to change as the niche evolves?

Does the incursion threaten the genetic diversity of the stock and ultimately the species? This is a much broader and more important question.

Farm stocks of salmon originate from one or more wild stocks so they will initially have the same genetic adaptability for the environment as their founding wild stock. However, the farm stocks have been under artificial selection for traits of economic importance and automatic selection for domestication for several generations. How will this selection effect the original fitness of the stock? Will the farm stock retain genetic diversity from its founder that might be useful for the wild genepool?

Peterson (1993) addressed these issues in a popular article. The study indicated that intrusion of farm genes into a wild genepool initially results in a reduction of the wild stocks' ability to compete but after several generations of natural selection the population returns to its original level or above.

How will the wild stock respond to natural selection with and without the addition of farm fish genes to the genepool? The genetic impact of escaped farm salmon on wild stocks is a complex issue and depends on the "genetics" of the wild stock and of the invading farm fish. To understand these issues requires some knowledge of natural selection, fitness and genetic variance. These are covered in the first section of the paper.

There is a large volume of literature on natural selection and how populations change both in the short-term and on an evolutionary scale. Falconer's (1989) text on quantitative genetics gives a readable and authoritative discussion of fitness as a quantitative trait and natural selection, in that context, and it is the primary reference used in this paper. Two popular books by Richard Dawkins (1986, 1996) provide interesting and readable discussions on these topics from the perspective of an evolutionary biologist. Many of these ideas on fitness and natural selection are, of course, in earlier scientific literature such as the treatise by Sewall Wright (1977).

2. Theoretical Background on Issues Relevant to the Interaction Between Wild and Farm Salmon

2.1. Fitness

Fitness of an individual is the contribution of genes it makes to the next generation, measured as the number of progeny surviving to maturity. Fitness is the end product of all of the animal's developmental and physiological functions. The differences between individuals can be seen, in theory, as a series of traits that quantify the results of these functions. These sub-traits of fitness are, in general, quantitative or metric traits, which means they are controlled by complex genetic systems with multiple loci and perhaps multiple alleles.

Complex feedback and interaction with the external environment (rearing resources, predation, etc.) and the "genetic environment" also influence the expression of these polygenic traits. The idea of a genetic environment is that individual gene function in a complex organism is subject to feedback from and interaction with other genes. In addition, the external environment plays a major role in defining fitness since it sets the criteria by which fitness is judged.

Fitness can be thought of as an index of the sub-traits. In this context it is a holistic single score that ranks animals based on the combined value of all the sub-traits weighed by their importance to overall fitness. For an individual fish, high merit for one sub-trait will compensate for lower merit in another. A simple example will demonstrate this point – a fish with above average number of eggs but egg quality below average can have the same fitness as another fish with the reverse scenario, low number and high quality of eggs. This means that there are many different genotypes that give the same index for fitness.

Two fish with the same expression of a fitness sub-trait (e.g., number of eggs) likely have different sets of genes controlling this trait. This is because these traits are controlled by polygenic systems, which will be discussed below.

There are many genetic solutions to the problem of fitness, posed by a given environmental situation. This is due to the combination of genetic diversity within polygenic traits and the diversity of expression for the fitness sub-traits that lead to a single value for fitness.

2.2. Genetic Variance

"The genetics of a metric character centers round the study of its variation, for it is in terms of variation that the primary genetic questions are formulated." (Falconer, p125).

The variance of fitness can, in theory, be partitioned into components based on causal effect. For our discussions, the partitions of interest are genetic and environmental causes and the genetic variance is further partitioned into additive and non-additive genetic variance. The magnitude of these components measures the genetic properties of a population.

Genetic variances are dependent on both gene action and gene frequency. The environmental variation is due to all non-genetic affects on the trait being studied. ***Additive genetic variance*** arises from the additive effects of genes that contribute to a trait. The definition of additive effect of a gene is a bit complicated (see Falconer), but is essentially, the average effect of the parental gene in the progeny population. ***Non-additive genetic variance*** comes from dominance and epistatic gene action.

The relative ***magnitude of the additive genetic variance determines the similarity between relatives*** in expression of the trait of interest (fitness). This, of course, is the ***key to selection***. The greater the similarity between parent and offspring the greater the opportunity for selection response.

Additive genetic variance plays several roles in selection. The magnitude of additive genetic variance relative to total variance is the heritability (h^2) of the trait and is used to predict outcome of SBP. In addition, selection response can be shown to equal the product of the square root of heritability multiplied by additive genetic variance multiplied by selection intensity.

The point is selection response is directly proportional to the relative magnitude of ***additive genetic variance*** and in the absence of this variance a population will not respond to selection.

2.3. Natural Selection

Natural selection is simply selection for the trait fitness. Individual fish contribute genes to the next generation based on their fitness relative to other fish in the population (relative fitness).

Response to natural selection is measured by the change in the overall fitness from the parental to progeny generation. The change in fitness due to one generation of natural selection is the magnitude of the additive genetic variance for fitness as shown by Fisher’s fundamental theorem (Falconer, p346).

Change in fitness due to natural selection will not be observable if constraints in the environment limit the population size. If there is additive genetic variance for fitness in the population, natural selection will continue to improve fitness and when conditions in the environment permit the population will expand. Populations that are able to expand with increasing resources are competitive for their environment and are “healthy” with respect to fitness. Such healthy populations have the best opportunity of surviving over a long period of time.

“Fitness landscape” was used as a metaphor by Sewall Wright (1977) to describe the canalization of fitness and evolution caused by natural selection. Natural selection tends to be a one-way street and once selection moves (changes gene frequencies) toward a particular genetic solution it does not easily revert to another alternative. The landscape Wright described is a mountainous terrain with hills and mountains separated by valleys. The peak of each hill and mountain represents an area of maximum fitness and the higher the mountain the higher the fitness. Dawkins (1996) called ***the highest peak in this landscape Mount Improbable***, in one of his popular books on evolutionary biology.

Natural selection, selecting for fitness forces the population up the nearest mountain. “Nearest” is simply the genetic solution dictated by the genes (and their frequencies) that happen to be in the fittest individuals at that time. Gene frequencies are important as they play a role in terms of critical mass – desirable genes at low frequencies are likely to be lost because of the sampling nature associated with gamete formation.

Natural selection cannot force the population down the mountain, across the valley, and up another potentially higher fitness peak that favours a different configuration of the fitness sub-traits. This is because natural selection increases the frequencies of genes favoured by the “current” fitness peak and tends to eliminate other genes even if they might be superiour for a higher alternate fitness peak.

For natural selection to force a population up Mount Improbable means it must keep climbing mountains until it gets the right one. However, to explore the fitness landscape requires that additive genetic variance is available for alternate fitness peaks and that natural selection is “relaxed” to a point that a new direction becomes the focus of selection. In the normal evolutionary process, this situation arises from a combination of gene flow between populations and moderate to large changes in the environment, which redefines fitness.

Changes in the environment will likely alter the definition of optimal fitness and cause an overall reduction in fitness of an existing population. To use the landscape metaphor, change in the environment is analogous to an earthquake altering the altitude of all the mountains in the fitness landscape and perhaps creating new mountains and valleys. This may put a population formerly at a fitness peak back to sea level. Natural selection will then start over and push the population up the “nearest” mountain. However, this requires that genetic variation is present and applicable to the new version of fitness.

Populations that have limited genetic variance are at risk, as natural selection is not able to restore fitness under these conditions. Small changes in the environment occurring over a long period in a consistent direction will likely not impact fitness of populations with a reasonable level of genetic variability. Natural selection, in this case, will keep up with changes in the environment. However, natural selection will not be able to maintain or improve fitness, in the absence of genetic variance for the evolving definition of fitness.

Populations that are reproductively isolated for many generations will loose genetic variance for fitness since natural selection, like most forms of selection, “consumes” additive genetic variance. Natural selection continues even in stable populations, where size is constant from one generation to the next, until all additive genetic variance for fitness is exhausted. Clearly, genetic isolation is not a favorable condition for wild populations.

2.4. Fitness Profiles

Natural selection causes change in the sub-traits of fitness and it is important to understand these changes in order to predict how the gene pools of wild stocks change under natural selection and how traits important to fitness might change in a farm SBP. Clearly these are important issues in predicting the interaction between escaped farm fish and nearby wild stocks. Falconer (1989) grouped sub-traits of fitness into three classes and developed theoretical “fitness profiles” for each of these classes based on how and why they change under natural selection.

2.4.1. Major Components of fitness, such as the number of eggs produced, increase in nearly a linear manner with selection for fitness since they are directly related to the number of progeny. At the upper range, the profile changes and becomes negatively associated with fitness. This is due to the relationship between traits, for example as egg number increases beyond some point, egg quality decreases. Natural selection, in a stable environment, will cause these traits to go to fixation for the alleles favoured by the definition of fitness. The approach to fixation will be fairly rapid and depends on the relative importance of the sub-trait to fitness.

2.4.2. ***Neutral Class*** of fitness sub-traits contains traits that likely contribute to fitness, but within their normal range of expression, do not alter fitness. This type of trait will maintain genetic variation until random drift causes fixation of relevant loci.

2.4.3. ***Intermediate Optimum*** sub-traits of fitness are those where individuals with values near the population mean have the highest fitness. Selection response in these traits depends on their relationship to fitness.

Sub-traits directly associated with fitness lose genetic variance, under natural selection, by a process called stabilizing selection. After many generations of natural selection, in a stable environment, the loci involved in these sub-traits are expected to go toward fixation. Since selection is for an intermediate, some loci will become homozygous for alleles that increase and other loci for alleles that decrease the expression of the trait. The genetic variance for these traits will go to zero within the stock, but at the population or species level genetic variation is retained.

Thermal insulation of mammalian coats is a good example of this type of trait. The conflict between the need to conserve body heat during rest and yet dissipate heat during activity gives highest fitness to an optimum coat density.

Another example of intermediate optimum is clutch size in birds. The number of eggs laid is directly related to fitness but the environment limits the number of offspring that can be reared. Natural selection in this case would be stabilizing selection and lead to reduced genetic variance. However, if environmental resources expand then optimum clutch size should increase if there is additive genetic variation for the limiting sub-traits.

Sub-traits <u>not</u> directly associated with fitness are maintained at intermediate optimum levels by correlations to traits that do directly effect fitness. Mature body size is an example of this type of intermediate optimum in many species. In mice, female body size is positively correlated to litter size, which is a major component of fitness. However, body size is negatively correlated to predator avoidance and fitness appears to favour intermediate body weights.

The distinction between these two forms of intermediate optimum sub-traits is important since indirect selection does not reduce the genetic variance of the trait to the same degree as stabilizing selection. Genetic variation has been found for mature body size in most wild species indicating this trait is maintained at an intermediate optimum by indirect selection.

For example, heritabilities for mature size observed in wild Pacific salmon stocks (0.2 to 0.4) indicate that levels of additive genetic variance are quite high, this suggests the trait is maintained at an intermediate optimum by indirect selection. Mature weight (growth) is a key trait in the discussion of the interaction between wild and farm salmon stock since it is the focus of most SBP.

2.5. Polygenic Systems

2.5.1. Many genotypes produce the same expression of a trait in polygenic systems. The sub-traits of fitness, and fitness itself, are polygenic and assumed to be controlled by many loci with perhaps multiple alleles. The expression of the trait is the aggregate contribution from all genes effecting the trait. This genetic configuration means that a large number of genotypes can give rise to the same expression for that trait.

A simple example will demonstrate this point. Assume an arbitrary trait is controlled by 100 loci each with three alleles. Further assume at each locus the three alleles contribute +5, 0 or –5 units. Recall that an individual (diploid) has 200 alleles at these 100 loci. So that at the one extreme an individual could have an expression of 1000 units (200 x 5) and on the low side, -1000 units. How many genotypes would produce a score of 500 units? Obviously, a large number of genotypes would give this same expression.

2.5.2. Major component of fitness. Consider that the arbitrary trait is egg number. Natural selection will increase egg number by selecting “+5” alleles until it reaches the maximum imposed by other traits (e.g., egg quality). Assume this maximum is 800 units. Natural selection over a long period would force fixation of alleles such that the composite value is 800. For example 80 loci fixed at “+5” and 20 at “0” or 5 loci at “-5”, 85 at “+5” and 20 at “0”. The genetic variances for each case is zero – all loci are homozygous. However, if you cross several populations that have gone through the same selection process you restore the genetic variance. This is true because the allele that becomes fixed, at a particular locus, in one stock may be fixed for another allele in another stock.

2.5.3. Intermediate optimum sub-trait directly related to fitness. In this situation the 100 loci that control the trait would go to fixation by stabilizing selection. The end result would be that sets of loci would become homozygous for alleles such that the aggregate value would be the level favoured by fitness. But it would be chance that dictates which allele is fixed at each locus. The difference between this case and the previous one is that number of loci fixed for each type of allele would not be skewed favoring one extreme. Crosses between populations would be expected to restore genetic variation. This is true because the alleles that are fixed, at a particular locus, in one stock may be fixed for a different allele in another stock.

2.5.4. Intermediate optimum sub-traits <u>not</u> directly associated to fitness and neutral sub-traits. These traits, for example mature weight, would retain genetic variation for a long period. But with prolonged genetic isolation these sub-traits will eventually lose genetic variation primarily from random drift but natural selection will also have an impact. These sub-traits are expected to recover the genetic variation by crossing stocks.

2.5.5. Polygenic systems conserve genetic variation. Long-term natural selection in stable environments and random drift cause loci to become homozygous. This means that genetic variation is reduced to zero in genetically isolated stocks. However, fixation is not uniform across stocks as different alleles are fixed at a locus because of chance and differences in the definition of fitness from stock to stock. This means that pooling genomes of several stocks into a synthetic will restore genetic variation for fitness. The amount of variance that is restored depends on the number and diversity of the stocks in the synthetic.

2.5.6. Migration of fish between wild stocks is the normal way stocks maintain or restore genetic variation. Obviously, migration between related stocks is less useful, in this regard, than between unrelated stocks.

3. Expected Fitness of Wild Salmon Stocks

Wild stocks are both the source of the farm stocks and the recipient of genetic intrusion from the escapees from farm sites. It follows that the expected fitness of wild stocks is important as both the starting point for farm stocks and as the base line for assessing the impact of farm genes migrating into a wild stock.

3.1. Large wild stocks. Wild populations that are reasonably large and have been stable in size for a long time are likely at, or near, a fitness peak. The genetic variation for major components of fitness will be small and perhaps near zero. Genetic variance of intermediate optimum traits will be low for those subjected to stabilizing selection, but additive genetic variance will be present for traits like mature size since they are held at an optimum level by indirect selection. Neutral traits will retain genetic variation since natural selection will not alter these.

When a stock is stable the overall fitness is the same as the mean relative fitness of the individuals comprising that stock. However, natural selection may still operate if the additive genetic variances of traits directly effecting fitness (major components and intermediate optimum) are not yet exhausted. In time these variances will be exhausted and most loci will be homozygous, if the population remains isolated.

3.2. Small wild stocks. Natural selection will act on small wild stocks or those that have gone through a genetic bottleneck in the same way as for larger populations. The difference is that as population size decreases the rate of random drift increases. Random drift causes a loss of genetic variation and in very small populations the loss of favorable genes due to chance.

Perhaps the biggest hazard for small wild populations is that a combination of natural selection and random drift exhausts genetic variance for fitness and the population can not respond to small changes in the environment.

4. How Does Fitness of a Farm Stock Change in a SBP?

4.1. ***Farm stocks originate from wild populations and are then selected for economic traits and for domestic fitness.*** The change in fitness, under this selection, depends on the level and variance of fitness and fitness sub-traits in the founding genepool and on how artificial and domestication selection effects these traits.

4.1.1. ***The selection objectives of most salmon SBP include*** increased growth rate and survival to market under a normal culture program. Mature weight is generally considered an important fitness trait and it is highly correlated to growth, therefore mature weight is expected to change in SBPs. The SBP may also impose selection for disease resistance and quality characteristics such as flesh colour and fat content. Selection for these traits will likely alter the original fitness since fitness is the end product of all the animal's developmental and physiological functions including traits of interest to a SBP.

4.1.2. ***Farm fish are not subjected to natural selection, per se***, as fitness is defined in terms of natural environments. However, they are subjected to automatic selection for ***"domestic fitness"*** which is the relative success of a fish surviving and reproducing in the farm production system and SBP. Like fitness, this trait (domestic fitness) is the end product of all the animal's developmental and physiological functions and is polygenic. But unlike fitness, domestic fitness is constrained by the SBP and the fish culture methods used by the farm.

4.2. ***The genetic background of the farm stock plays a major role*** in the effect of artificial and domestication selection on fitness. Farm stocks developed from a single wild stock will differ dramatically from those developed from several wild sources in both the initial fitness and how fitness changes under a SBP.

4.2.1. ***Farm stocks created from a single wild stock***. Consider farm stocks that were created from a single large stable wild stock and assume they were based on a good representative sample of the founder genepool.

Selecting for growth will not affect fitness directly but is expected to alter the major components that are correlated to growth. The forces that keep mature weight at an intermediate optimum will be reversed, but it is difficult to predict how these traits might change, if at all.

Natural selection may have exhausted the additive genetic variances for these traits in the founding wild stock, which means that the genes and gene combinations should remain unchanged by SBP selection.

If the wild population still has genetic variation for major fitness traits this will come from migration or mutation. In this case the SBP may alter fitness sub-traits. For example, large females produce more eggs than smaller fish but are less able to avoid predators in the natural environment. Hence an intermediate weight would be favoured in the wild. Under farm culture predator avoidance is not important but increasing egg number is still an advantage, but more importantly selection for growth would lead to larger mature weight. The reduced success in predator avoidance caused by selecting for growth would simply be fish size and not other aspects of predator avoidance.

Selection for survival in the SBP and automatic selection for domestic fitness likely will only effect fitness traits if genetic variance is present. Natural selection is expected to eliminate most of the additive genetic variance for major fitness traits.

If genetic variance is present, then selection of the farm fish will effect some sub-traits of fitness and not others. Traits such as predator avoidance are not important in most farm situations and any impact would arise from correlated responses to selection for traits that are important, such as, feeding behaviour.

Are there any major fitness traits in salmon where changing the trait in one direction increases fitness but decreases domestic fitness? Are there any intermediate optimum traits, perhaps colour, in salmon where the optimum level of a trait are different for fitness and domestic fitness?

If the answer is yes, to either question, these traits will change under selection in the farm stock. Again, selection may cause relatively large phenotypic changes but genetic changes are expected to be small since the traits are polygenic.

4.2.2. Synthetic farm stocks created from several wild populations. Consider a synthetic farm stock created from two or more large, stable and unrelated wild stocks. Assume that good representative samples were obtained from each stock and that the farm SBP pooled gametes of the founder stocks to form a single synthetic. Recall from the discussion on natural selection that the wild stocks used as founders are expected to be at fitness peaks in their own fitness landscape.

Expect to see differences between the founding stocks for all sub-traits of fitness and for fitness itself. Some of these differences will be due to natural selection in the unique environmental landscape of each stock and others simply by chance. Some of these differences may be striking and recognized as unique attributes of individual stocks (e.g., colour). These traits could be simply inherited, or polygenic, but with regard to fitness they are likely either neutral or intermediate optimum traits.

The presence or absence of colour, albino vs. pigmented, is likely simply inherited. However, different shades of pigment is likely polygenic and it could be argued that colour shade has an intermediate optimum for a specific ecological niche. The point is these unique characteristics of a stock fall within Falconer's fitness profiles. The genetic differences in fitness between wild stocks are, of course, much greater than that caused by these stock specific characters.

The additive genetic variance for traits directly associated with fitness in each founding stock has likely been consumed by natural selection. This means that the majority of loci involved in these traits is homozygous and have no genetic variation. But no one stock is likely homozygous for all of the desired alleles. The reason of course, is that the stocks come from local fitness peaks in their respective environments and the alleles that are fixed depend on the genes available in the founder population and subsequent immigrants, chance and natural selection history of the stock.

Creating the synthetic restores the additive genetic variance for fitness sub-traits lost due to natural selection, chance and random drift. It is almost certain that a synthetic wild population would end up at a higher fitness peak in any of the original fitness landscapes than the resident native stock. This is because each stock is expected to contribute some superior genes not found in the other stocks.

In the synthetic, traits that directly effect fitness (major components and intermediate optimum) will have genetic variance, which was exhausted in the original wild stocks. The mean relative fitness of the synthetic may be lower than the resident stock in the first few generations, but the variance of relative fitness will be high and the response to natural selection will be very rapid.

The hazard is that by chance, natural selection will force the synthetic stock up a local fitness peak that is lower than the original. However, the likelihood of this undesirable outcome decreases as the genetic variance for fitness increases, so it is related to the number of stocks in the synthetic.

Selection for growth in a synthetic will have the same impact, with respect to fitness, as selection in a farm stock from one founder stock. Genetic variation for fitness within the farm stock will not change very much and little, if any, genetic information will be lost because of selection for growth.

Selection for domestic fitness (including survival) in a synthetic will likely have more impact on fitness traits than in stocks from a single source. Most stocks from a single founder are expected to have little or no additive genetic variance for traits directly effecting fitness, and as a result selection can not change these traits very much. The synthetic is expected to have additive genetic variance so selection could alter fitness substantially. How this selection effects fitness is difficult to say because it depends on the differences between natural fitness and domestic fitness.

Traits that are important to fitness but ignored in domestic fitness will not be altered and will presumably retain the mean and variance they had at the start. Automatic selection for traits where domestic fitness and fitness are in conflict, improving domestic fitness reduces fitness, will lower the mean fitness. On the other hand if shifting a trait in one direction improves both domestic fitness and fitness then selection in the farm stock will improve fitness.

4.3. Polygenic traits, like those associated with fitness, are expected to retain genetic variation while under selection. The synthetic farm stock, even after several generations of selection or domestication, will likely have more genetic variation for fitness than farm stocks from a single founding stock, or than most wild stocks for that mater. This is not to say that the absolute fitness of a synthetic farm stock will be equal to a wild population, but additive genetic variance for fitness will likely be greater in the synthetic farm stock. This means the potential fitness will be higher.

5. Expected Genetic Impact of Escaped Farm Fish on Wild Stocks

5.1. Escaped farm fish are expected to be less fit than wild fish from an adjacent wild stock. The SBP and automatic selection for domestic fitness alters the expression of the sub-trait of fitness of farm stocks relative to the original wild stocks. This will alter the balance between sub-traits of fitness and likely reduce fitness.

In addition, the farm stock is likely not related to adjacent wild fish, in which case the differences in fitness would include those of the original founding genepool. If the local wild stock is not as fit or fitter than the farm fish, then the wild stock is in trouble and may require help to survive.

A “healthy” wild stock, with respect to fitness, has some additive genetic variance for fitness. Such a population would expand to the limits of the ecological niche and would not really be stable but would increase and decrease in size depending on the resources in the environment. If a stock is not competitive for resources in its environment then it is subject to be replaced by a competitor.

5.2. ***Intrusion of escaped farm fish into a healthy wild stock.*** The escaped farm fish will, on average, have a relative fitness below the average of the wild fish. However, the absolute fitness of the "new" stock (wild + farm) may still have a mean relative fitness of one or greater and show no reduction in fitness. This depends on the size of the escapement, relative reproductive success of farm fish that enter the spawning grounds and many other issues. But from a genetic encroachment perspective, it is not the number of escapees that is important, but the number of offspring that appear in the next generation of the wild stock.

5.2.1. ***If the farm stock came from the wild stock in question*** it will have little genetic impact, as it will not add any new genetic variation for fitness. The intrusion of farm fish will likely reduce fitness of wild stock as explained above. Natural selection is expected to restore fitness but more slowly than if the farm and wild stock are unrelated and, of course, since no new genetic variance has been added the fitness of the wild stock will not exceed the pre intrusion level.

5.2.2. ***If the farm stock is a synthetic or derived from a single stock unrelated*** to the local wild stock, then the intrusion of farm will have a different result. The intrusion may initially reduce fitness of the wild stock. However, in this case the farm fish will contribute genes for fitness that are not present in the wild stock. This will provide genetic variation for relative fitness. But the real impact is long-term, since the new genes will integrate with the wild stock genepool and provide additive genetic variation for fitness in subsequent generations. Natural selection will then increase relative fitness of the stock and may move the stock toward a new higher fitness peak.

5.3. ***The best way to maintain a healthy wild stock*** is to provide a moderate, but diverse gene flow into the stock. This will provide the additive genetic variation needed for natural selection to respond to changes in the ecological niche of the stock. This will assure, as much as possible, the long-term survival of the stock and this is the best way to maintain genetic diversity at the population and ultimately at the species level. Migration from other wild populations is the normal source of gene flow, but farm stocks, particularly synthetic ones, can provide the same genetic material. The only genes that are at risk under a moderate migration program are those genes that are negatively related to fitness.

The number of genes that migrate by this route is important. A small constant migration of genes from a variety of sources would be beneficial to nearly any wild stock as the influx of genetic variation would allow natural selection to explore the “fitness landscape”. This is even more important if the landscape is changing.

Without gene flow, useful genes may be lost by fixation due to natural selection and random drift. Clearly, large escapements of farm fish could flood a wild stock and overwhelm the process of natural selection and in the short-term this may put the wild population at risk. Measures should be taken to minimize this risk.

5.4. Numerically small wild stocks are probably at risk for several reasons. If the ecological niche of the stock does not have enough resources to support it at some critical minimum level then the stock should be moved or allowed to become extinct, following natural course of events. The alternative is to fix the deficiencies in the environment. There are no genetic solutions to this problem. However, if the stock is small and does not expand in an environment with adequate resources then the fitness of the stock is the likely problem. The solution is to add new genetic variation for fitness from as many sources as possible. This should be done slowly to try and utilize the fitness of the existing stock and maintain its unique characteristics, if they are deemed to be important.

6. Concluding Remarks

This paper is not a recommendation for regular releases of farm fish into wild populations. But is rather an attempt to focus on the real genetic issues involved in preserving genetic diversity in wild salmon.

The focus of this paper has been on genetic variance, which is simply the statistical quantification of genetic diversity. Maintaining genetic diversity in wild species of salmon is best managed by maximizing genetic diversity in the populations of the species. In turn, the best way to maintain genetic diversity in a population is to maximize genetic diversity in the stocks of the population.

Genetic isolation of a stock will lead to loss of genetic variation and, in the long term, to extinction of the stock. Allowing or providing gene flow between populations and between stocks is the only way that stocks will remain healthy with regard to fitness over long periods of time. These healthy stocks will respond to the inevitable changes in their ecological niche by adapting to environmental changes through natural selection. If the stock has limited genetic diversity and more particularly, no additive genetic variance, then if can not respond to new environmental conditions. Ultimately, the stock will not be competitive for its ecological niche and will be replaced by a competitor.

Small escapements of farm fish into reasonably large health wild stocks are expected to have a small negative effect on fitness, in the short-term. But these intrusions will likely have long-term benefits by providing genetic variance for relative fitness.

Large intrusions of farm genes into wild genepools will likely cause a severe decline in fitness, in the short-term. Recovery is likely, but several generations would be required and the stock may not survive the initial flood. The affect on fitness depends on the diversity of genes contributed by the farm stock.

“Small” and “large” are vague terms and defining these with some confidence will require extensive and well-designed field research and simulation studies.

References

Dawkins, Richard, 1986. The Blind Watchmaker. W.W Norton & Company, London & New York

Dawkins, Richard, 1996. Climbing Mount Improbable. W.W Norton & Company, London & New York

Falconer, D.S., 1989. Introduction to Quantitative Genetics, 3rd ed. Longman Scientific & Technical, London (Co-published by John Wiley & Sons, New York). Chapter 20.

Peterson, R.G., 1993. What is the genetic implication on wild stocks when farm fish escape. Northern Aquaculture, Vol. 9, No. 5, p 23-26.

Wright, Sewall, 1977. Evolution and the Genetics of Populations. The University of Chicago Press, Chicago.

R.G. (Ray) PETERSON, Ph.D.

4291 West 16th Ave., Vancouver, BC, V6R 3E5, Canada
Phone: (604) 224-1919, e-mail: peterson@unixg.ubc.ca

Professional Profile

Partner, TRI-GEN Fish Improvement Ltd. Vancouver, BC
Company founded in 1988 to research, develop and manage salmonid broodstocks.

Associate Professor (*emeritus*) of Animal Breeding and Genetics (1968-1998).
Department of Animal Science, University of British Columbia, Vancouver, BC

Ph.D. and M.Sc. University of Illinois, in Quantitative Genetics and Animal Breeding.

Thirty years of research and teaching quantitative and population genetics and animal breeding in the Department of Animal Science, University of British Columbia.

Membership in Professional and Learned Societies

Aquaculture Association of Canada. (1989-current)
Canadian Society of Animal Science. (1969-1990)

Professional Committees

Canadian/New Zealand Genotype x Environmental Interaction Trial (CANZ). (1984-1990 chair). The CANZ Trial is a cooperative research effort involving 8 Canadian Universities and Colleges, Agriculture Canada, and in New Zealand the Livestock Production group and Massy University. Ten Canadian herds and 20 New Zealand herds cooperated in the study.

Technical Committee of the Canadian Genetic Evaluation Board. (1980-1995 chair 1982-88). This committee deals with the technical issues involved with genetic analyses and selection programs for the Canadian dairy industry.

Canadian Genetic Evaluation Board for Dairy Animals. (1990-1995), The mandate of this committee is to provide direction and assistance to the dairy industry for genetic improvement programs.

List of Recent Salmonid Publications

Peterson, R., B. Swift, M. Mulholland and D. Tillapaugh. 1995. Feasibility Analysis for Development of a Selective Breeding Program for the BC Atlantic Farming Industry. Prepared for BC Salmon Farmers Association, Vancouver, BC.

Winkelman, A.M. and R.G. Peterson. 1994. Heritabilities, Dominance Genetic Variation, Common Environmental Effects and Genotype by Environment Interactions for Weight and Length in Chinook Salmon. Aquaculture 125: 17-30.

Winkelman, A.M. and R.G. Peterson. 1994. Genetic Parameters (Heritabilities, Dominance Ratios and Genetic Correlations) for Body Weight and Length Measurements of Chinook Salmon after 8, 21 and 28 Months of Saltwater Rearing. Aquaculture 125: 31-36.

Peterson, R.G., 1993. What is the genetic implication on wild stocks when farm fish escape. Northern Aquaculture, Vol. 9, No. 5: 23-26.

Peterson, R.G., A.M. Winkelman, R. Devlin and W. Harrower. 1992. The Effect of Sex on Growth of Chinook Salmon. Bul. Aquacul. Assoc. Canada 92-3: 37-39.

Winkelman, A.M., R.G. Peterson and W. Harrower. 1992. Dominance Genetic and Tank Effects for Weight and Length in Chinook Salmon. Bul. Aquacul. Assoc. Canada 92-3: 40-42.

Swift, B.D., R.G. Peterson and A. Winkelman. 1991. Heritability and Genetic Correlation Estimates for Weight, Length and Survival of S_0 and S_1 Coho Salmon (*Oncorhynchus kisutch*). Bul. Aquacul. Assoc. Canada 91-3: 19-21.

Winkelman, A.M., R.G. Peterson and W. Harrower. 1991. Strain Comparisons and Estimation of Genetic Parameters in Chinook Salmon. Bul. Aquacul. Assoc. Canada 91-3: 22-24.

Peterson, R.G., L.K. Dunn and G.A. Hunter. 1990. Proposed Newfoundland Atlantic Salmon Broodstock Program. Department of Fisheries and Oceans, Science Branch, Newfoundland Region. 168 pp.