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Genetic Risks and Hazards in Hatchery Operations: Fundamental Concepts and Issues

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Abstract.—As concern over erosion of genetic diversity in fish stocks has increased over the years, so has concern about the role of hatcheries in influencing genetic change. Whereas past genetic concerns regarding hatchery operations have tended to emphasize effective population size of hatchery broodstocks, now hatchery managers need to consider a more comprehensive view of genetic risk. In this paper we present some basic concepts and associated issues in such a broad view. We recognize four fundamentally different types of genetic hazard: (1) extinction, (2) loss of within-population variability, (3) loss of among-population variability, and (4) domestication. The importance of type-2 hazards in hatchery operations has long been realized, but types 3 and 4 are controversial because of a scarcity of empirical data and because consideration of them has great ramifications for hatchery operations. Precise quantification of genetic impacts in terms of fitness depression is likely to remain a difficult if not impossible task. Ultimately, incorporation of genetic concerns into hatchery operations and other aspects of fisheries management will require managers to shift their perspective from one of managing fitness to one of managing genetic diversity.

During the last century, fishery managers increasingly have asked hatcheries to meet the demands of growing human populations for fish. This trend shows no signs of abating. In the Columbia River basin alone, more than 90 state and federal hatcheries raise and release 190 million juvenile Pacific salmon *Oncorhynchus* spp. annually, and even more hatcheries are planned (Anonymous 1990a). In the past, hatcheries produced fish primarily to augment fisheries. Now hatcheries in the Columbia River basin and elsewhere also are asked to help conserve and restore depleted natural populations. Implicit in this shift—from producing an exploitable commodity to conserving populations—is the need to protect the capacity of populations to persist and be productive.

The productivity of populations and their resilience to environmental change is a result of the genetic diversity they contain. In the last 20 years, fish geneticists increasingly have become aware of how hatchery operations can alter genetic diversity, and managing these changes is now a great concern (Bakke 1989; Hindar et al. 1991; Hilborn 1992; Meffe 1992). Unlike disease or nutritional problems that can be corrected in the next cycle of hatchery production, unwanted changes in appropriate forms or combinations of genes in populations can depress productivity for many years. Genes are transmitted over generations, and productive combinations of genes evolve in populations over many hundreds or thousands of generations (Dobzhansky et al. 1977).

Despite recent efforts to acquaint fishery managers and aquaculturists with genetic concepts (Kapuscinski and Jacobson 1987; Ryman and Utter 1987; Tave 1993), awareness of genetic concerns in hatchery operations varies widely among fishery professionals. Not surprisingly, many do not recognize genetic threats to the success of hatchery programs intended to conserve or restore natural populations. Our goal in this paper is to acquaint fishery professionals with both the basic concepts of genetic risk and the issues of risk management in the culture and uses of hatchery fish. We review the genetic vocabulary necessary to describe genetic risks and hazards and their relationship to genetic diversity. We classify the basic types of genetic hazards, describe their potential sources in hatchery programs, and discuss issues related to them. We close by recommending a shift from fitness-based to diversity-based management of genetic risk.

Basic Terms

Risk often is used ambiguously in describing a threat. Here, we distinguish between hazard and risk. A hazard is a potentially adverse consequence of an event or activity, whereas risk is the probability of the hazard occurring (Smith 1992). Thus,

production of fewer offspring is a hazard of interbreeding with a foreign population; risk is the probability of producing few offspring.

The most general definition of genetic hazard is loss of genetic diversity. Genetic diversity is all the genetic differences contained within a population or group of populations. A population here means a group of interbreeding individuals. A gene is a hereditary unit of genetic information, which is contained at a site on a chromosome called a locus. A trait controlled by a single gene is called a single-locus trait. Each fish usually has two copies of each gene—one from each parent. Copies may be biochemically different forms of the gene, or alleles. The fish is homozygous if the copies are the same allele and heterozygous if they are different. Likewise, different fish may have different numbers of chromosomes. Genetic diversity then primarily consists of the quantity and variety of alleles, chromosomes, and arrangements of genes on the chromosomes that are present in the population(s).

Fundamental to how we manage genetic diversity is how we detect and measure it. Here the distinction between genes and their effects is very important. The genetic composition of a fish at one or more loci is its genotype. The biochemical, physiological, morphological, or behavioral expression of the genotype is the phenotype. Phenotypic differences may be the expression of genes at one to hundreds of loci. Expression of biochemical markers such as allozymes is typically controlled by one or two gene loci. Expression of traits related to fish performance is typically controlled by numerous loci. Such traits are called quantitative traits.

We easily can detect and quantify many single-locus genotypic differences through DNA and protein electrophoretic analyses. Well-established statistical methods exist for evaluating single-locus diversity within and among populations and higher taxa (e.g., Weir 1990). However, it is difficult to predict the effect of these differences on the performance of individuals.

In contrast, we often do understand the effects of phenotypic variation in quantitative traits, such as physiological, morphological, or behavioral differences. However, we usually can measure genotypic differences in quantitative traits only indirectly by examining differences in phenotype, and the phenotype is also influenced by the environment (Falconer 1981; Tave 1993). Consequently, to manage genetic differences within or among populations at quantitative traits, we need to understand potential variation caused by different genes in different environments, the cumulative effects of different alleles over all loci, the interaction of different alleles at each locus, and the interaction of alleles at different loci. This requires elaborate breeding experiments in controlled environments. Thus, an interesting dichotomy exists: measurement of genotypic diversity is straightforward at many traits that do not clearly relate to phenotype, but measurement of genetic diversity is extremely difficult at traits that do relate directly to phenotype. This dichotomy is at the root of many of the genetic risk issues described later in this paper.

Genetic Hazards

Any condition that has the potential to decrease either within- or among-population genetic diversity is a source of genetic hazard. Consideration of four major types of genetic hazards have been useful in planning multispecies hatchery programs for Pacific salmon (Clune and Dauble 1991): (1) extinction; (2) loss of within-population genetic variability; (3) loss of among-population genetic variability; and (4) domestication. When natural populations are used as sources of broodfish or artificially propagated fish are released into the wild, hazards occur at multiple geographical and genetic scales. For example, loss of a unique population means extinction at the population level but loss of among- and within-group diversity at the species level. Likewise, a threat to a hatchery population may also threaten wild populations but with a different kind or magnitude of hazard. Consequently, hazards need to be always defined relative to a reference population or scale.

Extinction

Definition.—Extinction is the complete loss of all genetic information. It is the most serious hazard, because once a population is gone, all the unique aspects of the diversity it contained also are lost. Because different populations have different gene pools, extinction of any population also reduces overall genetic diversity of the species.

Mechanisms.—Extinction significantly differs from the other hazards in hatcheries because it is primarily caused by nongenetic mechanisms. In most cases, the main causes of extinction have been grouped into three nongenetic sources of fluctuations in population size (Shaffer 1981): (1) demographic variation or random differences in reproductive success, (2) environmental variation, and (3) catastrophes. Genetic mechanisms that potentially reduce reproductive success, such as inbreed-

ing in very small populations, in theory can also contribute to an "extinction vortex" (Gilpin 1987).

Sources.—Extinction is the primary focus of most risk assessment in conservation biology (e.g., Burgman et al. 1993), but it has been overlooked in hatchery programs. One of the attractions of artificial propagation is that it can reduce environmental variation, thereby reducing the risk of extinction. However, hatchery programs still can be abundant sources of uncontrolled demographic, environmental, and catastrophic changes. Broodfish may be taken from small, wild populations without replacement. Disease, power failures, and dewatering can be catastrophic to even the best hatchery programs. Ecological interactions between released hatchery fish and wild fish that may depress populations (e.g., Sholes and Hallock 1979; Nickelson et al. 1986; Hindar et al. 1991) are another uncontrolled source of demographic variation.

Unresolved issues.—Consideration of extinction as a hazard of hatchery operations is new. It is important because, more and more, hatcheries are being identified as a means of recovering populations in danger of extinction. If we can identify and remove sources of this hazard in hatcheries, and genetic function of the population has not been impaired, the risk of extinction may be reduced, and the population may be able to grow (but see Peterman 1987).

Methods for assessing risk of extinction are rapidly developing. However, it remains very much a theoretical and modeling process (Gilpin and Soule 1986; Goodman 1987; Shaffer 1987; Burgman et al. 1993), and is likely to remain so. Validation of theory requires extinctions to be carefully observed, which is not likely to occur when we are intervening to prevent extinction. Viability analyses of fish populations (e.g., Rieman and McIntyre 1993) are rare but will undoubtedly become more common.

Loss of Within-Population Variability

Definition.—Loss of within-population variability (diversity) is the reduction in quantity, variety, and combinations of alleles in a population. Quantity is the proportion of an allele in the population. Variety is the number of different kinds of alleles.

Mechanisms.—Two mechanisms of genetic change influence within-population diversity: random genetic drift and inbreeding. Genetic variability is lost in all populations through random genetic drift. It occurs because during spawning, many more gametes are produced by the parents than actually unite to become new zygotes. Each new generation, then, is a sample of the quantity and variety of alleles present in the gametes of the previous generation. Most of the time, the quantity and variety of alleles present in the progeny will not be an exact copy of the parents; the rarer the allele and the smaller the number of gametes that start the next generation, the more likely that the allele will not be represented exactly. Consequently, over time, variation will be lost, especially in small populations.

In the real world, sampling of gametes is not random. Gametes of some fish in the population will be better represented because there were an unequal number of males and females, some individuals reproduced more than once or at older ages, or more of their offspring survived to reproduce. All these variables make the genetically effective population size smaller than the census size. To compare rates that heterozygotes or alleles are lost in different populations, therefore, geneticists correct for several deviations from the ideal state: unequal sex ratio, age structure, differences in family size, and temporal fluctuations in population size (Falconer 1981). For example, a broodstock of 4 males and 100 females will lose as much variability due to drift as a population of 8 males and 8 females, everything else being equal.

Inbreeding is the breeding of related individuals. By itself, inbreeding does not lead to changes in frequency or variety of alleles in a population (Falconer 1981). Rather, inbreeding increases individual and population homozygosity, because more closely related individuals are more likely to have the same alleles than are less related individuals. This leads to changes in the frequency of phenotypes in the population. If selection then acts on these phenotypes, allele frequencies can also change.

Many studies have documented poor phenotypic performance associated with inbreeding (inbreeding depression) in captive fish populations (see Tave 1993; Waldman and McKinnon 1993). Inbreeding depression has two different genetic sources. First, it may result from increases in phenotypic expression of homozygous genotypes for rare, harmful alleles that are normally hidden in the population in heterozygotes. Second, if heterozygotes normally perform better than do homozygotes, then the decrease in heterozygosity will lead to a decrease in performance (Waldman and McKinnon 1993).

Sources.—The concept of effective population size can be used to identify potential sources of random genetic drift in hatchery programs and to

recommend guidelines to minimize it (Kapuscinski and Jacobson 1987; Simon 1991; Tave 1993). Potential sources of smaller effective population size in artificial propagation systems are easily identified and many have been documented. These sources include using small numbers of broodfish, using more females than males (or the alternative) and pooling gametes, changing age structure, and allowing progeny of some matings to have greater survival than allowed others (Gharrett and Shirley 1985; Simon et al. 1986; Withler 1988).

The most important source of small effective population size is the variance of family size, or variation among families in the number of offspring that survive to reproduce (Falconer 1981). Simon et al. (1986) documented sources of this hazard in common hatchery procedures and noted that making simplified assumptions about family size variance could lead to large overestimates of effective population size. Likewise, simplifying assumptions about family size variance may also lead to serious overestimates of effective population size for natural populations in which hatchery fish mingle with wild fish when there is an overall survival difference between hatchery and wild fish (Ryman and Laikre 1991).

Reduced genetic diversity in hatchery stocks compared with their wild counterparts (Allendorf and Phelps 1980; Ryman and Stahl 1980; Waples et al. 1990) indicates the potential for random genetic drift in hatcheries. Leberg (1992) experimentally verified the loss of genetic diversity due to genetic bottlenecks—temporary reductions to very small effective population sizes (Nei et al. 1975)—predicted by genetic theory. Bottlenecking can have significant effects on the success of hatchery strains. For example, populations of eastern mosquitofish *Gambusia holbrooki* established from a small number of related founders grew at much lower rates than did those established from unrelated founders (Leberg 1990).

Unresolved issues.—Most questions pertain to two major issues. First, what are the critical thresholds for loss of within-population variability? Second, can this hazard be controlled?

When hatchery programs are judged based on the performance of the fish they produce, it is critical to know the biological threshholds that lead to reduced genetic diversity. However, threshholds, such as the degree of relatedness beyond which inbreeding depression becomes significant or the minimum effective population size that can be maintained over time before population growth suffers, are unknown and may be different for each population (Shields 1993). How large an effective population size should a population have? Lande and Barrowclough (1987) suggest that 500 individuals may be sufficient for conservation of genetic diversity underlying quantitative traits. What level of heterozygosity is desirable? This depends on whether fish performance declines because of an increase in the number of harmful alleles at homozygous loci or because of the loss of superior heterozygous loci (Lande 1988; Mitton 1993). How do we judge impairment? At what point do we view a population as genetically damaged? And what course of action should we take? Answers to these questions require detailed genetic knowledge of quantitative traits, which is nearly impossible for hatchery managers to monitor.

Total control over random loss of within-population genetic diversity is very difficult. Managing loss by controlling broodfish number, sex ratios, and age structure is possible, though logistically difficult. Because variance in family size is measured on adult progeny, it cannot be estimated directly without a pedigree, which is usually unavailable.

Loss of Among-Population Variability

Definition.—Loss of among-population variability is the reduction in differences in quantity, variety, and combinations of alleles among populations. As with the loss of within-population diversity, consequences of this hazard can be viewed from two different perspectives. At the multipopulation level, the potential evolutionary consequence is reduced ability of the species or group of populations to respond differently to environmental change. At the individual population level, at which most hatchery programs operate, this hazard is the loss of genetic uniqueness with a concurrent reduction in performance of the fish.

Mechanism.—The genetic mechanism for loss of among-population genetic diversity is gene flow at excessive levels or from nonnatural sources. For management purposes, we often consider populations as reproductively isolated units. In many fish species, however, naturally occurring gene flow is an important factor in maintaining genetic diversity. Consequently, the standard for judging gene flow is natural levels and from natural sources.

Excessive gene flow may reduce performance of individual populations (outbreeding depression) by disrupting their genetic organization (Shields 1982). Outbreeding depression has two possible genetic sources (Templeton 1986). The first is loss of adaptation. A population is adapted to a local environ-

ment if its gene pool contains high frequencies of alleles that help it do well there. Introduced alleles from other populations that have evolved in different environments may be less beneficial than are the native ones. Their presence automatically reduces the frequency of favorable alleles. The net result is that the population becomes less well adapted.

The second cause of outbreeding depression is the breaking up of favorable combinations of alleles called coadapted complexes. Recall the complex relationships between alleles and loci that underlie the expression of quantitative traits. As immigrating alleles replace existing alleles in the population, new less favorable allelic combinations may be formed, reducing performance. Whereas outbreeding depression caused by loss of adaptation can be expected to become evident the first generation after the gene flow occurs, outbreeding depression caused by breakdown of coadapted complexes may not be apparent until the second generation (Gharrett and Smoker 1991; Lynch 1991).

Sources.—Conditions that increase gene flow are common to past and present hatchery practices in this country (Philipp et al. 1993). In the Columbia River, for example, hatcheries routinely transfer eggs and fish from different populations between hatcheries to meet production needs (Howell et al. 1985). Likewise, stocking programs commonly release fish into streams outside the original distribution of the introduced fish, resulting in gene flow if stocked fish survive to reproduce with native fish. Many management programs have combined both practices by using hatchery stocks of mixed ancestry over wide geographical areas (Howell et al. 1985).

Evidence of loss of among-population genetic diversity as a result of hatchery programs is extensive, especially for North American salmonids. Numerous distinctive populations of western trout *Oncorhynchus* spp. have been lost by hybridization with introduced rainbow trout *O. mykiss* (Behnke 1992; Busack and Gall 1981; Campton and Johnston 1985). Other studies have noted that in some environments native genotypes may persist in spite of large levels of stocking (Wishard et al. 1984; Currens et al. 1990), presumably because introduced fish were poorly adapted to these environments.

Unresolved issues.—As with loss of within-population diversity, the major unanswered questions for managers revolve around two issues: identifying threshholds for managing hatchery operations based on fish performance and determining how loss of genetic diversity can be realistically controlled.

Although evidence exists for local adaptation, especially in salmonids (reviewed by Taylor 1993), we know of no empirical data on outbreeding depression in fish that involves anything but extremely distantly related populations (e.g., Gharrett and Smoker 1991). Thus questions about how much outbreeding depression can be expected under different circumstances remain unanswered. For management of hatcheries based on performance measures, there are few standards for monitoring the risks of this hazard and consequently few incentives. Most evidence of local adaptation, for example, is circumstantial (Taylor 1993). Many studies suggest adaptation, but definitive proof is difficult because natural selection is difficult to study. Theoretical models of outbreeding depression (Emlen 1991; Lynch 1991) may be helpful but will be difficult to verify. Other threshholds for managing based on performance include determining whether there is a maximum acceptable level of genetic or ecological distinctness between populations beyond which performance suffers with gene flow. If gene flow has already occurred, how fast can natural selection overcome outbreeding depression? In what cases will benefits of gene flow be expected to outweigh the temporary cost of outbreeding depression (Templeton 1994)?

Unlike measures of performance, genetic diversity among populations can be measured and monitored. Sources of the hazard can be eliminated, although it might be expensive and logistically difficult. Consequently, loss of genetic diversity among populations can be potentially managed. However, the critical question is how to measure natural levels and sources of gene flow that lead to patterns of among-population differences. This is especially important if gene flow is to be used as a means of rejoining fragmented populations.

Domestication

Definition.—Domestication is the changes in quantity, variety, or combination of alleles within a captive population or between a captive population and its source population in the wild as a result of selection in an artificial environment. This hazard is similar to loss of within-population diversity with two important differences. First, changes in genetic diversity by genetic drift are random in character, whereas diversity lost due to domestication is directly related to specific traits. Second, diversity is lost through random genetic drift at a rate inversely proportional to effective population size, whereas through domestication it is lost at a rate dependent

on the genetic nature of the traits and selection intensity imposed. Domestication makes fish culture more effective, but it may also decrease the performance of hatchery fish and their descendants in the wild.

Mechanism.—Taking fish into an artificial environment for all or part of their lives imposes different selection pressures on them than does the natural environment. Decreased reproductive success of some genotypes in the hatchery environment leads to genetic changes in the population.

Sources.—Domestication can occur at single-locus traits, but in general it is expressed as changes in quantitative traits. We recognize three types of domestication selection: (1) intentional or artificial selection, (2) biased sampling during some stage of culture, and (3) unintentional selection. In practice, it may be nearly impossible to distinguish and control these separately.

Artificial selection is the deliberate effort to alter a population to suit management needs, such as development of rainbow trout stocks with specific spawning timing (e.g., Busack and Gall 1980). Artificial selection becomes a hazard when fish to be released into the wild perform well in the hatchery but poorly in the wild because of divergence from their source population at the intended traits or because of correlated changes in other traits. Additionally, if hatchery fish survive to reproduce in the wild, performance of the wild population may be reduced by outbreeding depression.

Biased sampling originates more from error than intent. It can happen during any stage of hatchery operation where genetic variability might be excluded. For example, a common source of biased sampling is broodstock collection. Ideally fish are chosen randomly. More often, however, fish are chosen to represent the distribution of spawning timing, size, age, or some other trait of the source population. If sampling errors are random or involve traits that do not respond strongly to selection, little or no genetic change results. But if sampling errors are systematic and involve traits that respond easily to selection, variability is lost. The potential for genetic change in hatchery operations because of sampling error has been demonstrated by Leary et al. (1986), who found that electrophoretically detectable allele frequencies in a rainbow trout hatchery stock varied over the course of a spawning season.

Unintentional selection is genetic change that results from uncontrolled differences in reproductive success imposed by the hatchery environment and rearing regimes. The fundamental reason for operating hatcheries is to achieve a survival advantage by altering the environment. Fish in hatchery environments may be exposed to higher densities or different food, drift, flow, substrate, protective structure, photoperiod, and so on. These changes in environment allow more fish to survive in the hatchery than survive in the wild, but they also produce the opportunity for genetic change.

The biggest obstacle to serious consideration of the hazard of domestication is a tenacious belief that hatcheries can not impose selection simply because they allow so many fish to survive. Rather than impose selection, the reasoning goes, they release fish from it. This is genetically naive for three reasons. First, what is important is survival to reproductive age, not juvenile survival. Mortality rates of stocked and wild fish are high. If the offspring of hatchery fish that survive to reproduce differ genetically from those in the wild, selection has occurred. Second, death of less fit individuals is not a prerequisite for selection. All that is required is for some genotypes to leave more adult offspring than others. If reproductive potentials among genotypes differ because of the hatchery environment, domestication selection has occurred. This fact points to the third flaw in the "benign environment" notion. Hatcheries may release fish from many of the selection pressures they would have encountered in nature, but this shift in the relative fitness of genotypes will also cause selection to occur. For example, release from competition for mates may be changing expression of secondary sexual characteristics in coho salmon *O. kisutch* (Fleming and Gross 1989). In summary, a natural population continually receiving hatchery introductions is essentially being simultaneously selected for performance in two different environments, with the possible outcome being reduced fitness in the wild. Consequently, potential exists for hatchery-dependent populations in which the spawner–recruit relationship is reduced by domestication to the point where populations are no longer self-sustaining in the wild.

As with the other types of genetic hazard, theoretical argument exceeds empirical evidence. This is not surprising. Domestication selection is measurable in quantitative rather than qualitative traits, and it is difficult to separate genetic and environmental effects on the phenotype (Hard 1995, this volume). Many arguments for domestication are based on evidence of selection regimes. Captive propagation of any organism poses very different selection regimes than does the wild (Frankham et al. 1986). Consequently, selective changes are expected to be fairly strong (Kohane and Parsons

1988). Doyle (1983), for example, showed that high selection differentials can easily exist in hatchery environments.

Many studies have demonstrated phenotypic differences between hatchery and wild fish, but in relatively few are the effects clearly genetic. The best study to date is that of Reisenbichler and McIntyre (1977), who compared early survival of a two-generation-old hatchery stock of steelhead (anadromous rainbow trout) with the wild stock from the same stream. Hatchery fish exhibited a statistically significant survival advantage over wild fish in hatchery environments; the situation was reversed in natural environments. Swain and Riddell (1990) noted that hatchery juvenile coho salmon exhibited more agonistic behavior than did wild juveniles. Also, differences in foraging behavior have been noted between wild and hatchery × wild steelhead juveniles (Johnsson and Abrahams 1991).

Unresolved issues.—Of all the issues surrounding genetic hazards and risk, probably the most controversial is domestication selection, because it strikes at the heart of hatchery technology. Hatchery situations can be envisioned in which the other types of hazards are controlled. Complete control of domestication, however, would require perfect random sampling of broodfish and eliminating differences between hatchery and natural environments. This is unimaginable. Hatcheries exist because they offer very different environments from nature, which allow higher juvenile survival. Like other hazards, the main issues for managing domestication are whether we know enough about biological thresholds to manage based on performance of the fish, and whether we can control for possible sources of loss of genetic diversity without such information.

Lack of empirical data on domestication is a major problem. If hazards in hatcheries are to be managed based on performance measures (e.g., survival rate to harvest and fecundity), there are no standards by which to monitor the risks and, consequently, little incentive to consider it. We believe domestication should be considered a ubiquitous phenomenon in hatchery operations until it is shown otherwise. It is one of the costs of using hatcheries. The challenge is to learn enough about the types and magnitude of the changes in hatcheries so that both short-term and long-term costs can be understood.

There are ways to reduce domestication. The way to reduce the intentional selection component is obvious: stop artificial selection or stop using the selected stock. The problem is that hatchery managers can only stop artificial selection of which they are aware. For example, using only the early spawners is obviously artificial selection and can be eliminated; but how much artificial selection results from routine culling that occurs during hatchery rearing?

Control of domestication due to biased sampling depends on the ability to incorporate random sampling into hatchery procedures and the kind of traits that are important. True random sampling is virtually impossible, however. Rigorous sampling methods can be developed for easily observed and readily measured traits, but random sampling for many traits of a population is impossible. Thus, some loss of diversity due to sampling seems inevitable. Intentional gene flow from wild populations might reduce this loss.

There are two obvious ways to reduce unintentional selection in hatcheries. First, selection potentials can be decreased by minimizing the time fish are exposed to the hatchery environment. For example, only wild fish can be used as broodstock so that hatchery fish are regularly cycled through the natural environment and the proportion of hatchery fish on the spawning grounds can be limited (Anonymous 1990b). Second, hatchery environments can be made more similar to the wild without loss of efficiency (Maynard et al. 1995, this volume). Recently Allendorf (1993) has suggested a third method that is applicable only in pedigreed populations: reducing selection potentials by equalizing family size.

The Future of Genetic Risk Management

Many fisheries scientists have concluded that the empirical data do not support current concerns about genetic risks and hazards. We disagree with such a conclusion. We are unaware of rigorous research designed to detect genetic impacts that has failed to find them. Such data would be very important. The data that do exist support current concerns.

Clearly, we need more research into genetic risk (see also Campton 1995, this volume). The single greatest need is a rigorous treatment of outbreeding depression, but work on domestication selection runs a close second. We also need studies of the effect of reducing effective population size from optimal to various lower, but not pathologically low, levels. For all three areas, we need to understand not only immediate, short-term consequences, but also recovery time from genetic impacts. Ideally these effects would be studied in the species of greatest management interest. However, con-

straints of money and time will make this very difficult in some species, such as Pacific salmon. In addition, legal protection may preclude research on certain species. Thus, we encourage research on small "laboratory" species (e.g., Leberg 1990, 1992). Although data collected from carefully controlled experiments is most desirable, other avenues of obtaining information should not be overlooked. Large-scale management research, perhaps via adaptive management (Walters and Hilborn 1978), is another alternative. Genetic monitoring of hatchery programs will also provide valuable information. Theoretical work, including modeling, is vital, both to provide new information and to provide management with some guidance in the absence of empirical data.

Although we enthusiastically support all these approaches to reducing the uncertainties surrounding genetic risk, we believe it is unrealistic to expect too much from these efforts. Certainly important illustrative examples will appear, and mechanisms will become better understood. But uncertainty will continue to be a fact of life in managing genetic risks simply because genetic impacts are often a function of chance. One of the biggest unknowns in predicting the magnitude of a genetic impact is the genetic composition of the population(s) involved. The genetic composition of a population at any given time is a product of its entire history of selection, mutation, gene flow, and drift. Stated more simply, any two fish populations subjected to the same genetic effect can be expected to respond differently. A recent sobering illustration of the dependence of genetic effect on genetic composition is provided by a study of inbreeding depression in mice *Peromyscus* sp. (Brewer et al. 1990). Theory predicts that chronically small populations should be less susceptible to inbreeding depression than are large or recently large ones. When mice from several populations of differing current and recent abundance levels were inbred, the relative levels of inbreeding depression were quite different from those expected.

In summary, we see the current situation in genetic risk management as follows. There are sound theoretical reasons for expecting genetic impacts from many common types of hatchery practices and operations, and the empirical record supports these. More research will make management of genetic risk easier, but there may be real limits to our ability to predict genetic effects. We can expect the relationship between genetic structure and function to become clearer, but certainly not as clear as we would like it to be.

Faced with present uncertainties and the possibility that additional research will not provide all the answers, we believe the only realistic approach to genetic risk management is to manage based on maintaining diversity rather than performance. We do not want to imply that diversity-based management is a second choice approach, however. It is a fundamentally sounder approach because it addresses the source of fitness.

Diversity-based management is not a new idea among conservation biologists (e.g., Meffe 1987; Ryman 1991), but the idea is fairly new to many managers. The basic precept is the same as that of ecosystem conservation—conserve function by conserving diversity (Meffe et al. 1994). Rather than managing to keep performance depressions within acceptable limits, we should manage instead to maintain diversity. Essential elements of such an approach are inventories of genetic diversity and programs to monitor it. These programs may be based on allozyme data, DNA analyses, and studies of quantitative genetic variation. Diversity-based programs would stress keeping effective population sizes high, allowing gene flow among closely related populations only, and minimizing domestication selection. They should also include creation of genetic refuge areas where no hatchery activities take place. These kinds of programs may be expensive or logistically difficult in the short term, but we see them as the only way to protect the productivity and resilience of populations for the future.

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