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Genetic Variability in Chinook Salmon Stocks from the Columbia River Basin

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Abstract.—Levels of allozymic variability at 33 protein loci are reported for juvenile chinook salmon *Oncorhynchus tshawytscha* collected at 28 locations in the Columbia River basin. Fish were classified as spring, summer, or fall run types, depending on time adults reentered the river. Average heterozygosity per sample (H) ranged from 0.023 to 0.097; H over all samples was 0.070 (0.003 SE). On average, fall-run chinook salmon had significantly greater H values than the spring- or summer-run fish. Spring-run chinook salmon from the Snake River had the lowest values of H (mean, 0.044) in relation to other stocks of spring-run fish, i.e., almost 50% less allozymic variability than spring-run chinook salmon in the lower Columbia River. The probable cause for low levels of heterozygosity in these upriver populations is an increased frequency of natural and human-related population bottlenecks. Measures are recommended for hatcheries to maintain effective population sizes and thereby minimize the loss of genetic variability.

Geneticists have long been aware of the importance of genetic variability in animal populations (Lewontin 1974). Whereas a genetically homogeneous population may lack sufficient variability to adapt or evolve, a population with a large degree of genetic variability has a better chance to withstand environmental changes involving, for example, thermal, osmotic, or pathogenic stresses (Allendorf and Leary 1986). Recently, biologists have emphasized the effect of reduced genetic variability on captive or cultured populations of mammals (Ralls and Ballou 1983; O'Brien et al. 1985) and fishes (Allendorf and Phelps 1980; Ryman and Stahl 1980; Cross and King 1983; Leary et al. 1985). Studies of inbreeding of fishes indicate that a loss of genetic variation may be associated with decreased survival, growth, and food conversion efficiency, and increased morphological deviations (Kincaid 1983; Leary et al. 1985). It is important to know the amount of genetic variation in populations of fish if appropriate management and conservation programs are to be designed.

Genetic research at the Northwest Fisheries Center (Seattle) of the National Marine Fisheries Service has focused on describing the genetic population structure of Pacific salmon and trout *Oncorhynchus* spp. by the technique of protein gel electrophoresis (Utter et al. 1980). The allozymic data set has been used primarily to identify the major wild and hatchery populations that contribute to oceanic and riverine fisheries (Milner et al. 1985). In 1982, chinook salmon *O. tshawytscha* from 28 locations in the Columbia River basin were surveyed for allozymic variation at 33 protein-coding loci. This paper summarizes the results of this survey with respect to the levels of genetic variability. Stocks of spring-run chinook salmon from the Snake River are emphasized because of their historical importance in the basin (Williams 1989).

Methods

Thirty-two samples of juvenile chinook salmon were netted from hatcheries or streams at 28 locales in the Columbia River basin in 1982 (Figure 1). Fish in each sample were classified as either spring, summer, or fall run type depending on the time adults reentered the Columbia River; four localities were represented by two run types (Table 1). This traditional classification does not necessarily imply that a specific run type constitutes a cohesive genetic unit. Extracts of muscle, heart, liver, and eye were taken from each fish and immediately frozen on dry ice until they could be stored at −80°C in the laboratory. Allozymic variation was determined at 33 protein-coding loci in approximately 50 fish per sample by standard electrophoretic techniques (Aebersold et al. 1987). Electrophoretic conditions and allelic data are presented in Table 2. The average heterozygosity over all loci examined (H) was used as a measure of the overall genetic variability in a sample (Nei 1975). For statistical analyses, the H-values were arcsine-transformed (Sokal and Rohlf 1969; e.g., see Simon and Archie 1985).

Results

Considerable allelic variation was detected in the samples of chinook salmon. The majority of loci had two or three allelic variants; the modal

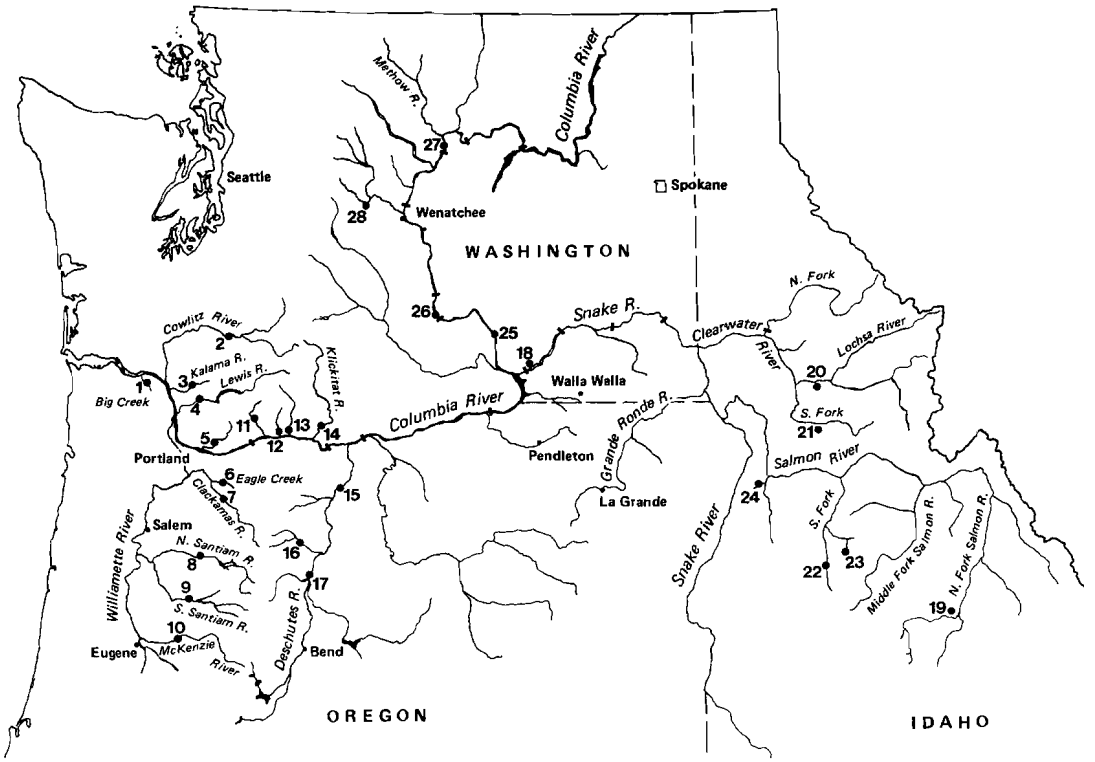

FIGURE 1.—Sampling locations in the Columbia River drainage. Locales are indicated by numbers corresponding to the list in Table 1. Dams are indicated as bars. Samples 1–5 are from the lower Columbia River, 6–10 are from the Willamette River, 11–17 are from the middle Columbia River, 18–24 are from the Snake River, and 25–28 are from the upper Columbia River.

value was two alleles (Table 2). Most of the allelic variation was rare, however; approximately one-third of the loci were characterized by a common allele with two or three rare alleles (i.e., three or fewer heterozygotes per sample). No allelic variation was detected at three loci, and five loci had four alleles. The largest heterozygosity values were associated with six loci: aconitate hydratase (*Ah*), isocitrate dehydrogenase (*Idh-3,4*), mannose-6-phosphate isomerase (*Mpi*), superoxide dismutase (*Sod-1*), and glutathione reductase (*Gr*). Although these six loci were principally two-allele loci, up to 40–50% of the individuals in a sample were heterozygous at one or more of these loci.

Over all the loci, average heterozygosity (H) ranged almost fivefold from 0.023 in fish from Johnson Creek to 0.097 at Wells Dam (Table 1). The mean H value over the 32 samples was 0.070 (±0.003 SE). The differences between the run types, i.e., spring-run (N = 17), summer-run (N = 3), and fall-run (N = 12) fish, were statistically compared in a Duncan's multiple-range test. Average heterozygosity values for summer-run (H = 0.053) and spring-run (H = 0.065) fish were not significantly different, but both were significantly lower ($P < 0.05$) than values from fall-run fish (H = 0.081).

Spring-run chinook salmon from the Snake River basin had the lowest H values (mean, 0.044; N = 4) in relation to the other spring-run samples (Figure 2). For example, the Snake River stocks had almost 50% lower H values than stocks in the Willamette River group (H = 0.080; N = 4) and fish in the lower Columbia River (H = 0.082; N = 3). Spring-run chinook salmon from the middle and upper Columbia River had an average H of about 0.060.

Discussion

Allozymic studies have been conducted on many plants and animals (Ayala 1976; Nevo 1978; Hamrick et al. 1979). Most recently, Nevo et al. (1984) compiled published information for 183 species of fish and reported an average heterozygosity of 0.051 (±0.004 SE). In comparison, chinook salmon in the Columbia River have above-

TABLE 1.—Information for samples of chinook salmon taken in the Columbia River basin in 1982. The amount of genetic variability in each sample is estimated as average heterozygosity over 33 protein-coding loci. Locations are illustrated in Figure 1.

Location of samples		Timing of adult return	Number of specimens	Heterozygosity (H)
Name	Source			
Lower Columbia River basin[a]				
1. Big Creek	Hatchery	Fall	50	0.074
2. Cowlitz	Hatchery	Spring	50	0.086
	Hatchery	Fall	50	0.074
3. Kalama	Hatchery	Spring	50	0.078
	Hatchery	Fall	50	0.080
4. Lewis	Hatchery	Spring	50	0.082
	Hatchery	Fall	50	0.093
5. Washougal	Hatchery	Fall	50	0.086
Willamette River				
6. Eagle Creek	Hatchery	Spring	50	0.076
7. Clackamas	Hatchery	Spring	66	0.091
8. North Santiam	Hatchery	Spring	25	0.075
9. South Santiam	Hatchery	Spring	40	0.076
10. McKenzie	Hatchery	Fall	38	0.084
Middle Columbia River basin[b]				
11. Carson	Hatchery	Spring	50	0.053
12. Little White Salmon	Hatchery	Spring	48	0.051
	Hatchery	Fall	50	0.078
13. Spring Creek	Hatchery	Fall	50	0.074
14. Klickitat	Hatchery	Spring	50	0.086
15. Deschutes River	Stream	Fall	49	0.072
16. Warm Springs	Stream	Spring	50	0.056
17. Round Buttes	Hatchery	Spring	59	0.052
Snake River				
18. Ice Harbor Dam	Stream	Fall	50	0.077
19. Sawtooth River	Stream	Spring	62	0.043
20. Kooskia	Hatchery	Spring	50	0.057
21. Red River	Stream	Spring	40	0.036
22. South Fork Salmon River	Stream	Summer	50	0.039
23. Johnson Creek	Stream	Summer	56	0.023
24. Rapid River	Hatchery	Spring	50	0.042
Upper Columbia River basin[c]				
25. Hanford Reach	Stream	Fall	50	0.085
26. Priest Rapids	Hatchery	Fall	50	0.091
27. Wells Dam	Hatchery	Summer	50	0.097
28. Leavenworth	Hatchery	Spring	50	0.061

[a] Below Bonneville Dam (Bonneville Dam is approximately at the crest of the Cascade Mountains).

[b] Between Bonneville Dam and the confluence of the Snake River and Columbia River.

[c] Upriver of the confluence of the Snake River and Columbia River.

TABLE 2.—Enzymes and electrophoretic conditions for genetic studies of chinook salmon. Allelic variability at the isoloci (designated with 1,2 or 3,4) was regarded as originating from one locus.

Protein name and number (IUBNC 1984)	Locus	Number of alleles	Tissue[a]	Buffer system[b]
Aconitate hydratase (4.2.1.3)	*Ah*	4	L	2
Adenosine deaminase (3.5.4.4)	*Ada-1*	2	E,H,M	1
	Ada-2	1	E,H,M	1
Aspartate aminotransferase (2.6.1.1)	*Aat-1,2*	3	H,M	1
	Aat-3	3	E	1
Creatine kinase (2.7.3.2)	*Ck-1*	4	M	3
	Ck-2	1	M	3
Dipeptidase (3.4.13.11)	*Dpep-1*	2	E,H,M	1,3
	Dpep-2	2	E	1,3
Fumarate hydratase (4.2.1.2)	*Fh*	1	M	2
Glucose-6-phosphate isomerase (5.3.1.9)	*Gpi-1*	3[c]	M	3
	Gpi-2	2	M	3
	Gpi-3	3	M	3
Glutathione reductase (1.6.4.2)	*Gr*	2	L,H	2
Hydroxyacylglutathione hydrolase (3.1.2.6)	*Hagh*	2	L	1
Isocitrate dehydrogenase (1.1.1.42)	*Idh-3,4*	4	E,L,H,M	2
Lactate dehydrogenase (1.1.1.27)	*Ldh-3*	2	E,H,M	3
	Ldh-4	2	E,L,M	1
	Ldh-5	3	E	1
Malate dehydrogenase (1.1.1.37)	*Mdh-1,2*	3	L,H,M	2
	Mdh-3,4	4	E,H,M	2
Mannose-6-phosphate isomerase (5.3.1.8)	*Mpi*	3	E,L,H,M	1
Phosphoglucomutase (5.4.2.2)	*Pgm-1,2*	4	E,L,H,M	2
Phosphogluconate dehydrogenase (1.1.1.44)	*Pgdh*	2	E,M	2
Phosphoglycerate kinase (2.7.2.3)	*Pgk-2*	2	E,L,M	2
Proline dipeptidase (3.4.13.9)	*Pdpep-2*	2	E,M	1
Superoxide dismutase (1.15.1.1)	*Sod-1*	3	L	1
Tripeptide aminopeptidase (3.4.11.4)	*Tapep-1*	2	E,L,H,M	1,3

[a] L = liver; E = eye; H = heart; M = muscle.

[b] 1 = tris (0.18 M), boric acid (0.1 M), and EDTA (0.004 M), after Markert and Faulhaber (1965); 2 = citric acid (0.04 M) adjusted to pH 7.0 with *N*-(3-aminopropyl)-morpholine and EDTA (0.01 M), after Clayton and Tretiak (1972); 3 = tris (0.03 M) and citric acid (0.005 M), pH 8.4, in the gel, and lithium hydroxide (0.06 M), boric acid (0.3 M), and EDTA (0.01 M), pH 8.0, in the electrode trays, after Ridgeway et al. (1970).

[c] A rare polymorphism was detected by a lack of staining activity at the location of the *Gpi-1/Gpi-3* interlocus heteromeric band.

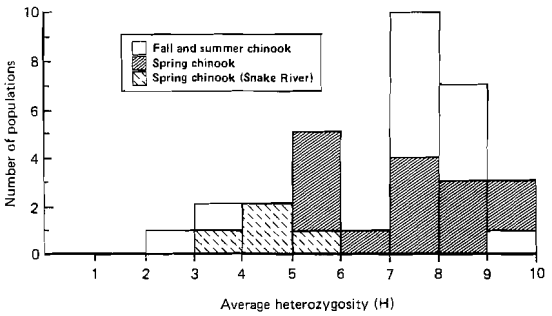

FIGURE 2.—Frequency histogram of average heterozygosity in samples of chinook salmon from the Columbia River basin; the abscissa scale is 100 × H. Heterozygosity values are based on the allozymic variability at 33 loci. The overall mean is $H = 0.070$. The three samples of summer-run chinook salmon occupy both ends of the histogram at $H = 0.023$ and 0.039 (Snake River) and $H = 0.097$ (upper Columbia River); the other run types are as indicated.

average levels of genetic variation among fish. Spring-run and summer-run chinook salmon have statistically less genetic variability than fall-run chinook salmon. Of the 17 samples of spring-run chinook salmon from the five portions of the Columbia River basin, the Snake River samples have the least amount of allozymic variability.

What are the causes of low levels of genetic variability? When only a few individuals effectively contribute gametes to a generation, genetic variation is lost; the fewer the individuals, the faster the variation is lost. This phenomenon, a constriction in effective population size, is referred to as a population genetic bottleneck (Hartl 1980). Population bottlenecks can occur naturally or as a result of human activities. For example, when an anadromous fish population depends on survival of outmigrant smolts in freshwater and returning adults from seawater, various natural meteorological, geological, or biological events can reduce population size in one or both life stages and effect a population bottleneck. Similarly, various human activities can limit population sizes of anadromous fish in the Columbia River through destruction of spawning habitats, hydroelectric development, chemical and thermal pollution, and other consequences (e.g., Damkaer and Dey 1986).

If reduced genetic variability in populations of anadromous chinook salmon is caused primarily by natural or human-related bottlenecks, it follows that the frequency of population bottlenecks is related to the distance populations must move to and from the ocean. Thus, upriver populations of spring-run chinook salmon in the Snake River might have been expected to have lower levels of genetic variability than downstream populations—as seen here. That the two summer-run samples from the Snake River have the lowest values of heterozygosity also supports this theory. The higher values of H in the sample from Ice Harbor Dam (fall-run fish) in the Snake River and the sample at Wells Dam (summer-run fish) in the upper Columbia River contradict this notion—but both values may be artificially high due to the presence of several stocks in our collections.

Genetic variability in a population helps buffer against the vagaries of the environment. Thus, regardless of the cause(s) of low levels of genetic variability in the Snake River populations, the adaptability of these fish is reduced. A general assumption in allozymic investigations is that the set of protein-coding loci is an indicator of the entire genome (Lewontin 1974). Thus, an overall reduction in variability in a set of allozymic loci very likely means there is a corresponding reduction in variability at other loci. A majority of populations of chinook salmon in the Columbia River are maintained in hatcheries and are subject to new selective pressures like stress and disease. It is valuable, therefore, to consider whether decreased allozymic variability in selected populations is related to decreased genetic variability at other loci that may directly affect the susceptibility of these fish to these hatchery-related pressures.

A recent study of cheetahs *Acinonyx jubatus* illustrated a correlation between allozymic loci and immunogenetic loci—and the suspected relationship to disease resistance (O'Brien et al. 1985). The investigators detected no allozymic variability at 52 loci in captive populations of cheetahs. From skin-grafting experiments, they deduced no genetic variability at the major histocompatibility complex (MHC), loci associated with immune reaction. They also reported that these captive populations were highly sensitive to a viral pathogen that did not elicit disease in African lions *Felis leo* and common cats *F. catus*. Based on these observations, they suggested that the susceptibility of captive cheetahs to disease was a consequence of the lack of genetic variability. Although we can not easily extrapolate from these findings to fish—we know little about immune-related loci in fishes—they are a warning. Genetic variability can be associated with the health of a species.

Recent data indicate that environmental stress affects disease susceptibility in salmonids (e.g., Pickering and Pottinger 1985; Maule et al. 1987). Because juvenile outmigration is stressful for the

chinook salmon in the Snake River (Williams 1989), this is an important consideration. Outmigrants encounter up to four hydroelectric facilities in the Snake River basin and four more facilities in the Columbia River. Williams (1989) reviewed smolt transportation research in the Snake River. He pointed out that whether fish bypass or are transported from hydroelectric projects, they are detectably stressed. Preliminary data discussed by Williams indicate that stressed fish suffer high mortality due to bacterial kidney disease, a chronic problem for spring chinook salmon in the Columbia River basin. Williams concluded that highly stressful riverine conditions coupled with a low stress tolerance of Snake River stocks of chinook salmon exacerbate disease problems in these fish and have decreased their overall survival. Currently there are no comparative data relating disease susceptibility among the different stocks within and outside of the Snake River basin.

The effects of bottleneck events can persist for hundreds of generations in the absence of migration (Nei et al. 1975). In natural populations genetic mutations and gene flow among neighboring populations replenish genetic variation within populations if large effective breeding populations are maintained. The maintenance of chinook salmon in the Snake River basin is becoming dependent on hatcheries (Williams 1989); thus management practices must address this issue. Hatchery managers can follow these simple recommendations to maintain effective population sizes: (1) use all temporal segments of a run; (2) use individuals of all shapes, sizes, and ages, i.e., do not select by eye the most "fit" fish; (3) use equal numbers of males and females in pairwise spawning (1 male : 1 female) (Allendorf and Ryman 1987). The erosion of genetic variability is minimal with effective numbers of at least 100 males and 100 females.

An alternative management approach can be considered. Artificial, or human-induced gene flow could also be used to check the erosion of variability. This approach could theoretically be as simple as introducing one individual per population per generation (see Allendorf and Phelps 1981). To preserve the genetic integrity and characteristics of the population, donors should be preadapted fish from adjacent populations that are genetically and ecologically similar to the host population (Krueger et al. 1981). In actuality, a management program incorporating artificial gene flow is complex and involves problems such as the selection of donor populations, the frequency and intensity of gene flow, and population monitoring. To consider gene transfer as a management plan, experimental data are required.

Reasons for success or failure in the culture of anadromous populations of chinook salmon do not have to be obscure. Allozymic information can support the formulation of genetically sound management programs (see Soule and Wilcox 1980; Soule 1986; Ryman and Utter 1987). This study demonstrates another case in which allozymic data can be used to identify genetically depauperate populations. These types of genetic data, alone or in conjunction with correlated character sets (e.g., response to skin transplants, O'Brien et al. 1985; asymmetry of meristic traits, Leary et al. 1985; variability in monoclonal antibodies, Lundstrom 1987), are recommended for intelligent genetic management of anadromous salmon. To persist through geological time, a species may draw on genetic variability found among extant populations. Fish conservationists and managers must recognize that continual loss of genetic variability will result in reduced fitness and possible extinction of unique and valuable genetic races of chinook salmon.

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