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Abstract.— We analyzed the protein products of 78 isozyme loci in 37 populations of chinook salmon *Oncorhynchus tshawytscha* from California and Oregon. Allele frequencies at 47 polymorphic loci revealed substantial genetic variability within the study area. The collections of chinook salmon studied could be differentiated into five major groups located in the following geographical areas: (1) Smith River–Southern Oregon area, (2) Middle Oregon Rivers, (3) Klamath–Trinity Basin, (4) Eel River–California Coastal area, and (5) Sacramento–San Joaquin Basin. Average heterozygosity estimates were lowest in collections from the Klamath–Trinity area and highest in the Oregon populations. Gene diversity analysis indicated that differences among fish within samples accounted for 89.4% of the total diversity, whereas intersample differences accounted for 10.6%. Estimates of the average level of historical gene flow between populations ranged from 15.57 migrants per generation in the Sacramento–San Joaquin River system to 3.97 in the Klamath–Trinity Basin; an overall estimate of number of salmon exchanging genes between populations per generation was 2.11. Although these data appeared to reflect primarily population structures existing prior to the 20th century, evidence of some effects of hatchery management and transplantations was detected.

Geographic variation in population genetic structure of chinook salmon from California and Oregon

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Chinook salmon *Oncorhynchus tshawytscha* is the most abundant and commercially important species of Pacific salmon native to California and Oregon (Moyle 1976), but stocks have declined (Netboy 1974), in some cases to near extinction. Efforts to manage and preserve the chinook fishery have involved traditional methods such as tag and recapture estimations and restrictive fishing regulations. Recently, however, population genetic analysis of Pacific salmon has emerged as a major tool in fishery management to estimate population subdivision, migration, gene flow, and stock composition of ocean fisheries (Ryman and Utter 1987).

Genetic studies on chinook salmon have refined our understanding of these populations. Examination of large numbers of polymorphic loci revealed geographic associations among populations of chinook salmon (Gharrett et al. 1987, Utter et al. 1989, Bartley and Gall 1990, Shaklee

et al. 1990b). Genetic differences among chinook salmon stocks from different geographic areas are being used to identify the stock composition of mixed ocean salmon fisheries (Pella and Milner 1987, Utter et al. 1987, Shaklee et al. 1990b, Brodziak et al. 1992). In addition, genetic studies have indicated the effects of climate and geological events on the population structure of chinook salmon (Gharrett et al. 1987, Bartley and Gall 1990).

Utter et al. (1989) and Bartley and Gall (1990) recently described California populations of chinook salmon using data sets with 53 isozyme loci for 35 populations, and 25 polymorphic loci for eight populations, respectively. The objectives of the study reported here were to further refine the description of chinook salmon populations in California and southern Oregon, expand the baseline genetic data available for genetic stock-identification studies (Shaklee et al. 1990b, Brodziak et al. 1992),

and provide estimates for heterozygosity, allele frequencies, and genetic identities as used for optimum estimation of stock composition of mixed fisheries.

Materials and methods

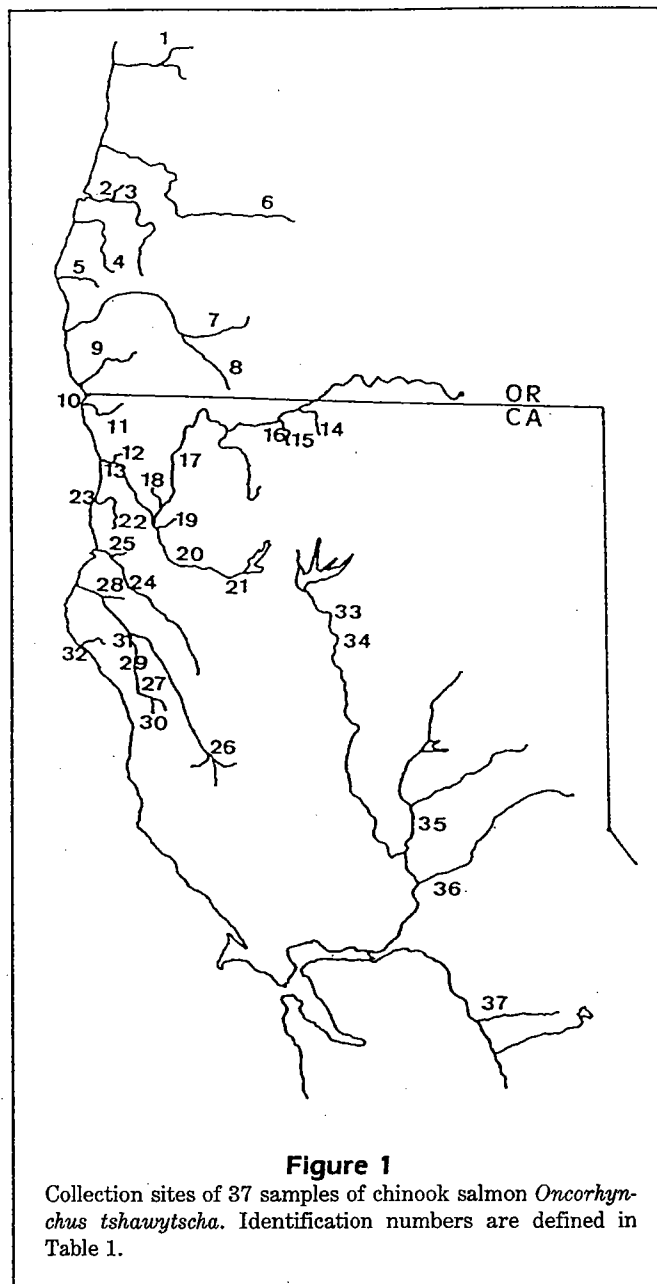
Samples

A total 37 samples of juvenile chinook salmon were collected from northern California and southern Oregon during 1987–88 (Fig. 1, Table 1). Fifteen of these samples were from fish hatcheries and pond rearing projects. All the samples represented fall-run fish with the exception of the upper Sacramento sample (#33) which represented winter run salmon. To collect out-migrant chinook salmon from the wild, two fyke nets (1.5 × 2.1 × 15 m) were placed in a stream approximately 1.6 km apart and allowed to set overnight. Juvenile salmon were removed from the nets the following morning and frozen on dry ice. Juvenile chinook from hatcheries were collected with dip nets. A small number of salmon was taken from each raceway that contained salmon until a total of 200 fish was collected. At the laboratory, liver, muscle, heart, and eye tissue were removed from 100 fish from each collection, placed in individual tubes, and stored at -80°C . The remaining 100 salmon were frozen at -80°C in an archival collection.

Electrophoresis

Tissue preparation and horizontal starch-gel electrophoresis followed standard procedures (Aebersold et al. 1987). Gels were made with 12% hydrolyzed potato starch (Connaught Labs.) and one of the following buffer solutions: CAM, an amine citrate buffer from Clayton and Tretiak (1972) adjusted to pH 6.8; TBCL, the discontinuous buffer system of Ridgway et al. (1970) at pH 8.0; TC-4, a Tris citrate buffer of 0.223 M Tris, 0.083 M citric acid pH 5.8 as electrode buffer, and a 3.7% mixture of buffer in distilled water for the gel (Schaal and Anderson 1974); and TG, a Tris glycine buffer of 0.025 M Tris and 0.192 M glycine pH 8.5 for both gel and electrode buffers (Holmes and Masters 1970). The protein systems analyzed, locus designations, tissue distribution of isozymes, and buffer systems used are presented in Table 2. Because of recent changes in genetic nomenclature (Shaklee et al. 1990a), other locus name synonyms are presented in Table 2 to facilitate comparisons with other studies. Allele designations followed Allendorf and Utter (1979).

Histochemical staining procedures followed Shaw and Prasad (1970) and Harris and Hopkinson (1976). The data set described herein constitutes baseline data



reported in Gall et al. (1989) and used in maximum-likelihood estimates for the California mixed ocean salmon fishery (Brodziak et al. 1992). The duplicated isoloci AAT-1,2, IDH-3,4, MDH-1,2, MDH-3,4, and PGM-3,4 each were treated as two loci. Variant alleles were preferentially assigned to one locus, whereas common alleles were assigned to the other (Gharrett et al. 1987). Variation at the IDH-3,4 isoloci was ascribed to specific loci as described by Shaklee et al. (1990b). Our method of scoring isoloci is not the method of choice for studies of genetic mechanisms, as it may not reflect the true genetic distribution of alleles

Table 1

Thirty-seven collections of juvenile chinook salmon from five areas of California and Oregon. Locations of collections are designated on Figure 1 by identification number (ID#). *N* = number of fish analyzed.

Area	ID#	Collection site	<i>N</i>	No. of loci scored	Average heterozygosity (Nei 1973)
Middle Oregon	1	Fall Creek Hatchery	100	78	0.072
	2	Morgan Creek Hatchery	10	78	0.076
	3	Millacoma River	100	78	0.072
	4	Coquille River, South Fork	100	78	0.073
	5	Elk River Hatchery	100	78	0.076
	6	Rock Creek Hatchery	100	78	0.054
S. Oregon/N. California Coastal	7	Rogue River	100	78	0.052
	8	Applegate River	100	78	0.054
	9	Chetco River Hatchery	100	78	0.063
	10	Rowdy Creek Hatchery	62	77	0.067
	11	Smith River, Middle Fork	99	77	0.059
Klamath-Trinity Basin	12	Blue Creek	100	77	0.059
	13	Omagar Creek Pond-Rearing Facility	100	78	0.064
	14	Irongate Hatchery	99	78	0.031
	15	Bogus Creek	128	77	0.030
	16	Shasta River	100	77	0.028
	17	Salmon River	98	76	0.038
	18	Camp Creek Pond-Rearing Facility	100	77	0.044
	19	Horse Linto Creek	100	77	0.045
	20	Trinity River, South Fork	100	77	0.039
	21	Trinity River Hatchery	120	77	0.030
Eel River-California Coastal	22	Redwood Creek at Orick	95	77	0.050
	23	Redwood Creek Lagoon	100	77	0.054
	24	Mad River Hatchery	99	77	0.045
	25	Mad River, North Fork	61	77	0.054
	26	Eel River, Middle Fork	95	76	0.043
	27	Eel River, South Fork	99	78	0.048
	28	Van Duzen River	100	77	0.050
	29	Redwood Creek, South Fork Eel	93	77	0.046
	30	Hollow Tree Creek	100	78	0.045
	31	Salmon Creek, South Fork Eel	96	77	0.044
	32	Mattole River	100	77	0.049
	Sacramento-San Joaquin	33	Upper Sacramento River	94	77
34		Coleman Hatchery	100	77	0.063
35		Feather River Hatchery	100	78	0.061
36		Nimbus Hatchery	100	78	0.064
37		Merced River Hatchery	100	78	0.057

(Allendorf and Thorgaard 1984, Waples 1988). However, our method of scoring increases the power of maximum-likelihood estimates of stock composition by equalizing the importance of variant alleles at isoloci and non-duplicated loci. Furthermore, our system was maintained for consistency with other research (Gall et al. 1989, Brodziak et al. 1992).

A missing heteromeric isozyme between GPI-1 and GPI-3 was observed in some fish. We scored this pattern, as described in Bartley and Gall (1990), by assigning variation to an artificial locus named GPI-H and

labeling the common and variant alleles *Gpi-H*(100) and *Gpi-H*(*), respectively. However, Utter et al. (1989) described breeding data that indicated the variation should be assigned to either GPI-1 or GPI-3.

Due to the difficulty of identifying heterozygote banding patterns from GPI-H, LDH-1, and MDHP-2, allele frequencies at these loci were calculated from the square root of the frequency of the alternate homozygote. The frequency of the *Tpi-3*(106) allele also was calculated from the square root of the frequency of the homozygous *Tpi-3*(106) pattern.

Table 2

Enzyme systems, IUBNC enzyme number, isozyme loci, buffer systems, and tissues used in electrophoretic analyses of chinook salmon. For loci, m = mitochondrial. M = muscle, H = heart, L = liver, E = eye. Buffers explained in the text. Locus designations (synonyms) are locus names used by (1) present study, (2) Bartley and Gall (1990), (3) American Fisheries Society (Shaklee et al. 1990a), and (4) Utter et al. (1989).

Enzyme name	Enzyme no.	Locus designations				Tissue	Buffer
		1	2	3	4		
Aspartate aminotransferase	2.6.1.1	AAT-1	AAT-1	<i>sAAT-1,2*</i>	Aat-1,2	M, H	TC-4
		AAT-2	AAT-2			M, H	TC-4
		AAT-3		<i>sAAT-3*</i>	Aat-3	E	TC-4
		AAT-4	AAT-3	<i>sAAT-4*</i>		L	TC-4
		mAAT-1		<i>mAAT-1*</i>		M, H	CAM
		mAAT-2		<i>mAAT-2*</i>		M, H, L	CAM, TC-4
		mAAT-3		<i>mAAT-3*</i>	M, H	CAM, TC-4	
Acid phosphatase	3.1.3.2	ACP-1		<i>ACP-1*</i>		M, L	CAM
		ACP-2		<i>ACP-2*</i>		M	CAM
Adenosine deaminase	3.5.3.3	ADA-1		<i>ADA-1*</i>		M	TG
		ADA-2		<i>ADA-2*</i>		M	TG
Alcohol dehydrogenase	1.1.1.1	ADH	ADH	<i>ADH*</i>		L	TC-4, TBCL
Aconitate hydratase	4.2.1.1	AH-1	AH	<i>sAH*</i>		L, M, E	CAM, TC-4
		mAH-1		<i>mAH-1*</i>		E, H	CAM
		mAH-2		<i>mAH-2*</i>		E, H	CAM
		mAH-3		<i>mAH-3*</i>		M, H	CAM
		mAH-4		<i>mAH-4*</i>		M, H	CAM
Alanine aminotransferase	2.6.1.2	ALAT		<i>ALAT*</i>		M	TG
Creatine kinase	2.7.3.2	CK-1	CK-1	<i>CK-A1*</i>		M	TBCL, CAM
		CK-2	CK-2	<i>CK-A2*</i>		M	TBCL, CAM
		CK-4	CK-3	<i>CK-A2*</i>		E	CAM
Esterase	3.1.1.1	EST-3		<i>EST-D*</i>		M, E	TG, TBCL
Fructose-biphosphate aldolase	4.1.2.13	FBALD-4	FBA	<i>FBALD-4*</i>		E	CAM, TC-4
Fumarate hydratase	4.2.1.2	FH	FH	<i>FH*</i>		M	CAM
Glycerol-3-phosphate dehydrogenase	1.1.1.8	G3PDH-1	GPDH-1	<i>G3PDH-1*</i>		M	CAM, TC-4
		G3PDH-2	GPDH-2	<i>G3PDH-2*</i>		M	CAM, TC-4
		G3PDH-3	GPDH-3	<i>G3PDH-3*</i>		M	CAM, TC-4
		G3PDH-4	GPDH-4	<i>G3PDH-4*</i>		M	CAM, TC-4
Glyceraldehyde-3-phosphate dehydrogenase	1.2.1.12	GAPDH-5	GAPDH-3	<i>GAPDH-5*</i>		E	CAM, TC-4
		GAPDH-6	GAPDH-4	<i>GAPDH-6*</i>		E	CAM, TC-4
Glucose-6-phosphate isomerase	5.3.1.9	GPI-1	GPI-1	<i>GPI-B1*</i>	Gpi-1	M	TG, TBCL
		GPI-2	GPI-2	<i>GPI-B2*</i>	Gpi-2	M	TG, TBCL
		GPI-3	GPI-3	<i>GPI-A*</i>	Gpi-3	M, E	TG, TBCL
		GPI-H	GPI-H	<i>GPIr*</i>	Gpi-1	M	TG, TBCL
Glutathione reductase	1.6.4.2	GR	GR	<i>GR*</i>	Gr	M, E, L	TG, TBCL
β -Glucuronidase	3.2.1.31	GUS		<i>GUS*</i>		M	CAM, TC-4
Hydroacylglutathionine hydrolase	3.1.2.6	HAGH		<i>HAGH*</i>		L, M, E	TG
L-Iditol dehydrogenase	1.1.1.14	IDDH-1	IDDH-1	<i>IDDH-1*</i>		L	TBCL
		IDDH-2	IDDH-2	<i>IDDH-2*</i>		L	TBCL
Isocitrate dehydrogenase	1.1.1.42	IDH-1	IDH-1	<i>mIDHP-1*</i>		M	CAM
		IDH-2	IDH-2	<i>mIDHP-2*</i>		M	CAM
		IDH-3	IDH-3	<i>sIDHP-1*</i>	Idh-3, 4	M, E, L	CAM, TC-4
		IDH-4	IDH-4	<i>sIDHP-2*</i>		E, L	CAM, TC-4
L-Lactate dehydrogenase	1.1.1.27	LDH-1	LDH-1	<i>LDH-A1*</i>		M	TBCL, TC-4
		LDH-2	LDH-2	<i>LDH-A2*</i>		M	TBCL, TC-4
		LDH-3	LDH-3	<i>LDH-B1*</i>		H, E	TBCL, TC-4
		LDH-4	LDH-4	<i>LDH-B2*</i>	Ldh-4	L, E	TC-4
		LDH-5	LDH-5	<i>LDH-C*</i>	Ldh-5	E	TC-4
α -Mannosidase	3.2.1.24	MAN	MAN	<i>αMAN*</i>		L	TC-4

Table 2 (continued)

Enzyme name	Enzyme no.	Locus designations				Tissue	Buffer
		1	2	3	4		
Malate dehydrogenase (NADP)	1.1.1.40	MDHP-1		<i>sMEP-1*</i>		M	TC-4
		MDHP-2		<i>sMEP-2*</i>		M, E, L	TC-4
		mMDHP-1		<i>mMEP*</i>		M	TC-4
Malate dehydrogenase (NAD)	1.1.1.37	MDH-1	MDH-1	<i>sMDH-A1,2*</i>	Mdh-1,2	E, M	TC-4
		MDH-2	MDH-2			E, M	TC-4
		MDH-3	MDH-3	<i>sMDH-B1,2*</i>	Mdh-3, 4	M, E	CAM, TC-4
		MDH-4	MDH-4			M, E	CAM, TC-4
		mMDH-1		<i>mMDH-1*</i>		M, E	CAM
		mMDH-2		<i>mMDH-2*</i>		M, H	CAM
Mannose-6-phosphate isomerase	5.3.1.8	MPI	MPI	<i>MPI*</i>	Mpi	E, M, L	CAM
Phosphogluconate dehydrogenase	1.1.1.44	PGDH	PGDH	<i>PGDH*</i>		M, E, L	TC-4
Phosphoglucokinase	2.7.2.3	PGK-1		<i>PGK-1*</i>		L	CAM
		PGK-2	PGK-2	<i>PGK-2*</i>	Pgk-2	M, E, L	CAM
Phosphoglucosmutase	5.4.2.2	PGM-1	PGM-1	<i>PGM-1*</i>	Pgm-1,2	M, E	CAM
		PGM-2	PGM-2	<i>PGM-2*</i>		M, E, L	TG, TC-4
		PGM-3		<i>PGM-3,4*</i>		E, L, M	TG, TC-4
		PGM-4				E, L, M	TC-4
Pyruvate kinase	2.1.7.40	PK-1	PK-1	<i>PK-1*</i>		M	TC-4
		PK-2	PK-2	<i>PK-2*</i>		M	CAM
Superoxide dismutase	1.15.1.1	SOD-1	SOD-1	<i>SOD-1*</i>	Sod	L, M	CAM
		mSOD		<i>mSOD*</i>		H, M, E	TG
Triosphosphate isomerase	5.3. 1.1	TPI-3		<i>TPI-2.1*</i>		E	TC-4
		TPI-4		<i>TPI-2.2*</i>		M, E, L, H	TG, TBCL
β -N-Acetyl-D-glucosaminidase	3.2.1.30	a-GA		<i>βBGLUA*</i>		L	TG, TBCL
Peptidases (substrates)	3.4.*.*						
Glycyl leucine		DPEP-1	PEPA-1	<i>PEP-A*</i>	Dpep-1	M, E, H	CAM, TG
		DPEP-2	PEPA-2	<i>PEP-C*</i>	Dpep-2	E	TG, TBCL
Phenylalanyl proline		PDPEP-2	PDPEP-2	<i>PEP-D2*</i>		M, E	TC-4
Prolyl leucine		PEPLT		<i>PEP-LT*</i>		M	TG
Leucylglycyl glycine		TAPEP	PEPB	<i>PEP-B1*</i>	Tapep-1	M, E	TBCL, TG

Analyses

Genetic variability for each collection of salmon was assessed by calculating the frequencies of alleles at each locus and average heterozygosity assuming Hardy-Weinberg proportions (Nei 1973). A locus was considered variable if we observed polymorphism in at least one sample. Analyses were based on a maximum of 78 loci. If a sample was not scored for a particular locus, the locus was retained for analyses involving multiple samples. Deviations from expected Hardy-Weinberg genotypic proportions were tested by chi-square goodness-of-fit tests (Sokal and Rohlf 1981). Variant allele frequencies were pooled so the expected number of genotypes in a given class was always five or greater. Some loci could not be tested for goodness-of-fit because pooling allele frequencies to achieve a minimum class-size reduced the degrees of freedom to zero. In addition, the loci, PGM-3 and PGM-4, were excluded from goodness-of-fit tests due to the arbitrary

nature of assigning variation to a specific locus. GPI-H, LDH-1, and MDHP-2 were excluded because of the method of calculating allele frequencies from the frequency of the alternate homozygotes.

Genetic identities (I) were calculated for each pair of samples (Nei 1972) and a dendrogram was constructed from estimates of I using the unweighted pair-group method (UPGMA) (Sneath and Sokal 1973). Total gene diversity (H_T) was partitioned to estimate within-sample (H_S) and between-sample (D_{ST}) components, and to estimate relative gene diversity ($G_{ST} = D_{ST}/H_T$) (Nei 1973, Chakraborty and Leimar 1987). Total gene diversity was partitioned into three hierarchical levels: panmixia (T), area or drainage (D), and sample (S) based on *a priori* geographic considerations (Table 1).

An estimate of average gene flow was calculated from Wright's (1943) fixation index

$$F_{ST} = 1/(4Nm + 1) \quad (1)$$

where N_m is the average number of migrants exchanging genes per generation. Equation (1) was solved for N_m by setting F_{ST} equal to the relative gene diversity appropriate for the hierarchical level of interest. This formulation provided an estimate of the number of migrant fish exchanging genes among samples per generation under the assumptions of selective neutrality of alleles and Wright's (1943) island model of migration. Slatkin and Barton (1989) discussed the sensitivity of equation (1) relative to various methods of estimating F_{ST} in the presence of selection and alternative population structures, and found it to be fairly robust.

Results

A total of 96 isozyme loci were examined. Thirty-one loci were monomorphic, 47 were categorized as polymorphic (Appendix A), whereas variability of an unknown and undefined nature was detected at 18 loci. Details of genetic polymorphisms not described elsewhere are outlined in Appendix B. The enzyme systems involving the 18 loci for which evidence of probable polymorphisms was detected (not listed in Table 2) and warrant further study included: two adenylate kinase loci, creatine kinase, four fructose biphosphate aldolase loci, four glyceraldehyde-3-phosphate dehydrogenase loci, two beta-galactosidase loci, alpha-glucoside, superoxide dismutase, two peptidase loci, and a highly anodal acromatic band. Because of difficulties defining a genetic model of inheritance, poor band resolution, or incomplete data, these 18 loci were not included in the analyses.

Tests of conformance to Hardy Weinberg genotypic proportions revealed 37 out of 462 cases (8%) of disequilibria. For wild samples of chinook salmon, 13 of 252 tests (5%) revealed disequilibrium, whereas in hatchery samples, 24 of 210 tests (11%) showed non-conformance to Hardy-Weinberg expectations. However, in the Klamath Basin, a higher percentage of disequilibrium was found (13 of 97 cases or 13%) in hatchery and wild samples. The proportion of disequilibrium observed in Klamath and non-Klamath samples was found to be significantly different ($P < 0.05$) when tested for equality by the generalized likelihood-ratio test for binomial data (Larsen and Marx 1981). The proportion of disequilibrium observed in hatchery (including pond rearing programs) and wild chinook salmon populations also was significantly different ($P < 0.05$). The nature of the observed disequilibrium appeared to be random. That is, we did not observe consistent excesses or deficiencies of heterozygotes, nor did we observe specific loci that consistently deviated from Hardy-Weinberg expectations.

Estimates of average heterozygosity ranged from a low value of 0.028 in Shasta River (#16) to a high of 0.076 in the Morgan Creek (#2) and Elk River (#5) hatcheries. The Middle Oregon samples (#1-6) tended to have high estimates of average heterozygosity, whereas values for the Klamath-Trinity samples (#12-21) tended to be lower (Table 1).

Although genetic identity indices between all pairs of samples were greater than 0.982 (data not shown), the geographic distribution of alleles suggested population subdivision within the study area. For example, we found the *Aat-2(85)*, *Aat-3(90)*, *Aat-4(130)*, and *Iddh-1(0)* alleles predominantly in Oregon and north-coastal California (collections 1-11). The *mAh-4(112)*, *Gpi-H(*)*, and *Pgdh(90)* alleles were present mainly in the Sacramento/San Joaquin system (collections 33-37), whereas *Mdhp-1(92)* and *Gpi-2(60)* were less abundant in the Sacramento Basin compared with more northern areas. *Mdhp-2(78)* was a characteristic of the Klamath-Trinity system and a few coastal samples.

Cluster analysis of genetic identities revealed a strong geographic component to the grouping of chinook salmon samples. Five distinct clusters that reflected geographic areas were evident (Fig. 2): (1) Smith River-Southern Oregon rivers, (2) Klamath-Trinity Rivers, (3) Eel River system-California coastal rivers, (4) Middle Oregon rivers, and (5) Sacramento-San Joaquin system. The Smith River (#11) and the Rowdy Creek Hatchery (#10) samples were the most northern samples collected from California. Therefore, it is reasonable that they would be genetically similar to the southern Oregon samples. The sample from the Fall Creek Hatchery (#1) was the only sample from northern Oregon and therefore, appears as an independent cluster. Three samples, Rock Creek Hatchery (#6, middle Oregon), Blue Creek (#12, Klamath-Trinity Basin), and Omagar Creek (#13, Klamath-Trinity Basin), did not cluster in accordance with their geographic location.

Total gene diversity was 0.0620 (H_T) and average sample diversity was 0.0554 (H_S). Therefore, approximately 89.4% of the total genetic diversity was due to intrasample variability and 10.6% was due to intersample variation (Table 3). Further examination of the intersample diversity showed that genetic differences among samples within the five geographic groups identified from the dendrogram (see Table 1) accounted for about 3.2% of the total variation and 7.4% of the total diversity was due to differences between the major geographic areas. Gene diversity analysis for each geographic area treated separately revealed that although the Klamath-Trinity system possessed the lowest total gene diversity for a given area (H_D), relative gene diversity (G_{SD}) for this drainage was high

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