Phenotypic and Genetic Architecture of Juvenile Morphometry in Chinook Salmon

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Juvenile morphology can affect fitness of salmonids in nature, but the genetic basis for morphometry in salmonids is poorly understood. We mated chinook salmon in a half-sib/full-sib breeding design to determine the genetic and environmental components of morphometric variation. We characterized body size and shape in 20 progeny from each of 95 full-sib families by digitizing 30 landmarks on lateral, dorsal, and ventral images of the body form, and estimated genetic and environmental components of variance for 12 truss elements constructed from the landmarks. We then conducted principal component analyses of the phenotypic, genetic, and environmental covariance matrices for these elements. Most (83%) of the phenotypic variation in morphometry was expressed along axes of allometry, shape change during smoltification, and a three-dimensional body shape contrast (head width versus lateral profile depth). All morphometric traits showed substantial additive genetic variation and were highly correlated genetically as well as phenotypically. Principal components of genetic variation in morphometry resembled the phenotypic principal components. The analyses also detected strong environmental effects on body shape and indicated two distinct morphometric types arising from maternal or environmental sources. The results provide evidence for genetic coordination of body size and shape, which would be expected if these characters are tightly coupled developmentally.

Morphometry is an important ecological character in fishes because it can affect reproductive success through the abilities to forage, defend territories, avoid predators, and attract mates. Variation in morphometry can also be a sensitive indicator of stress manifested through congenital and developmental abnormalities. The extent to which morphometric variation is determined by genetic and environmental factors is poorly understood for most fishes, including salmonids (Gjerde and Schaeffer 1989; Martin 1949; Taylor and McPhail 1985). As is the case with other animals, this situation reflects the fact, noted by Atchley et al. (1981), that most studies of morphometry have been descriptive or comparative rather than experimental. Although recent studies are changing this situation (Beacham 1990; Fleming et al. 1994; Rye and Refstie 1995; Swain et al. 1991), a dearth of quantitative information on the inheritance of morphometric variation in salmonids remains.

Characterization of morphometric variation is less straightforward than for most phenotypic traits, and it is difficult to fully capture its variability from conventional measurements (Rohlf 1990). Disagreement exists about the most appropriate ways to evaluate morphometry (Corruccini 1987; Willig and Owen 1987; Willig et al. 1986), but a major advantage of a multivariate approach is its ability to incorporate covariation among morphometric traits directly into the analysis of variation. Kirkpatrick and Heckman (1989) and Kirkpatrick and Lofsvold (1989) argued that morphometric variation, like growth trajectories but unlike most other phenotypic traits including body size, cannot easily be represented by a set of discrete measurements. Much of this variation may be hidden if analyses rely exclusively on univariate variation. If possible, a comprehensive analysis of multivariate morphometry should also take into account developmental aspects of morphometric variation that are likely to have consequences for fitness in the wild.

Anadromous salmonids, which breed in freshwater but spend much of their lives growing at sea, represent a case in which almost no quantitative estimates of the genetic basis of morphometric variation exist. Most available information bearing on inheritance of salmon morphometry comes from a few types of studies. One type involves simple measurements of body size

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and shape taken from populations cultured in captivity under conventional breeding schemes (Beacham 1990; Gjerde and Gjedrem 1984; Gjerde and Schaeffer 1989; Gunnes and Gjedrem 1978, 1981; Rye and Refstie 1995). Another type involves comparison of aspects of body size and shape in populations sampled from or exposed to differing environments (Beacham 1985; Beacham and Murray 1985; Currens et al. 1989; Swain and Holtby 1989; Taylor 1986; Taylor and McPhail 1985). Still another evaluates variability in populations with different cultural histories reared in the same environment (Hjort and Schreck 1982; Riddell and Leggett 1981; Swain et al. 1991). Fleming et al. (1994) reared fish with a common genetic heritage under different experimental environments and evaluated ontogenetic changes in body form. The various approaches taken in these studies have differing strengths with regard to investigating genetic influence on morphometry, but information on the magnitude of this influence has to our knowledge typically been limited to a few conventional measures of morphometric variation, and not necessarily the most appropriate metrics to evaluate possible consequences of variation. Few studies (Currens et al. 1989; Swain et al. 1991) have applied multivariate analyses to morphometric variation in salmonids.

The utility of hypotheses about the inheritance and evolution of salmonid morphometry could be enhanced considerably by quantitative information on the genetic basis of multivariate shape. In this article we estimate the genetic basis of phenotypic variation in juvenile morphometry in a population of chinook salmon (*Oncorhynchus tshawytscha* Walbaum). We construct three trusses (McGlade and Boulding 1986; Strauss and Bookstein 1982; Winans 1984) from images of the body form and measure their components to evaluate variation along all three observed dimensions in underyearling fish at about the time of their major life cycle transition between freshwater and saltwater (smoltification). We then estimate the genetic and environmental components of morphometric variation with a hierarchical breeding design (Falconer 1989). Finally, we analyze the phenotypic, genetic, and environmental correlation matrices for a set of morphometric traits to evaluate their correspondence.

Materials and Methods

Breeding Design

We spawned ocean-type ("fall-run") chinook salmon (Healey 1983) released from Grovers Creek Hatchery near Kingston (Puget Sound), Washington (47°45′N, 122°33′W), as they returned to the hatchery between September 21 and October 31, 1994. Chinook salmon have been produced at this facility by the Suquamish Tribal Fisheries Department since 1981; the department founded its broodstock between 1978 and 1981 from the Washington Department of Fish and Wildlife's Soos Creek (Green River) Hatchery on Puget Sound near Auburn, Washington (47°18′N, 122°11′W). We sampled adults to establish the experiment from 541 males (55% of all returning males) and 225 females (58% of females) returning to the hatchery during the central portion of the run, October 3–21. We sampled adults without regard to observed phenotypic characters through the use of a random numbers table on each spawning date. Over this 3-week period, we mated 30 males and 120 females in a hierarchical half-sib/full-sib breeding design (Falconer 1989); by the time progeny emerged from the incubation substrate and were ready for transfer to rearing tanks, 96 full-sib families were large enough ($N > 1{,}000$) to include in the experiment and permit estimation of genetic and environmental components of phenotypic variation in morphometry. However, one of the males had produced sufficient progeny at initial rearing from only a single mating, so we excluded the data from this family from some tests to facilitate analysis. After this adjustment, 95 full-sib families nested within 29 half-sib families were available for analysis. The modal number of dams per sire was 3, with a mean of 3.3 (range 2–5). Full-sib family size at initial rearing ranged from approximately 1,000 to more than 5,100. We did not assign half- and full-sib families to rearing tanks at random; instead, we placed each full-sib family in an individual rearing tank arranged in a tank cluster assigned to an entire half-sib family to reduce the chance of accidental mixing of half-sib and unrelated individuals. This procedure, however, is likely to increase the environmental correlation of full- and half-sibs (Falconer 1989).

Morphometric Measurements and Analyses

From April 4 to April 26, 1995, we marked 257,093 underyearling chinook salmon representing the 95 families with family-specific coded microwire tags (Jefferts et al. 1963) at the hatchery. During the marking process, we sampled approximately 20 juveniles haphazardly from large dipnet samples of each of the full-sib families and photographed them. At the time of sampling, fish had been growing in 1.8 m^3 culture tanks, each assigned to a single full-sib family, for approximately 12 weeks. We anesthetized each of the 1,900 fish briefly and weighed them to the nearest 0.1 g; we then captured digital 8-bit grayscale images of the dorsal, lateral, and ventral sides from each anesthetized fish with a video camera slaved to a computer. We returned the fish to their tanks of origin after the procedure, which generally took less than 1 min. Once they recovered from anesthesia, fewer than 10 of the 1,900 fish had died within 1 h as a result of the procedure.

To capture lateral images for each anesthetized fish, we pinned fish by their fully extended fins to a porous foam board and indicated the posterior aspect of the neurocranium in the image with a dissecting pin. All images were of the left side. We used a small plexiglas "vee" trough to hold the anesthetized fish for dorsal and ventral images and used a dissecting pin to indicate the anterior insertion of the dorsal fin in the dorsal image and the anterior insertion of the anal fin in the ventral image. A metric ruler placed alongside the fish during the capture of each image at the same plane as the fish's midsection provided a baseline scale. The numbers of distinctive morphological landmarks used in each image to calculate truss network distances were 13 lateral, 6 dorsal, and 9 ventral. The mean lateral dimension (± 1 SE), computed from all interlandmark distances, was 12.29 $\pm$ 0.02 mm ($n = 1{,}877$). Mean (± 1 SE) wet weight was 2.71 $\pm$ 0.01 g ($n = 1{,}873$).

We used Microsoft Optimas software to determine the Cartesian coordinates of the landmarks for each of 1,900 fish images. One of us (J.C.R.) performed all digitizing; measurement error was less than 0.5 mm, based on five replicate measurements from each of eight images, and variation among these measurements accounted for less than 0.0002% of the total from ANOVA ($F < 0.10$, $P > .90$). For each of the three dimensions, we computed distances between landmarks in a truss-network pattern, modified from the method developed by Winans (1984), along the three dimensions. The lateral dimension contained six truss "cells" of interlandmark distances; the dorsal and ventral dimensions contained two and four truss "cells," respectively (Figure 1). After log transformation of all distances, we identified six outliers with Hadi's (1994) multi-

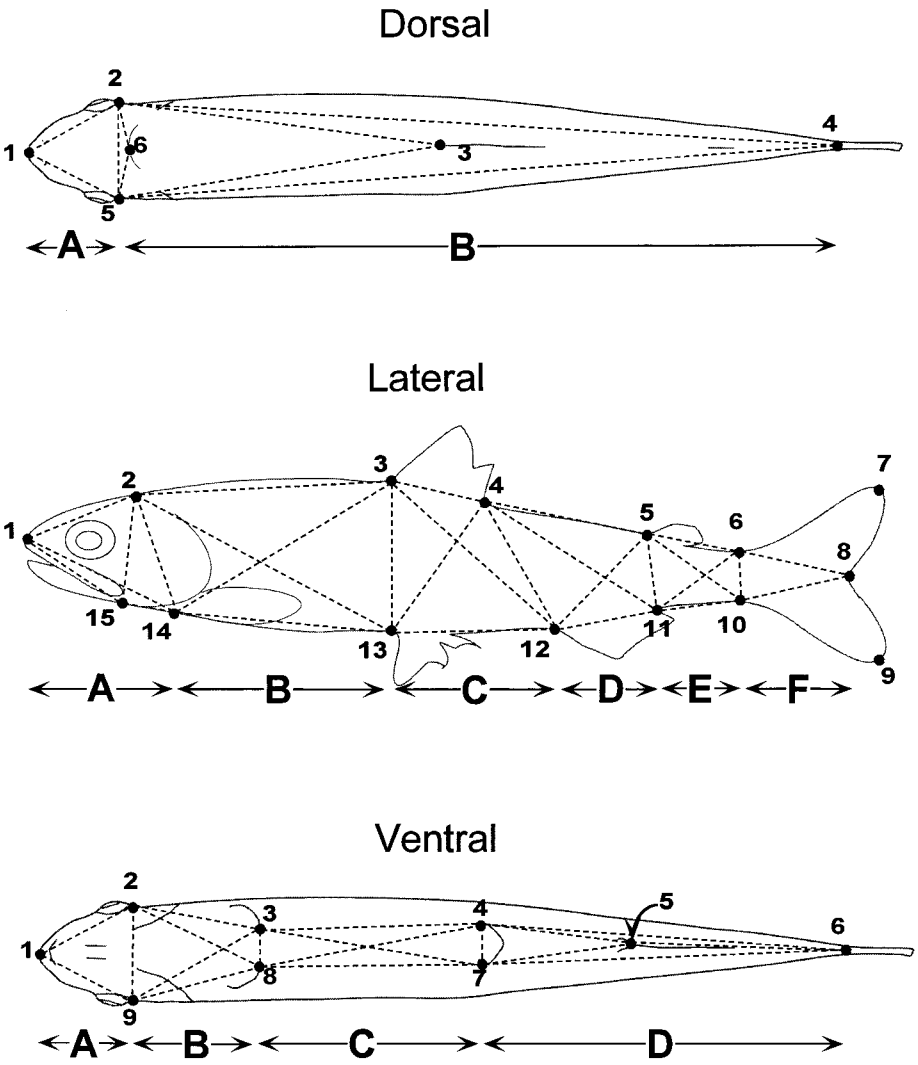


Figure 1. Identification of the 30 dorsal, lateral, and ventral landmarks used to characterize morphometry in a population of ocean-type juvenile chinook salmon. Landmarks are identified by numbers; truss cells (a three- or four-sided polygon represented by landmarks) are designated by letters. Fish were photographed before release to seawater from a hatchery. Dorsal landmarks 3 and 6, lateral landmarks 7, 9, 15, and 16, and ventral landmark 5 were excluded in computing the centroid sizes of each truss cell. Dorsal cells A (D1–2–5) and B (D2–4–5) have centroid sizes csd1 and csd2. Lateral cells A (L1–2–14–15) to F (L6–8–10) have centroid sizes csl1 to csl6, respectively. Ventral cells A (V1–2–9) to D (V4–6–7) have centroid sizes csv1 to csv4, respectively. See text for computation of centroid sizes.

variate detection method, which were removed before analysis. Twelve truss cells in three dimensions, based on a sample size of 1,871, thus constituted the basis for our analysis.

Variance Component Estimation

A preliminary genetic analysis based on a sheared principal components analysis (PCA) (Beeman et al. 1994; Bookstein et al. 1985; Rohlf and Bookstein 1987) of the 55 log-transformed truss distances was unsatisfactory because it led to some infeasible genetic parameter estimates ($h^2 < 0$, $h^2 > 1$, or $|r_A| > 1$) for the composite variables (principal components). Infeasible estimates may arise from sampling error, a large number of trait parameters being estimated, an inappropriate genetic model, or when true parameter values are near feasibility limits (Bridges and Knapp 1987; Lynch and Walsh 1998; Prabhakaran and Jain 1987). The likelihood of infeasibility may also be higher for multivariate vectors than other traits, especially when several vectors are analyzed jointly. An additional problem with this approach is that the validity of genetic correlations (r_A) between phenotypically orthogonal multivariate vectors may be questionable. Nevertheless, genetic analysis of principal components has been used in some recent studies (Diniz-Filho and Malaspina 1994; Diniz-Filho and Pignata 1994; Roff and Bradford 1998).

To circumvent potential problems associated with the genetic analysis of multivariate vectors, we conducted separate PCAs on the phenotypic, genetic, and environmental correlation matrices (Leamy 1977) of a smaller set of morphometric traits (log weight and the geometric sizes of the 12 truss cells). We computed the centroid sizes of each of the six lateral, two dorsal, and four ventral truss cells (Figure 1) from the sum of all squared interlandmark distances (log transformed) in each cell (Bookstein 1991); unlike some workers, we did not take the square root of centroid size. We used a broken-stick model and the Kaiser–Guttman criterion (Frontier 1976; Jackson 1993) to determine the number of components to retain for PCA. Where necessary, we subjected the principal components from the PCA of the centroid sizes to varimax rotation to achieve simple structure, but we did not shear the components because sheared components of genetic and environmental correlation matrices have no straightforward interpretation. Plots of the principal components identified an additional outlier, which was removed to result in 1,870 observations for genetic analysis.

We checked for outliers, collinearity, and departures from normality in two ways. First, we examined the distributions of centroid sizes with frequency histograms and probability plots of observed against expected values; although some traits were slightly right-skewed and leptokurtic, we detected no appreciable departures from normality. Second, for each centroid size, we examined bivariate scatterplots of the residuals from the principal components; with the exception of the residuals for the two posterior-most lateral centroid sizes (csl5 and csl6; Figure 1), which were positively correlated ($r^2 = 0.78$), these plots revealed no clear patterns, groupings, or outliers. We concluded that the distributions of these data do not seriously violate the assumptions of the PCA, which should be robust to modest violations of this sort (Rencher 1995).

We estimated the genetic components of covariance and their derivatives (h^2 and r_A) for each of the 12 centroid sizes and log body weight with restricted maximum likelihood (REML) (Harville 1977; Patterson and Thompson 1971; Shaw 1987). We used REML because variance component estimates derived by least squares (e.g., MANOVA) can be biased when family structure is unbalanced and the resulting trait covariance matrices are sparse, as in our case (Henderson 1953; Searle 1971). REML produces unbiased parameter esti-

mates; in balanced designs, estimates from REML and least squares are equivalent (Searle 1971). Clark (1990) showed empirically that estimates computed by the two methods can correspond well under a fairly typical experimental design.

The genetic model for variance component estimation was a mixed model of the form $\mathbf{y = Xb + Zs + Wd + Ie}$, where **y** is the vector of trait values, **X** is the incidence (design) matrix for the fixed effect of the grand mean, **Z** is the incidence matrix for the random effect of sires, **W** is the incidence matrix for the random effect of dams, **I** is the error matrix, and **b, s, d,** and **e** are vectors containing the effects of these fixed and random factors on the individual phenotype. The elements of the incidence matrices are 1 if individual *i* is progeny of sire or dam *j* and 0 otherwise. This model is easily reformulated in terms of the causal genetic and environmental components of variance and covariance that incorporate the vectors of breeding values (additive genetic effects), dominance deviations, and environmental deviations (Shaw 1987).

We used the derivative-free algorithm in the program DFREML (Meyer 1997) to estimate the genetic parameters for each pair of traits because it can use the average information matrix to derive these estimates and their approximate sampling errors for sparse covariance matrices (Johnson and Thompson 1995). Estimating all the variance and covariance components with DFREML required approximately 20 h of CPU time on a 400-MHz Pentium II PC with 128 Mb RAM to compute and invert the covariance matrices for each pair of traits (78 pairs total) and iterate the solution to achieve convergence. We estimated the environmental correlation between each trait pair, $r_{E(ij)}$, from the relationship between their phenotypic $r_{P(ij)}$ and genetic correlation $r_{A(ij)}$, according to the equation $r_{E(ij)} = [r_{P(ij)} - r_{G(ij)}(h_i h_j)]/\sqrt{[(1 - h_i^2)(1 - h_j^2)]}$ (Falconer 1989).

Results

Phenotypic Analyses

In the PCA of centroid sizes of the six lateral, two dorsal, and four ventral truss cells (Figure 1), the loadings on three components identified in the analysis (Table 1) suggested the following interpretation of the axes: PC1 is an allometry vector describing the effects of body size on shape; PC2 is a contrast between the sizes of the hindbody and the tail; and PC3 is a contrast between body depth and width. Loadings on PC1 were all large and positive, and this component accounted for most (65.4%; eigenvalue, $\lambda = 7.847$) of the variance in centroid sizes. PC2 was highly positively correlated with the centroid size of lateral cell 5 (csl5) and negatively with the centroid size of lateral cell 6 (csl6), explaining 9.6% ($\lambda = 1.195$) of the variance. PC3 was weakly correlated positively with the centroid sizes of the lateral cells (primarily csl4 and csl5) and negatively with the centroid sizes of dorsal and ventral cells (primarily the most anterior dorsal cell, csd1, and the most anterior ventral cell, csv1); PC3 explained an additional 8.1% ($\lambda = 0.971$) of the variance (Table 1). Inclusion of log weight into the PCA did not appreciably alter these components. Varimax rotation of the components essentially shifted PC2 to become the fourth largest component ($\lambda = 1.235$), identifying a new second component ($\lambda = 3.083$) that was evidently another allometry vector orthogonal to PC1 ($\lambda = 4.326$).

Table 1. Correlations of truss cell (see Figure 1) centroid sizes with principal components of their phenotypic correlation matrix (P) and their corresponding genetic (G) and environmental (E) correlation matrices

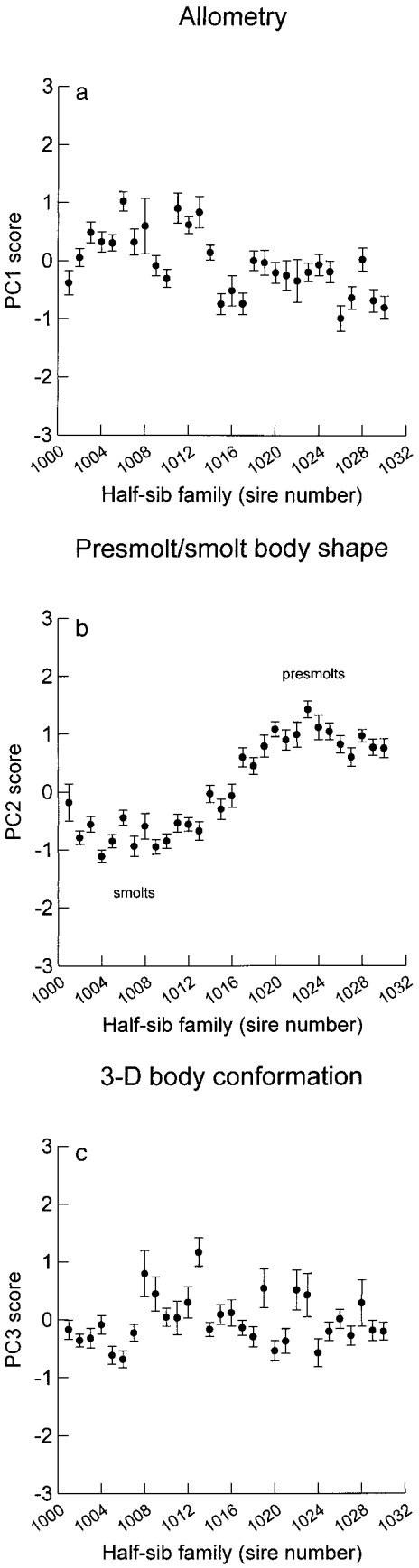
	P			G			E		
Variable	PC1	PC2	PC3	PC1	PC2	PC3	PC1	PC2	PC3
csl1	0.775	−0.007	0.282	0.832	0.208	0.030	0.777	0.224	−0.105
csl2	0.890	−0.003	0.280	0.927	0.194	−0.027	0.914	0.106	0.059
csl3	0.887	−0.008	0.286	0.907	0.304	0.001	0.880	0.240	−0.058
csl4	0.827	−0.001	0.377	0.889	0.320	0.054	0.837	0.262	−0.060
csl5	0.237	0.885	0.316	0.626	0.088	0.729	0.632	0.105	−0.665
csl6	0.670	−0.599	0.204	0.636	0.570	−0.375	0.601	0.528	0.483
csv1	0.793	0.001	−0.400	0.833	−0.430	−0.190	0.740	−0.405	0.175
csv2	0.861	0.130	−0.224	0.927	−0.253	0.000	0.836	−0.228	0.005
csv3	0.934	0.004	−0.118	0.905	−0.120	0.006	0.900	−0.086	−0.006
csv4	0.865	0.006	−0.118	0.922	−0.105	0.028	0.840	−0.064	−0.025
csd1	0.781	0.111	−0.400	0.846	−0.367	−0.120	0.748	−0.400	0.154
csd2	0.936	0.007	−0.223	0.942	−0.237	−0.039	0.931	−0.177	0.038
Eigenvalue	7.847	1.195	0.971	8.785	1.074	0.729	7.863	0.896	0.753
Total variance (%)	65.393	9.957	8.094	73.209	8.946	6.078	65.526	7.467	6.278

For each analysis, the loadings of each variable on the first three unrotated components (1–3) are shown. Centroid sizes of lateral truss cells, csl1–csl6; centroid sizes of ventral truss cells, csv1–csv4; and centroid sizes of dorsal truss cells, csd1 and csd2.

The distribution of mean principal component scores by sire for the first three phenotypic components is depicted in Figure 2. Significant among-dam, within-sire variation was detected for each of these components. In general, progeny of fish spawned after October 10 (i.e., sire > 1015) had relatively small PC1 and large PC2 scores, identifying smaller fish with relatively small tails (i.e., fish not yet completely smolted; Figure 2A,B). PC2 appears to be associated with smoltification, and members of families that had not yet completed the morphological transition during smoltification (progeny of sires 1018–1030 in Figure 2A) would have undoubtedly manifested smoltlike phenotypes within a few weeks of our sample (Winans and Nishioka 1987). Nevertheless, substantial among-sire variation in PC2 was evident in families that had already smolted (progeny of sires 1001–1013) and in those that had not yet initiated morphological smoltification (sires 1018–1030), indicating substantial additive genetic variation for csl5 and csl6.

Mean progeny PC3 values (Figure 2C) also differed among sires and identified a distinct group of fish that were progeny of 9 of the 30 sires and, within this sire group, were progeny of only 10 of 31 dams (Figure 3A). All but two individual progeny in these 10 full-sib families showed large positive scores for PC3 that differed distinctly from those of most progeny in the experiment (Figure 3B), a pattern that indicated two distinct phenotypes (cylindrical body-wide head versus laterally compressed body-narrow head) differentiated by common environmental or cytoplasmic factors, or by a genetically based maternal effect. PC3 scores declined similarly in the two groups with increasing scores along PC1, indicating that the two PC3 variants are maintained over the range of allometric variants observed during the 3 weeks of sampling (Figure 3B).

The 10 dams producing these progeny did not differ significantly from the other dams considered in Figure 3 with regard to spawning date ($F_{1,92} = 0.002$, $P > .95$), body length ($F_{1,92} = 0.351$, $P > .50$), body weight ($F_{1,92} = 2.357$, $P > .10$), or fecundity ($F_{1,92} = 1.084$, $P > .25$), and their progeny did not differ significantly in rearing density ($F_{1,92} = 0.246$, $P > .50$). Moreover, the distinctive pattern of variation in PC3



showed no systematic variation with sire or rearing-tank location. The mean egg weight of dams whose progeny had large positive PC3 scores was 10% greater than that of dams whose progeny had small PC3 scores (0.329 ± 0.013 g versus 0.299 ± 0.004 g; $F_{1,92} = 4.858$, $P = .030$), but a multivariate analysis of group differences in egg weight, fecundity, body length and weight, rearing density, and spawning date indicated that these two groups of females were similar in these respects (Wilks' $\Lambda = 0.892$, $P = .117$). It is nevertheless possible that common environmental influences on PC3 may have occurred through egg provisioning or other nonadditive factors, but do not appear to have included appreciable tank or culture effects. We interpret the decline in PC3 with increasing scores along the allometry vector PC1 (Figure 3B) to reflect primarily a shift in juvenile chinook salmon from a more cylindrical body shape in the presmolt phase to the acquisition of a more laterally compressed shape through a deeper lateral profile in the smolt and postsmolt phases.

Full-sib family size (or rearing density) had substantial effects on components of allometry but not the other shape vectors. The standardized regression of PC1 on full-sib family size was strongly negative ($b = -0.902$; $F_{1,1868} = 451.963$, $P < .001$). Full-sib family size showed no significant relationship with PC2 ($F_{1,1868} = 0.038$, $P > .75$). Although the relationships between family size and PC3 were significant ($P < .001$) in both of the progeny groups differentiated by PC3, these relationships were weak (low PC3: $F_{1,1675} = 77.919$, $P < .001$, $r^2 = 0.044$; high PC3: $F_{1,191} = 24.615$, $P < .001$, $r^2 = 0.110$).

Genetic Analyses

The REML analysis of the variance components for body weight and the 12 centroid sizes indicated that all were highly heritable (Table 2). The heritabilities of csl5 and csl6 were near the center of the range of values observed, being exceeded by those for five other traits. A PCA of the genetic correlation matrix of the 12 centroid sizes yielded two components based on the broken-stick model. The first component appeared to be a vector reflecting the magnitude of pleiotropy (i.e., a measure of the size of the genetic effect across loci), as all genetic variables showed large positive loadings on this component. This vector accounted for 73.2% ($\lambda = 8.785$) of the total variance in the genetic covariation among the centroid sizes. The second component was positively correlated with csl6 (and, to a lesser extent, with csl3 and csl4) and negatively correlated primarily with both csd1 and csv1, suggesting that this component reflects pleiotropic effects of genes affecting tail size and head width. This component explained 8.9% ($\lambda = 1.074$) of the variance. A third component was positively correlated with csl5 and negatively correlated with csl6, implying pleiotropy of genes affecting caudal peduncle size and tail size; this component accounted for 6.1% ($\lambda = 0.729$) of the variance.

A PCA of the corresponding environmental correlation matrix showed a remarkably similar pattern to the genetic analysis. We therefore interpreted the principal components of this matrix to represent combinations of nonadditive genetic and environmental factors affecting the centroid sizes in a manner similar to the genetic factors. Collectively the phenotypic, genetic, and environmental principal components provided evidence for common genetic control of several aspects of body size and shape, and phenotypic patterns were largely reflected by underlying genetic patterns. A plot of loadings of the centroid sizes on the first principal component from analysis of the genetic correlation matrix against loadings of these traits on the first principal component from analysis of the environmental correlation matrix (Figure 4A) indicates that each of the traits was correlated similarly with the "additive genetic" (pleiotropy) and "nonadditive genetic/environmental" vectors, suggesting that the traits contributed similar amounts of genetic and environmental influence on phenotypic variation. A corresponding plot for all 13 variables (Figure 4B) shows that most of these contributed more to the pleiotropy vector, with the notable exception of log weight and csl5, which contributed more to the nonadditive genetic/environmental vector.

The patterns of phenotypic and genetic correlations among the morphometric traits were similar. All traits were positively correlated both phenotypically and ge-

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Figure 2. Distribution of scores among 30 half-sib families of juvenile chinook salmon for the first three components from a PCA of truss cell (see Figure 1) centroid sizes. **(A)** PC1, interpreted as allometric variation in body shape. **(B)** PC2, interpreted as a presmolt/smolt body shape contrast. **(C)** PC3, interpreted as three-dimensional body conformation. Error bars in each panel denote two SE of the mean. The sires were spawned in numerical order on nine dates between October 3 and 21, with an average of 3.3 dams mated per sire.

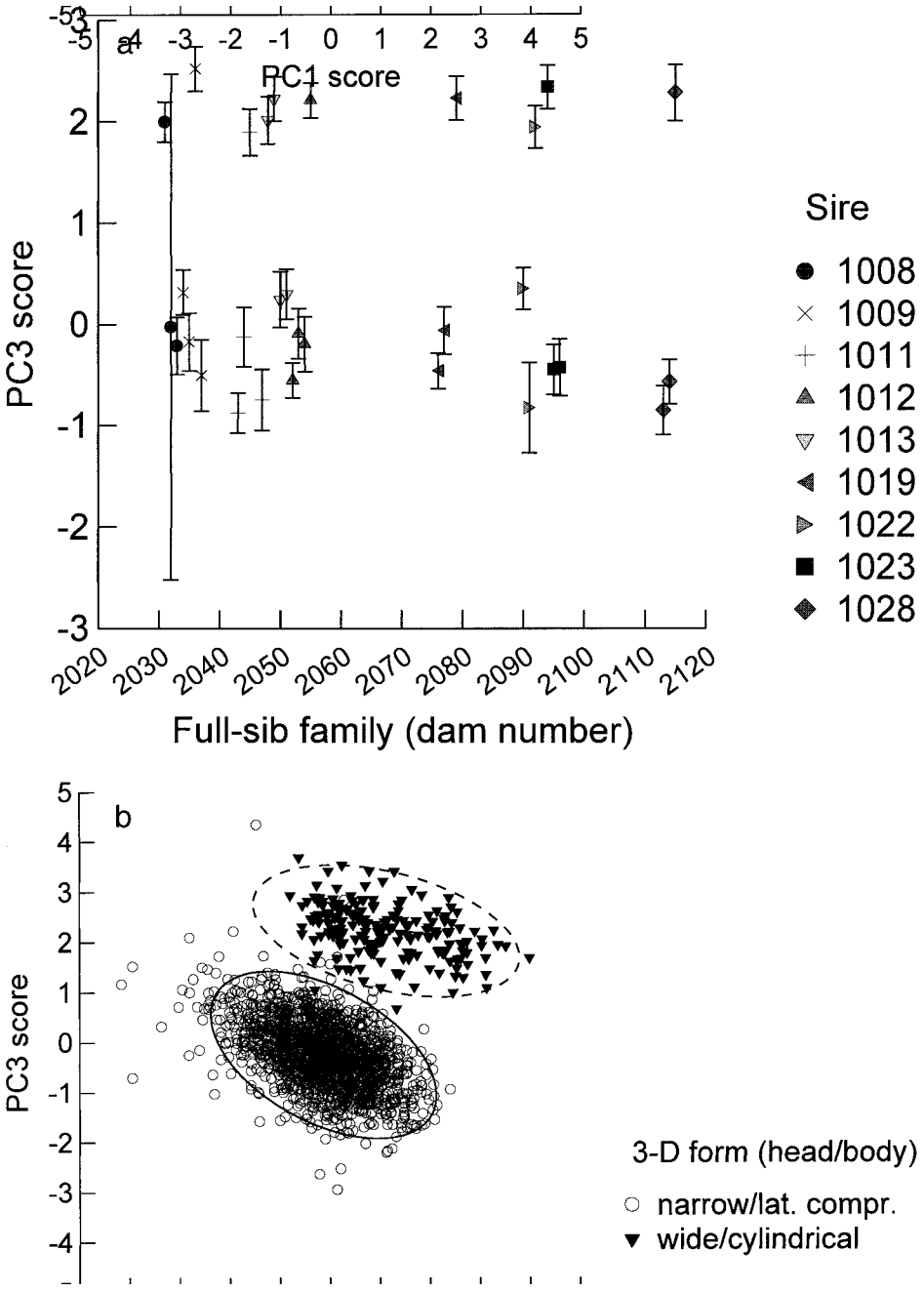

Figure 3. **(A)** Distribution of PC3 (three-dimensional body conformation) scores for the offspring of 31 dams mated to 9 sires, indicating high PC3 scores for progeny of 10 of these dams. Error bars denote two SE of the mean. **(B)** Scatterplot of PC3 scores on PC1 (allometry) scores from a PCA of truss cell (see Figure 1) centroid sizes, indicating the two distinct juvenile body shapes. The upper cloud of points corresponds to the high PC3 scores of progeny of the 10 dams identified in **(A).** Ninety-five percent confidence ellipses are shown for a laterally compressed form with narrow head and a cylindrical form with wide head.

netically, and all genetic correlations differed significantly from zero (Table 3). The smallest genetic correlation (0.265) was between csl5 and csl6, which also had the smallest phenotypic correlation (0.244). Most genetic correlations were larger than +0.6 and tended to be larger than their corresponding phenotypic correlations, implying that pleiotropy tended to affect centroid sizes in similar directions along the entire body form and environmental covariance between traits generally acted to erode genetically based trait similarities.

Figure 5 depicts patterns corresponding to the 10 largest and 10 smallest phenotypic and genetic correlations between traits. These patterns show that the lowest phenotypic and genetic correlations occurred between the depth of the hindbody and body width; the highest pheno-

Table 2. Heritabilities (h^2) and standard errors (SE) estimated by REML for log weight (logwt) and the 12 centroid sizes (csl1–csd2)

Trait	h^2	SE
logwt	0.995	0.058
csl1	0.834	0.023
csl2	0.913	0.017
csl3	0.922	0.019
csl4	0.834	0.024
csl5	0.953	0.037
csl6	0.882	0.048
csv1	0.760	0.025
csv2	0.788	0.016
csv3	0.997	0.016
csv4	0.797	0.018
csd1	0.752	0.023
csd2	0.984	0.008

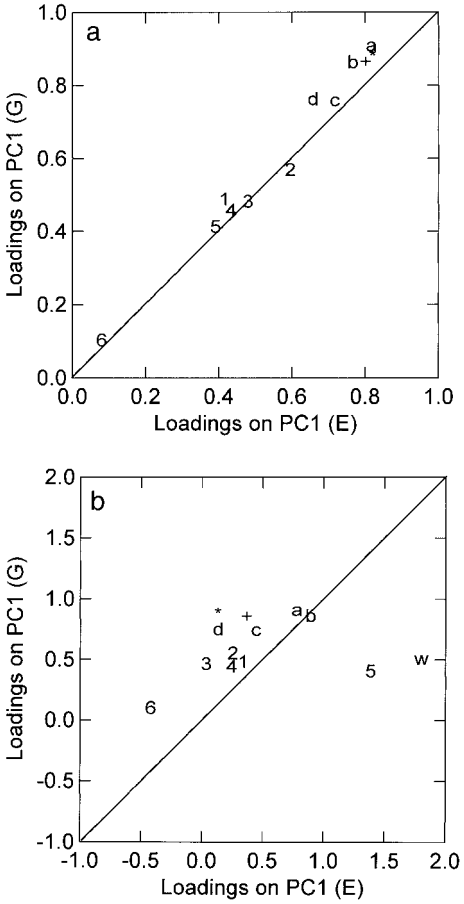

Figure 4. **(A)** Relationship of loadings of truss cell (see Figure 1) centroid sizes on the first eigenvector (from a PCA) of their genetic covariance matrix ($PC1_G$) to the corresponding loadings on the first eigenvector of their environmental covariance matrix ($PC1_E$). **(B)** Relationship of loadings of all morphometric variables on their $PC1_G$ to the corresponding loadings on their $PC1_E$. The diagonal line represents equal contribution of variables to the two eigenvectors. Variables: 1–6, csl1–csl6, respectively; a–d, csv1–csv4, respectively; *, csd1; +, csd2; w, log weight.

Table 3. Phenotypic (on and above diagonal) and genetic (below diagonal) correlation matrices for log weight and the 12 centroid sizes

Trait	logwt	csl1	csl2	csl3	csl4	csl5	csl6	csv1	csv2	csv3	csv4	csd1	csd2
logwt	1.000	0.751	0.923	0.909	0.844	0.580	0.590	0.612	0.727	0.847	0.748	0.623	0.822
csl1	0.791 (0.035)	1.000	0.621	0.720	0.694	0.487	0.474	0.513	0.597	0.637	0.600	0.488	0.651
csl2	0.943 (0.033)	0.688 (0.022)	1.000	0.857	0.811	0.555	0.584	0.578	0.702	0.819	0.706	0.603	0.800
csl3	0.963 (0.032)	0.810 (0.025)	0.928 (0.019)	1.000	0.766	0.539	0.583	0.543	0.661	0.783	0.704	0.562	0.764
csl4	0.915 (0.035)	0.818 (0.015)	0.906 (0.023)	0.866 (0.024)	1.000	0.523	0.531	0.498	0.623	0.710	0.686	0.517	0.725
csl5	0.580 (0.034)	0.529 (0.026)	0.572 (0.022)	0.560 (0.028)	0.577 (0.031)	1.000	0.244	0.364	0.497	0.521	0.493	0.383	0.536
csl6	0.672 (0.038)	0.587 (0.029)	0.686 (0.021)	0.697 (0.033)	0.664 (0.035)	0.265 (0.029)	1.000	0.348	0.415	0.462	0.436	0.349	0.484
csv1	0.612 (0.032)	0.647 (0.021)	0.693 (0.016)	0.574 (0.025)	0.565 (0.028)	0.387 (0.031)	0.401 (0.034)	1.000	0.655	0.653	0.601	0.632	0.709
csv2	0.727 (0.022)	0.716 (0.015)	0.800 (0.011)	0.749 (0.017)	0.720 (0.021)	0.571 (0.030)	0.477 (0.032)	0.876 (0.023)	1.000	0.760	0.675	0.647	0.807
csv3	0.847 (0.015)	0.684 (0.014)	0.835 (0.009)	0.824 (0.016)	0.761 (0.020)	0.521 (0.027)	0.462 (0.031)	0.770 (0.026)	0.853 (0.019)	1.000	0.746	0.649	0.861
csv4	0.845 (0.018)	0.729 (0.016)	0.813 (0.011)	0.811 (0.022)	0.814 (0.014)	0.551 (0.023)	0.485 (0.031)	0.792 (0.015)	0.863 (0.021)	0.834 (0.020)	1.000	0.578	0.803
csd1	0.724 (0.026)	0.610 (0.021)	0.716 (0.015)	0.668 (0.027)	0.634 (0.029)	0.445 (0.029)	0.401 (0.032)	0.871 (0.015)	0.849 (0.024)	0.747 (0.024)	0.751 (0.028)	1.000	0.761
csd2	0.824 (0.011)	0.702 (0.010)	0.818 (0.006)	0.782 (0.020)	0.764 (0.014)	0.536 (0.038)	0.486 (0.028)	0.854 (0.022)	0.938 (0.012)	0.879 (0.016)	0.908 (0.015)	0.883 (0.018)	1.000

Approximate standard errors (SE) for the genetic correlations are in parentheses.

typic correlations between traits. These patterns show that the lowest phenotypic and genetic correlations occurred between the depth of the hindbody and body width; the highest phenotypic correlations occurred between log weight and the depth and width of the midbody, but the largest genetic correlations tended to occur either between log weight and the depth of the midbody or between traits contributing to the width of the midbody. These results suggest that although genetic control of body dimension may have been coordinated (as reflected by large genetic correlations of the same sign), pleiotropic effects may also have constrained a change in body width and depth, particularly for the hindbody and tail relative to overall midbody dimension.

Discussion

Our analysis of morphometry in this population of juvenile chinook salmon indicates substantial genetic influence over body size and components of body shape. The phenotypic patterns observed in this population of juvenile chinook salmon reflect variation primarily along three axes: allometry ($\lambda = 7.847$), tail shape associated with smoltification ($\lambda = 1.195$), and a contrast between head width and body depth ($\lambda = 0.971$). Together these components account for more than 80% of observed variation in morphometry, and estimates of the variance components for the constituent traits indicate that substantial additive genetic variation exists for all major components of the truss (Table 2). These results correspond well to a sheared PCA of the 55 interlandmark distances composing the truss. An allometry vector explained 47.7% of the total variance, with scores along this axis varying positively with body weight ($r = 0.920$, $P < .0001$) and negatively (when summed over full-sib families) with tank rearing density ($r = -0.450$, $P = .033$). The second vector accounted for an additional 6.9% of the variance and indicated a shape contrast between the caudal peduncle (negative loadings for L10–11, L5–6, L6–11, and L5–10 in Figure 1) and the tail (positive loadings for L6–8 and L8–10). This sheared component closely resembles a character described by Winans and Nishioka (1987) for coho salmon (*O. kisutch*) that changes in association with smoltification. The third vector explained 5.1% of the variance and contrasted primarily lateral (negative loadings for nearly all distances) with dorsal and ventral measurements (positive loadings for most distances, especially D2–5 and V3–8). Therefore this component appears to represent variation in body shape ranging from a form with a large, wide head and less body depth (positive loadings) to a type with a relatively small, narrow head and deep body (negative loadings).

The corresponding axes of variation for the genetic correlation matrix among these traits show a close correspondence to those for the phenotypic correlation matrix, suggesting that the primary opportunities for short-term evolution of salmon morphometry are generally well reflected by observed variation in allometry, morphological aspects of smoltification, and overall body conformation. For all the traits, phenotypic and genetic correlations have the same sign and are similar in magnitude, with the genetic correlations tending to be somewhat larger. Thus in this case phenotypic correlations seem to be reasonable proxies for the underlying genetic correlations (Cheverud 1988; Diniz-Filho and Pignata 1994). Indeed, the correspondence of the phenotypic and genetic correlation structure lends strong

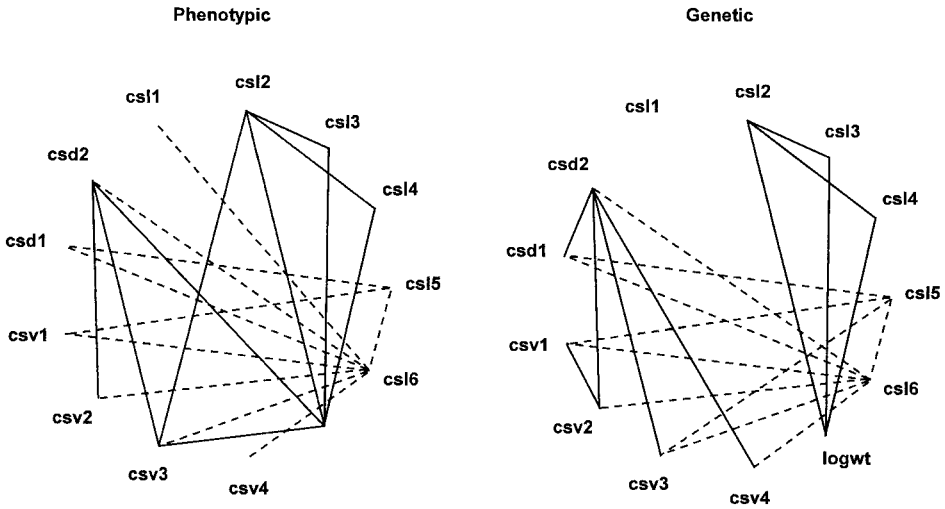

Figure 5. Representation of the 10 largest (solid lines) and 10 smallest (dashed lines) phenotypic (left) and genetic (right) correlations among all 13 morphometric variables (see Table 3). All correlations are significantly larger than zero. Variable names as in Table 1.

support to a hypothesis that body size and shape represent a genetically coordinated trait "syndrome," or what Leamy (1977) described as "morphological integration."

Nevertheless, these characters are not identical. The genetic analysis provides strong evidence for pleiotropy, but the high levels of genetic variation maintained in this set of characters and their nonuniform genetic correlation structure suggest the possibility that juvenile body conformation is under some form of selection to an optimal form. This selection would likely be mediated through the abilities to hold position in flowing water, defend territory, forage, and avoid predators. Selection after outmigration to seawater must also occur, and some of this selection may affect body conformation. As these fish mature and return to freshwater to breed, we shall have an opportunity to examine to what extent morphometric changes occur in these fish over their lifetimes and determine how the genetic and phenotypic relationships among these characters change during development. For example, does the genetic architecture of juvenile morphometry differ fundamentally from that of adults?

Previous morphometric studies of juvenile anadromous salmonids have found that the phenotypic expression of morphometry is genetically influenced (Beacham 1990; Fleming et al. 1994; Swain et al. 1991), but in most cases this conclusion must be tempered by the fact that environmental influences such as residual field effects or maternal/cytoplasmic effects on observed variation could not be ruled out. Of these studies, only that of Beacham (1990) estimated the genetic components of phenotypic variation in morphometry with quantitative-genetic methods. However, his analysis relied on univariate morphometric analyses.

Several years ago, Leamy and Atchley (1984) cautioned, in response to an assumption implicit in several earlier morphometric studies of different taxa, that shape is not necessarily less environmentally sensitive than size. Indeed, researchers working recently on salmonids (Currens et al. 1989; Fleming et al. 1994; Swain et al. 1991) concluded that aspects of body shape can be highly sensitive to environmental variation. In our study we determined that elements of multivariate shape can have an appreciable genetic influence in chinook salmon. However, determining the genetic basis of shape is not straightforward, in part because of the difficulty in defining this trait. Following the suggestion of Rohlf and Bookstein (1987) that multivariate shape be defined from a PCA of morphometric variables as its second and subsequent eigenvectors, we found that vectors describing both smolt morphology (PC2) and a three-dimensional contrast of head and body shape (PC3) show appreciable variation among half-sib families. Variation among half-sib families in PC2 scores was responsible for about 13% of the total variation (sires 1001–1013: $F_{11,32} = 16.384$, $P < .0010$; sires 1018–1030, excluding 1024: $F_{11,23} = 12.307$, $P < .0010$). Variation among half-sib families in PC3 scores was responsible for about 19% of the total variation ($F_{28,66} = 39.231$, $P < .0010$). We did not estimate heritabilities for PC2 or PC3 because of evidence of bimodality in both components.

The nearly bimodal variation in three-dimensional body form, as measured by PC3, appeared to result from maternal or closely related environmental variation; if the result of a maternal effect, we were not able to determine whether it was environmentally or genetically based. One possible source of this bimodality is variation among parental females in egg characteristics. We detected a mean difference of about 10% in egg weight between females whose progeny had low versus high PC3 scores. However, this difference represented less than one phenotypic standard deviation (5% of the total variation in egg weight was explained by group differences), and we believe it unlikely that egg size alone can account for the variation among groups in PC3 scores. Egg size may be correlated with an unknown nonadditive genetic or environmental trait that explains most of this variation.

Therefore our study indicates that morphometric variation in anadromous salmonids can be strongly influenced by environmental—or genetically based maternal—factors (Reznick 1981) as well as genetic factors. The relationship between the genetic and phenotypic correlation structure of morphometry indicates that environmental variation tended to reduce resemblance among relatives. However, one prominent aspect of environmental variation was only weakly correlated with body shape. Rearing density appears to have had little effect on multivariate shape relative to its effect on size and growth, at least at this life-history stage.

The elements of the genetic covariance matrix (Table 3) reveal the most prominent heritable constraints on as well as opportunities for morphometric change in this population, thereby revealing their likely pathways of short-term evolution (Lande 1979; Shaw et al. 1995). The phenotypic patterns of morphometric variation in this population identify opportunities for selection and show some similarities to morphological differences that have been observed between juvenile wild and hatchery salmonids (such as smaller heads and fins, less body depth, and smaller caudal peduncles in hatchery fish). This variation may represent evolutionary opportunities for environmental pressures to act to differentiate the two groups morphologically (Swain et al. 1991). For example, in the sheared PCA we estimated large, positive genetic correlations between both allometry and body weight and components highly correlated with anal fin base size, trunk width, and pectoral fin position. These correlations differed substantially from the small phenotypic correlations observed between the traits, and appear to represent heritable allometric changes in the sizes and position of fins. Selection on juvenile body size could indirectly alter fin size and position as well as other aspects of body shape, for which differences are apparent within (Bailey and Irvine 1991; Swain and Holtby 1989; Swain et al. 1991; Taylor and McPhail 1985) as well as among (Bisson et al. 1988) species. The correlated responses could affect performance in different habitats and, consequently, patterns of habitat use in hatchery-reared juveniles released to the wild. In evaluating shape differences on smaller morphometric scales, limitations of the truss may be apparent. Analyses of variation in fin position and size among hatchery and wild fish in nature involving the application of geometric techniques such as relative warp analysis may be more powerful (Bookstein 1991).

A multivariate analysis of morphometric characters in a quantitative genetic framework can permit a glimpse of the evolutionary constraints on and opportunities for morphometric variation in salmonids. Inclusion of two additional truss-network profiles (ventral and dorsal) in our analyses allowed us to detect, for the first time, a true three-dimensional shape variant (PC3) that discriminated fish of two distinct groups. This variant showed appreciable variation among families, and was not detectable in separate analyses of the three two-dimensional profiles.

The ecological and evolutionary consequences of this variant and its persistence during development in these fish are not

clear. Although the character varied with size in both groups, these groups remained distinct across the range of observed size. Because we could not attribute these differences to tank or culture effects, we conclude that maternal, cytoplasmic, or common environmental factors may have acted to retard morphological change during smoltification in some families of fish. Whether these factors are appreciable or diminish during development will require examination of siblings from these families at later stages of development.

At least three caveats associated with this study should be recognized. First, as with most quantitative genetic studies, we assumed in our analyses that 1) loci affecting morphometry are unlinked, 2) bias in our variance component estimates from nonadditive sources of genetic variation or variation due to common environment not accounted for in the model was negligible, 3) genotype × environment interaction and genotype-environment correlation could be ignored, and 4) the hatchery population had not experienced appreciable inbreeding. With the possible exception of tank effect or genotype-environment correlation associated with tank assignment, we have no reason to believe that these assumptions have been violated. Since 1982, the hatchery broodstock has been maintained at more than 1,000 adults, mated so that each female is mated simultaneously with two males (with the second male serving as a safeguard against male infertility), each year except for 1991 and 1992, when the number of adults dropped to as low as 200.

Second, the fish evaluated in this study were from a single hatchery population, and inferences about other—particularly natural—populations must be tempered by this knowledge. Although Pacific salmon reared in hatcheries typically spend much of their life cycle in the natural environment, the effects on morphometry of multiple generations of rearing during early life history in a captive environment have not been carefully documented (Fleming et al. 1994). For example, variation in skeletal morphometry has been shown to respond to inbreeding and/or domestication in other vertebrates (Lacy and Horner 1996), and inbreeding or domestication in hatchery fish (Campton 1995; Gall 1987) could act to differentiate hatchery from wild fish, with unpredictable results. Unfortunately the extent to which this might occur in most salmon hatcheries is not known.

Finally, the principal components of morphometric variation described here were captured from fish during smoltification, the pivotal life-cycle transition common to all anadromous salmonids. The patterns of morphometric variation observed here may change dramatically when hatchery fish experience natural selection in the wild later in life (Waples 1991). Whether the form of the allometric relationship or the two distinct morphological types observed in this study will persist across the major metamorphic transition of smoltification and through the life cycle is an open question. Quantitative genetic studies that examine patterns of genetic and phenotypic variation across this transition are needed to shed further light on the ontogenic profile of morphometry and its consequences for fitness of both hatchery and naturally reproducing populations.

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