

# Genetic Basis of Variation in Morphological and Life-History Traits of a Wild Population of Pink Salmon

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## Abstract

Understanding the genetic basis of phenotypic variation is essential for predicting the direction and rate of phenotypic evolution. We estimated heritabilities and genetic correlations of morphological (fork length, pectoral and pelvic fin ray counts, and gill arch raker counts) and life-history (egg number and individual egg weight) traits of pink salmon (*Oncorhynchus gorbuscha*) from Likes Creek, Alaska, in order to characterize the genetic basis of phenotypic variation in this species. Families were created from wild-caught adults, raised to the fry stage in the lab, released into the wild, and caught as returning adults and assigned to families using microsatellite loci and a growth hormone locus. Morphological traits were all moderately to highly heritable, but egg number and egg weight were not heritable, suggesting that past selection has eliminated additive genetic variation in egg number and egg weight or that there is high environmental variance in these traits. Genetic correlations were similar for nonadjacent morphological traits and adjacent traits. Genetic correlations predicted phenotypic correlations fairly accurately, but some pairs of traits with low genetic correlations had high phenotypic correlations, and vice versa, emphasizing the need to use caution when using phenotypic correlations as indices of genetic correlations. This is one of only a handful of studies to estimate heritabilities and genetic correlations for a wild population.

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The genetic architecture of phenotypic variation can be described by two primary parameters: heritability and genetic correlations. Heritability in the narrow sense ( $b^2$ ) is a dimensionless index of heritable variation, termed the additive genetic variance ( $V_A$ ). Heritability is defined as the proportion of within-population phenotypic variance ( $V_P$ ) that is additive genetic (i.e.,  $b^2 = V_A/V_P$ ;  $0 \leq b^2 \leq 1$ ). The importance of  $b^2$  for predicting a phenotypic response to selection can be seen with the breeder's equation  $R = b^2S$ , where  $S$  is the selection differential (the difference in the mean of a trait before and after selection within a generation) and  $R$  is the response to selection (the difference in the mean trait value between generations) (Roff 1997). Therefore the response to selection is a linear function of  $b^2$ . Moreover, because selection reduces  $V_A$ , the magnitude of  $b^2$  reflects the extent to which a trait has been under net directional or stabilizing selection in the past (Endler 2000).

Genetic correlations between pairs of traits also have important effects on the direction and rate of phenotypic evolution. The genetic correlation ( $\rho_A$ ) between two traits

describes the proportion of the phenotypic correlation between the traits that is caused by genetic variation that affects both traits simultaneously. Genetic correlations between characters can arise by two mechanisms, pleiotropy or gametic phase disequilibrium (Lynch and Walsh 1998). Pleiotropy occurs when a single gene affects multiple traits due to complex biochemical and developmental pathways (Wright 1968). Gametic phase disequilibrium is the tendency of genes affecting different traits to be positively or negatively associated in the same individuals. Both pleiotropy and gametic phase disequilibrium can cause positive or negative genetic correlations. Genetic correlations can constrain or enhance phenotypic evolution, depending on whether the correlations are positive or negative and on whether selection acts in the same or opposite directions with respect to fitness on the traits in question. For example, a positive genetic correlation will speed the rate of evolution of two traits if selection acts on both traits in the same direction with respect to fitness, but will slow the rate of change if selection acts in opposite directions.

Despite the importance of understanding the genetic basis of phenotypic variation in evolutionary biology, many basic questions about heritabilities and genetic correlations remain due to a paucity of estimates of these parameters for natural populations (for examples of studies that have estimated these parameters for natural populations, see Boag and Grant 1978; Conner and Via 1993; Gustafsson 1986; Kruuk et al. 2000; Young et al. 1994). One important question is whether certain categories of traits, including morphological, life-history, behavioral, and physiological traits, have higher heritabilities than others. Generalizations about the heritabilities of different categories of traits will improve our understanding of the potential for evolution of different traits as well as provide insight into the relative intensity of past selection. Several studies have shown that heritability estimates tend to be highest for morphological traits, lowest for life-history traits, and intermediate for behavioral and physiological traits (Gustafsson 1986; Kruuk et al. 2000; Mousseau and Roff 1987). This result has been interpreted as support for Fisher's fundamental theorem of natural selection, which predicts that additive genetic variance for traits with strong effects on fitness, such as life-history traits, will approach zero at equilibrium (Fisher 1930).

A second important question is whether adjacent morphological traits have higher positive genetic correlations than nonadjacent traits. A positive relationship between the physical proximity of morphological traits and genetic correlations might be expected if adjacent traits are encoded or regulated by the same genes. Alternatively, more similar types of morphological traits that are nonadjacent, for example, structures such as limbs, may have higher genetic correlations than adjacent traits. Selection experiments on *Drosophila* wings demonstrate that adjacent morphological traits can evolve independently, suggesting that genetic correlations among adjacent structures are not always high enough to constrain independent evolution (Weber 1992).

A final question is whether genetic correlations accurately predict phenotypic correlations. In general, genetic correlations should predict phenotypic correlations fairly accurately since genetic correlations, along with environmental correlations, determine phenotypic correlations. However, the degree to which genetic correlations predict phenotypic correlations will depend on how often environmental correlations oppose genetic correlations. If, for example, environmental variation tends to cause negative correlations between pairs of traits that have positive genetic correlations, then genetic correlations will do a poor job of predicting phenotypic correlations. The relationship between genetic correlations and phenotypic correlations has important practical implications. Because genetic correlations are difficult to estimate, it would be convenient to use phenotypic correlations as indices of genetic correlations. If genetic correlations accurately predict phenotypic correlations, and vice versa, using phenotypic correlations as indices of genetic correlations would be justified. Reviews of the literature suggest that phenotypic and genetic correlations tend to have the same sign and magnitude (Cheverud 1988, 1995; Roff 1995, 1996).

We addressed the above three questions for a natural population of pink salmon (*Oncorhynchus gorbuscha*) from Likes Creek, Alaska, by estimating heritabilities and genetic correlations for morphological and life-history traits. Pink salmon have a strict, two-year life cycle in which reproductive adults return to their natal streams 2 years after they begin development. We created families from wild-caught adults, raised fish to the fry stage in the lab, released fry into the wild, and caught returning adults and assigned them to families using microsatellite loci and one growth hormone locus. Heritabilities and genetic correlations were then estimated using parent-offspring regressions and covariances, respectively. The morphological traits examined were fork length, the number of pectoral and pelvic fin rays, and the number of upper and lower gill arch rakers. The life-history traits were egg number and individual egg weight. Our specific objectives were to test (1) whether heritabilities were higher for morphological traits than life-history traits; (2) whether genetic correlations of morphological traits depend on the physical proximity of the traits; and (3) whether genetic correlations accurately predict phenotypic correlations.

## Materials and Methods

### Production of Families and Recapture of Returning Progeny

We collected gametes from 34 female and 34 male pink salmon from the mouth of Likes Creek, Resurrection Bay, Alaska, in August 1999. Sixty-eight full-sib families were created from the gametes and reared at the Alaska SeaLife Center (ASLC) in Seward, Alaska. Eggs collected from each female were divided into two equal groups and each group was fertilized with sperm from one male. Each family was placed in a separate tray of a Heath rack and incubated in fresh water at 4–5°C. Embryos were raised following the procedures of Lindner et al. (2000).

In February 2000, approximately 25,000 fry (young fish) from 67 full-sib families were pooled. In April 2000 we collected a sample of 500 fry, marked the remaining fry by clipping the adipose fins, and subsequently released 24,216 marked individuals into Resurrection Bay. In August and September 2001, we collected 260 marked adult progeny by snag-hooking near the freshwater outlet at the ASLC where the fry were released, seining the rivers in upper Resurrection Bay, and holding a lottery to encourage recreational fishermen to turn in marked fish. Progeny therefore spent approximately 8 of 24 months (one-third of their lives) in the lab and 16 of 24 months (two-thirds of their lives) in the ocean. Progeny were caught from 63 of the 67 released families. The mean number of progeny caught from each of these families was 4.1 (range 1–11).

### Assignment of Parentage and Sex

DNA was extracted from fin clips or other collected tissues with the Puregene DNA isolation kit (Gentra Systems Inc., Minneapolis, MN). We analyzed each of the 68 parents

at nine microsatellite loci, including one duplicated locus (*SSA20.19-1,2*) and a growth hormone locus (*GH2*). Loci were amplified according to the original authors with minor modifications. Primers and annealing temperatures were as follows: *OmyRgt6*, 58–52°C (Sakamoto et al. 2000); *Ots1*, 56°C (Banks et al. 1999); *Ssa408*, 60°C (Cairney M, personal communication, 1997); *Ogo1c* and *Omy301*, 60°C and *Ogo8*, 58–52°C (Olsen et al. 1998); *Oneμ3*, 52°C (Scribner et al. 1996); *Ssa20.19-1,2*, 58–52°C (Sanchez et al. 1996); and *GH-2* intron C, 51°C (Spruell et al. 1999). Products from unlabeled primers were fluorescently tagged with TAMRA-labeled dUTP. Polymerase chain reaction (PCR) products were resolved via electrophoresis on denaturing 4.5% polyacrylamide gels and visualized and scored using a Hitachi FMBIO II fluorescent imager.

The returning adult progeny were analyzed at all 10 of these loci. Each fish was initially scored at three microsatellite loci (*OTS1*, *OMYRGT6*, and *SSA408*) and a list of possible families was made for each fish at each locus based on which alleles were carried by the parents of each family. Families not possible at all loci were eliminated, which reduced the number of possible families for most fish to between one and three. Fish were then run on gels next to possible parents for the remaining loci. After each locus was run, the alleles of each fish were compared with the alleles of potential parents, the list of possible families for each individual was revised, and the order of samples was changed so that fish were run beside parents and siblings. This allowed unambiguous family assignment of all fish and detection of any progeny with alleles whose lengths differed from those of their parents (mutants). Fish identified as mutants had allele combinations that placed them positively and uniquely in one family with the exception of a single allele that differed from the parental allele by one or two repeat units.

We also analyzed 240 of the 500 fry sampled prior to release at four loci (*OTS1*, *OMYRGT6*, and *SSA408*, and *OGO1C*), which allowed unambiguous assignment to families of all individuals as described above for adult progeny. Sex was assigned to fry using a Y chromosome-specific growth hormone pseudogene (Spruell et al. 1999). Presence of a 163 bp fragment indicated that a fish was male. However, eight sires lacked this diagnostic band, so the sex of the 49 fry in these families could not be determined.

### Morphological and Life-History Data Collection

We measured the fork length of adult parents from 1999, adult offspring from 2001, and progeny released as fry in 2000. Fork length was measured as the distance from the middle of the eye to the fork of the caudal fin. Pectoral and pelvic fin rays and upper and lower gill arch rakers were counted for parents and adult offspring using a dissecting microscope. Average individual egg weight was estimated by dividing the weight of 100 eggs by 100. Egg number was estimated by dividing total egg weight by the weight of 100 eggs and multiplying by 100. Egg weight was estimated after water absorption in the 1999 mothers, but immediately after catching fish in the 2001 daughters. Egg weight is there-

fore not comparable between mothers and daughters, but this inconsistency does not affect egg number estimates.

### Data Analysis

We compared the means of morphological and life-history traits between females and males and between parents and progeny using two-sample *t* tests. Variances were compared using *F* tests. Phenotypic correlations were calculated using Pearson correlations. All analyses were performed using MINITAB version 13.

We estimated heritabilities as the regression coefficients of regressions of family means against midparent values (Lynch and Walsh 1998). The significance of regression coefficients was assessed using *F* tests. Genetic correlations among traits were estimated from pairwise comparisons of parents and progeny (Lynch and Walsh 1998). Specifically, genetic correlations ( $\rho_A$ ) were estimated from the equation

$$\rho_A \cong \frac{\sigma(\bar{z}_{1x}, \bar{z}_{2y}) + \sigma(\bar{z}_{2x}, \bar{z}_{1y})}{2\sqrt{\sigma(\bar{z}_{1x}, \bar{z}_{1y}) \cdot \sigma(\bar{z}_{2x}, \bar{z}_{2y})}}, \quad (1)$$

where  $\sigma(\bar{z}_{1x}, \bar{z}_{2y})$  is the phenotypic covariance between midparents for trait 1 and offspring means for trait 2,  $\sigma(\bar{z}_{2x}, \bar{z}_{1y})$  is the covariance between midparents for trait 2 and offspring means for trait 1,  $\sigma(\bar{z}_{1x}, \bar{z}_{1y})$  is the covariance between midparents and offspring means for trait 1, and  $\sigma(\bar{z}_{2x}, \bar{z}_{2y})$  is the covariance between midparents and offspring means for trait 2. The significance of genetic correlations was determined as recommended by Lynch and Walsh (1998) from the regression of offspring means of trait 1 against midparent values of trait 2 and from the regression of family means of trait 2 against midparent values of trait 1, giving two *P* values for each genetic correlation. Finally, regressions of parental phenotypic correlations on genetic correlations and progeny phenotypic correlations on genetic correlations were used to test whether genetic correlations accurately predict phenotypic correlations.

## Results

### Phenotypic Variation and Phenotypic Correlations

Morphological and life-history trait means and variances differed between females and males and between parents and progeny (Table 1). Mean length of fathers was greater than mothers ( $t_{54} = 2.38$ ,  $P = .02$ ). Variance in length was also greater in males than females in both parents ( $n = 68$ ,  $F = 0.36$ ,  $P = .005$ ) and offspring ( $n = 257$ ,  $F = 0.42$ ,  $P < .001$ ). Mean length ( $t_{52} = 5.23$ ,  $P < .001$ ) and egg number ( $t_{57} = 11.29$ ,  $P < .001$ ) of daughters was greater than mothers. Similarly, the mean number of pectoral fin rays ( $t_{89} = 2.45$ ,  $P = .02$ ) and lower gill arch rakers ( $t_{98} = 5.81$ ,  $P < .001$ ) was greater in offspring than in parents. The variance in pectoral fin ray counts ( $n = 325$ ,  $F = 1.63$ ,  $P = .008$ ) and pelvic fin ray counts ( $n = 326$ ,  $F = 1.45$ ,  $P = .05$ ) was greater in parents than progeny, but variance in counts of upper gill arch rakers was greater in progeny than parents ( $n = 323$ ,  $F = 0.66$ ,  $P = .05$ ).

**Table 1.** Means and standard deviations for fork length, egg number, individual egg weight, and meristic traits of pink salmon parents and progeny from Likes Creek, Alaska

Trait	Parents		Progeny	
	Female	Male	Female	Male
Fry length	—	—	37.1 ± 3.6 (94)	36.2 ± 4.0 (93)
Length	456 ± 23 (34)	474 ± 39 (34)	480 ± 23 (122)	487 ± 36 (135)
Egg number	1191 ± 287 (34)	—	1836 ± 296 (104)	—
Egg weight	0.18 ± 0.01 (34)	—	0.15 ± 0.02 (110)	—
Pect	15.5 ± 0.6 (34)	15.8 ± 0.7 (34)	15.9 ± 0.5 (122)	15.8 ± 0.5 (135)
Pelv	10.8 ± 0.5 (34)	10.9 ± 0.5 (34)	10.8 ± 0.4 (122)	10.7 ± 0.4 (136)
UGA	13.1 ± 0.5 (34)	13.2 ± 0.5 (34)	13.1 ± 0.6 (121)	13.2 ± 0.7 (134)
LGA	17.4 ± 0.9 (34)	17.4 ± 0.7 (34)	18.0 ± 0.7 (121)	18.0 ± 0.7 (134)

Pect, pectoral fin rays; Pelv, pelvic fin rays; UGA, upper gill arch rakers; LGA, lower gill arch rakers.

All progeny values are for returning adults except for fry length, which was measured prior to release.

Sample sizes are shown in parentheses.

Length is in millimeters and egg weight is in grams; all other values are counts.

Eggs were weighed after water absorption for mothers and prior to water absorption for daughters, so egg weights are not comparable.

Phenotypic correlations are shown in Table 2. Many more pairs of traits had significant phenotypic correlations in progeny than in parents. In general, this is likely because of lower statistical power due to smaller sample sizes in parents. However, the phenotypic correlation between length and egg number was much lower in mothers (0.05) than in daughters (0.58) and likely reflects an actual difference between mothers and daughters.

### Heritabilities and Genetic Correlations

Heritabilities of all six morphological traits were significantly different from zero and ranged from 0.33 to 0.63 (Figures 1 and 2). Heritabilities ( $b^2$ ) were 0.34 for female length ( $F_{1,48} = 10.26$ ,  $P = .002$ ), 0.45 for male length ( $F_{1,53} = 8.85$ ,  $P = .004$ ), 0.55 for pectoral fin ray counts ( $F_{1,61} = 47.48$ ,  $P < .001$ ), 0.33 for pelvic fin ray counts ( $F_{1,61} = 8.47$ ,  $P = .005$ ), 0.63 for upper gill arch raker counts ( $F_{1,61} = 17.67$ ,  $P < .001$ ), and 0.45 for lower gill arch raker counts ( $F_{1,61} = 17.92$ ,  $P < .001$ ). In contrast, the two life-history traits examined—egg number ( $b^2 = -0.08$ ,  $F_{1,47} = 0.30$ ,  $P = .59$ ) and egg weight ( $b^2 = 0.22$ ,  $F_{1,48} = 2.62$ ,  $P = .11$ )—were not

significantly heritable. Moreover, neither female length ( $b^2 = 0.02$ ,  $F_{1,36} = 1.14$ ,  $P = .29$ ) nor male length ( $b^2 = 0.02$ ,  $F_{1,38} = 0.59$ ,  $P = .45$ ) were heritable when estimated using fry rather than adult offspring.

Genetic correlations were significant for four pairs of traits: pectoral fin rays and pelvic fin rays; pectoral fin rays and lower gill arch rakers; pelvic fin rays and upper gill arch rakers; and upper and lower gill arch rakers (Table 3). For the first and last of the above pairs of traits, both possible regressions of progeny family means on midparent values were significant. For the other two pairs of traits, only one of the two regressions was significant. Genetic correlations could not be estimated for pairs of traits including egg number because the covariance of mother and daughter egg number was negative, resulting in the square root of a negative product in equation 1. Similarly, genetic correlations could not be estimated for pairs of traits including egg weight because the covariance of mother and daughter egg weight was zero, resulting in a product of zero in the denominator of equation 1. However, regression analysis could still be used to test whether genetic correlations between egg number and other traits and between egg weight and other traits were

**Table 2.** Phenotypic correlations among traits of pink salmon from Likes Creek, Alaska

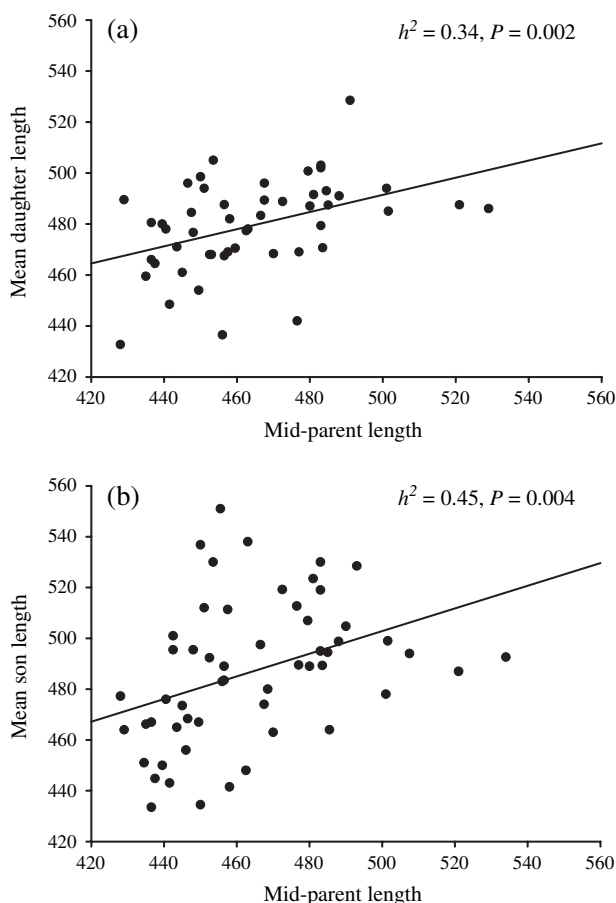
	Length	Egg no	Egg wt	Pect	Pelv	UGA	LGA
Length	—	0.05 (ns)	0.37 (0.03)	0.01 (ns)	0.010 (ns)	-0.02 (ns)	0.08 (ns)
Egg number	0.58 (<0.001)	—	-0.22 (ns)	-0.01 (ns)	0.23 (ns)	0.18 (ns)	0.18 (ns)
Egg weight	0.33 (<0.001)	0.07 (ns)	—	0.12 (ns)	0.10 (ns)	0.19 (ns)	0.16 (ns)
Pect	-0.15 (0.02)	0.01 (ns)	-0.23 (0.01)	—	0.02 (ns)	0.16 (ns)	0.12 (ns)
Pelv	-0.01 (ns)	0.03 (ns)	-0.06 (ns)	0.30 (<0.001)	—	0.16 (ns)	0.02 (ns)
UGA	0.05 (ns)	0.18 (ns)	0.26 (0.01)	0.04 (ns)	0.08 (ns)	—	0.35 (0.003)
LGA	0.14 (0.02)	0.10 (ns)	0.18 (ns)	0.02 (ns)	0.15 (0.02)	0.50 (<0.001)	—

Correlations above the diagonal are for parents and those below the diagonal are for progeny.

$P$  values are shown in parentheses for correlations significant at the  $\alpha = 0.05$  level.

“ns” indicates that a correlation was not significant.

Length, fork length; Pect, pectoral fin rays; Pelv, pelvic fin rays; UGA, upper gill arch rakers; LGA, lower gill arch rakers.

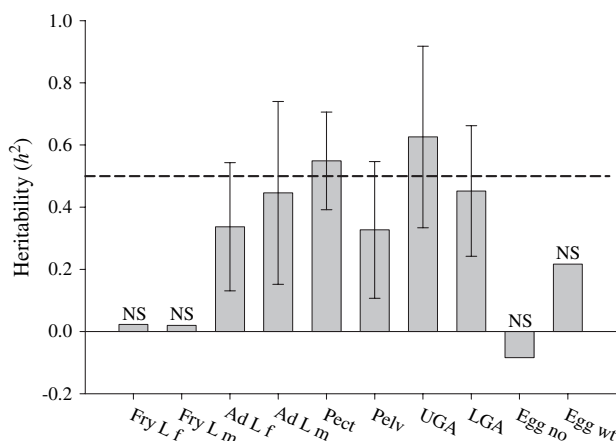


**Figure 1.** Regressions of (a) mean daughter fork length and (b) mean son fork length on midparent fork length. The heritability of length is the slope of the regression line.

significant. No genetic correlations were significant between egg number and any other trait nor between egg weight and any other trait.

**Regression of Phenotypic Correlations on Genetic Correlations**

The regression of phenotypic correlations on genetic correlations is positive using parent phenotypic correlations and progeny phenotypic correlations, but only the regression using progeny phenotypic correlations is significant (Figure 3). A few noteworthy outlier data points were observed. Some pairs of traits had high phenotypic correlations, but low genetic correlations. The genetic correlation between length and egg weight was indistinguishable from zero, but the phenotypic correlation between these traits was 0.37 in parents and 0.33 in progeny (Table 2). Similarly the genetic correlation between length and egg number was not significantly different from zero, but the phenotypic correlation between these traits was 0.58 in progeny (Table 2). Conversely, other pairs of traits had low phenotypic correlations, but high genetic correlations. Pelvic and pectoral fin ray



**Figure 2.** Heritability estimates and 95% confidence intervals for female and male fry fork length (Fry L f and Fry L m, respectively), female and male adult fork length (Ad L f and Ad L m, respectively), pectoral (Pect) and pelvic (Pelv) fin ray counts, upper (UGA) and lower (LGA) gill raker counts, egg number (Egg no), and egg weight (Egg wt) in pink salmon from Likes Creek, Alaska. NS indicates heritabilities that were not significantly different from zero.

counts had a high genetic correlation of 0.64 (Table 3), but a phenotypic correlation of only 0.02 in parents (Table 2). Likewise, pelvic fin rays and upper gill arch rakers had a genetic correlation of 0.76 (Table 3), but a phenotypic correlation of only 0.08 in progeny (Table 2).

**Discussion**

**Heritabilities of Morphological and Life-History Traits**

Our heritability estimates for pink salmon corroborate previous studies demonstrating higher heritabilities for morphological traits than life-history traits (Gustafsson 1986; Kruuk et al. 2000; Mousseau and Roff 1987). Heritabilities of the six morphological traits we examined ranged from 0.33 to 0.63, whereas the heritabilities of egg number and egg

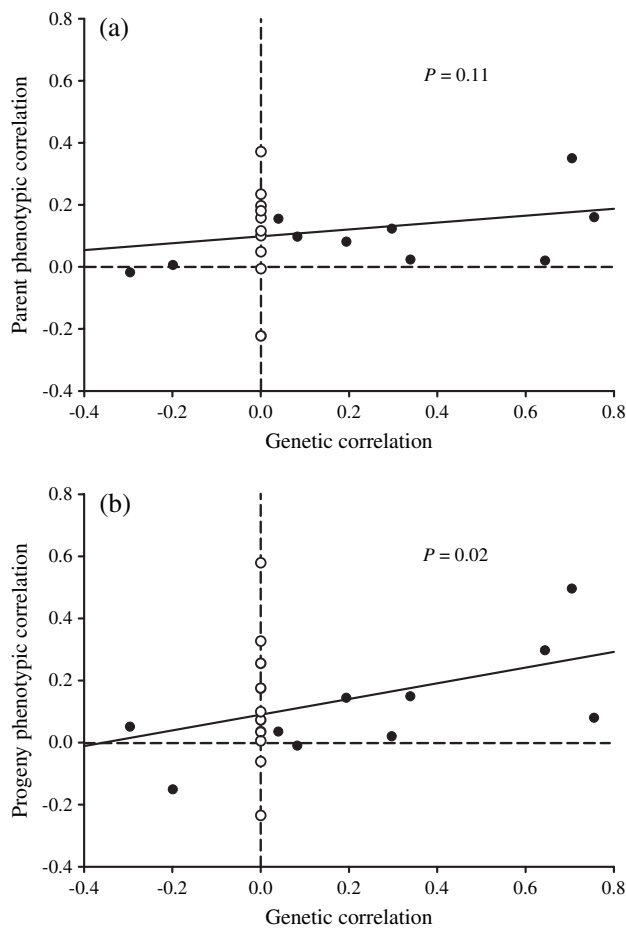
**Table 3.** Genetic correlations among traits of pink salmon from Likes Creek, Alaska

	Length	Pect	Pelv	UGA
Pect	-0.20 (ns)	—	—	—
Pelv	0.08 (ns)	0.64 (0.02, 0.01)	—	—
UGA	-0.30 (ns)	0.04 (ns)	0.76 (0.57, <0.001)	—
LGA	0.19 (ns)	0.30 (0.91, 0.02)	0.34 (ns)	0.71 (0.04, 0.001)

P values are shown in parentheses for both possible regressions of progeny family means against midparent values for each pair of traits for cases in which at least one regression was significant at the  $\alpha = 0.05$  level.

“ns” indicates that neither regression was significant.

Length, fork length; Pect, pectoral fin rays; Pelv, pelvic fin rays; UGA, upper gill arch rakers; LGA, lower gill arch rakers.



**Figure 3.** Regressions of (a) parent phenotypic correlations and (b) progeny phenotypic correlations on genetic correlations among phenotypic traits in pink salmon from Likes Creek, Alaska. Genetic correlations that could not be estimated because of negative or zero covariances between parental and offspring traits are represented by open circles (see text for details).

weight were indistinguishable from zero (Figure 2). Our heritability estimates for the four meristic traits examined (pectoral fin rays, 0.55; pelvic fin rays, 0.33; upper gill arch rakers, 0.63; and lower gill arch rakers, 0.45) were similar to heritability estimates for these same traits in rainbow trout (0.52, 0.84, 0.67, and 0.37, respectively) (Leary et al. 1985). Our heritability estimate for egg weight in Likes Creek pink salmon (0.22) was also similar to a heritability estimate for egg weight in captive Chinook salmon of 0.26 (Heath et al. 2003), although egg weight was not significantly heritable for pink salmon, but was significant for Chinook salmon.

The observation of higher heritabilities for morphological traits than for life-history traits suggests that in this population of pink salmon, the morphological traits examined are expected to respond more strongly to selection than egg number and egg weight. It also suggests that egg number and egg weight have been more strongly selected in the past than length, fin ray counts, and gill arch raker counts.

Because pink salmon are semelparous, like all Pacific salmon, there is likely very strong directional selection for increased egg number. There also may be directional selection for increased egg weight, since egg weight is positively related to embryo and fry survival in captive Chinook salmon (Heath et al. 2003). Alternatively, low heritabilities of life-history traits may reflect high levels of environmental variance in these traits (Price and Schluter 1991).

Although length was significantly heritable when estimated using adult progeny, it was not significant when estimated using progeny in the fry stage (Figure 2). This suggests that the inherited variation in physiological and behavioral traits that influence the length of returning spawners is not expressed until the oceanic life-history stages. This result is in agreement with previous work showing that male progeny sired by large males do not begin growing faster than progeny sired by small males until the spring of the year of maturity (Beacham and Murray 1988). In contrast, heritabilities of meristic traits can be estimated from regressions of juvenile offspring on adult parents because final meristic counts are usually determined early in development. For example, Leary et al. (1984) found no significant differences in meristic counts of rainbow trout analyzed 182 days after fertilization and fish from the same families analyzed 394 days after fertilization.

### High Genetic Correlations of Nonadjacent Morphological Traits

We found that pairs of nonadjacent morphological traits had genetic correlations as high as pairs of adjacent traits (Table 3). Genetic correlations between pectoral and pelvic fin rays, pectoral fin rays and lower gill arch rakers, and pelvic fin rays and upper gill arch rakers ranged from 0.30 to 0.76, similar to the genetic correlation of 0.71 between upper and lower gill arch rakers, which are adjacent. The high genetic correlation between pectoral and pelvic fin ray counts is not surprising since they are similar structures and therefore might be expected to be under similar gene control. However, the observation of high genetic correlations between pectoral fin rays and lower gill arch rakers and between pelvic fin rays and upper gill arch rakers is somewhat surprising since they are both nonadjacent and different “types” of structures. This result suggests that different types of morphological traits may be regulated by similar genes regardless of the distance among the traits. This explanation is supported by the work of Leary et al. (1984), showing that expression of *Pgm1* by rainbow trout heterozygous or homozygous for a rare allele at the regulatory *Pgm1-t* locus developed faster and had lower counts of all meristic traits than individuals homozygous for the common allele.

### Prediction of Phenotypic Correlations from Genetic Correlations

In general, genetic correlations accurately predicted phenotypic correlations for this population of pink salmon, as expected (Figure 3). The regression of parental phenotypic correlations on genetic correlations was not significant,

but this was likely due to relatively imprecise estimates of phenotypic correlations for parents due to fairly small sample sizes. The positive relationship between genetic correlations and phenotypic correlations in this population of pink salmon is in line with other studies showing a similar trend (Cheverud 1988, 1995; Roff 1995, 1996). Another similarity between this study and others is that the regression of phenotypic correlations on genetic correlations is less than unity (Kohn and Atchley 1988; Koots et al. 1994; Searle 1961). Some have argued that the regression coefficient converges on one when sampling variance is removed (Cheverud 1988, 1995; Roff 1995, 1996), but Willis et al. (1991) challenged the results of Cheverud (1988). Because phenotypic correlations are a function of genetic correlations as well as environmental correlations, there should be some correspondence between phenotypic and genetic correlations. However, further study is needed to understand whether the observation of smaller phenotypic correlations than genetic correlations is due entirely to sampling error or whether it has a biological explanation.

The outlier data points of the regressions of phenotypic correlations on genetic correlations provide insights into the interactive effects of genetic and environmental variation on phenotypic correlations. Pairs of traits with high phenotypic correlations despite low genetic correlations are likely phenotypically correlated because of a common effect of environmental variation on the two traits in question. For Likes Creek pink salmon, such pairs of traits included length and egg weight and length and egg number. Therefore, whatever environmental factors cause an increase in fish length, perhaps food resources, apparently cause an increase in egg weight and egg number.

In contrast, pairs of traits with high genetic correlations, but low phenotypic correlations, have low phenotypic correlations because of environmental variation that acts in opposition to the genetic correlations. The phenotypic correlation between pelvic and pectoral fin ray counts was only 0.02 in parents despite a genetic correlation of 0.64 in Likes Creek pink salmon, suggesting that common environmental variation has opposing effects on these two traits. Pelvic fin rays and upper gill arch rakers also had a phenotypic correlation of 0.08 in progeny despite a genetic correlation of 0.76, once again pointing to a negative environmental correlation between these two traits.

### High Variability of Male Length

Length was significantly more variable among males than among females, as has been observed previously for pink salmon (Table 1) (Beacham and Murray 1985). Beacham and Murray (1985, 1988) suggest that the high variability of male length in pink salmon may stem from alternative large- and small-male breeding strategies. They note that small males resemble females, suggesting that small males may mimic females to reduce aggression from larger males.

A prediction of Beacham and Murray's hypothesis for high variability of male length in pink salmon is that length should not be more variable in males than in females within

year classes in other salmonid species in which there are multiple year classes of males on the spawning grounds. In species with multiple year classes on the spawning grounds, younger males are so much smaller than older males that there would be no advantage of being a small, older male. Therefore all males that return to spawning grounds later should be selected to grow large and there should be little variability in length within year classes. We are unaware of any published studies showing a greater variability of male length than female length within year classes for any salmonid species other than pink salmon, supporting Beacham and Murray's hypothesis. However, within-year class variability may be overlooked in salmon species with multiple year classes present on the spawning grounds because the difference in size among year classes is so much greater than length variability within year classes.

### Generational Variation in the Phenotypic Correlation Between Length and Egg Number

The high phenotypic correlation observed between length and egg number observed in daughters was not seen in mothers. This highlights the fact that phenotypic correlations, like phenotypic variation, are highly dependent on environmental variation. Whatever common environmental factor caused the strong phenotypic correlation between length and egg number in daughters was not present in the parental environment.

Moreover, this observation has important implications for the relationship between fitness, defined as the number of returning progeny per parent, and length in pink salmon. If fitness is proportional to egg number and egg number is phenotypically correlated with length only in some years, one should expect to see temporal variation in the relationship between length and fitness. Our data suggest that fitness should be correlated with egg number because no negative phenotypic or genetic correlation was observed between egg number and egg weight, suggesting that there is no tradeoff between fecundity and egg size in this population. The lack of a phenotypic correlation between length and egg number in the parents suggests that length should not be correlated with fitness in this generation, assuming that other factors do not influence the relationship between fitness and length. In fact, no relationship was seen between fitness and length during the parental generation (unpublished data). In contrast, the high positive correlation between length and egg number in the offspring generation suggests that there should be a strong correlation between length and fitness in this generation.

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## References

- Banks MA, Blouin MS, Baldwin BA, Rashbrook VK, Fitzgerald HA, Blankenship SM, and Hedgecock D, 1999. Isolation and inheritance of novel microsatellites in chinook salmon (*Oncorhynchus tshawytscha*). *J Hered* 90:281–288.
- Beacham TD and Murray CB, 1985. Variation in length and body depth of pink salmon (*Oncorhynchus gorbuscha*) and chum salmon (*Oncorhynchus keta*) in southern British Columbia, Canada. *Can J Fish Aquat Sci* 42:312–319.
- Beacham TD and Murray CB, 1988. A genetic analysis of body size in pink salmon (*Oncorhynchus gorbuscha*). *Genome* 30:31–35.
- Boag PT and Grant PR, 1978. Heritability of external morphology in Darwin's finches. *Nature* 274:793–794.
- Cheverud JM, 1988. A comparison of genetic and phenotypic correlations. *Evolution* 42:958–968.
- Cheverud JM, 1995. Morphological integration in the saddle-back tamarin (*Saguinus fuscicollis*) cranium. *Am Nat* 145:63–89.
- Conner J and Via S, 1993. Patterns of phenotypic and genetic correlations among morphological and life-history traits in wild radish, *Raphanus raphanistrum*. *Evolution* 47:704–711.
- Endler JA, 2000. Adaptive genetic variation in the wild. In: *Adaptive genetic variation in the wild*, vol 1 (Mousseau TA, Sinervo B, and Endler J, eds). New York: Oxford University Press; 251–260.
- Fisher RA, 1930. *The genetical theory of natural selection*. New York: Dover.
- Gustafsson L, 1986. Lifetime reproductive success and heritability: empirical support for Fisher's fundamental theorem. *Am Nat* 128:761–764.
- Heath DD, Heath JW, Bryden CA, Johnson RM, and Fox CW, 2003. Rapid evolution of egg size in captive salmon. *Science* 299:1738–1740.
- Kohn LA and Atchley WR, 1988. How similar are genetic correlation structures? Data from mice and rats. *Evolution* 42:467–481.
- Koots KR, Gibson JP, and Wilson JW, 1994. Analyses of published genetic parameter estimates for beef production traits. 2. Phenotypic and genetic correlations. *Anim Breed Abstr* 62:825–853.
- Kruuk LE, Clutton-Brock TH, Slate J, Pemberton JM, Brotherstone S, and Guinness FE, 2000. Heritability of fitness in a wild mammal population. *Proc Natl Acad Sci USA* 97:698–703.
- Leary RF, Allendorf FW, and Knudsen KL, 1984. Major morphological effects of a regulatory gene: *Pgm1-t* in rainbow trout. *Mol Biol Evol* 1: 183–194.
- Leary RF, Allendorf FW, and Knudsen KL, 1985. Inheritance of meristic variation and the evolution of developmental stability in rainbow trout. *Evolution* 39:308–314.
- Lindner KR, Seeb JE, Habicht C, Knudsen KL, Kretschmer E, Reedy DJ, Spruell P, and Allendorf FW, 2000. Gene-centromere mapping of 312 loci in pink salmon by half-tetrad analysis. *Genome* 43:538–549.
- Lynch M and Walsh B, 1998. *Genetics and analysis of quantitative traits*, vol 1. Sunderland, MA: Sinauer Associates.
- Mousseau TA and Roff DA, 1987. Natural selection and the heritability of fitness components. *Heredity* 59:181–197.
- Olsen JB, Bentzen P, and Seeb JE, 1998. Characterization of seven microsatellite loci derived from pink salmon. *Mol Ecol* 7:1087–1089.
- Price T and Schluter D, 1991. On the low heritability of life-history traits. *Evolution* 45:853–861.
- Roff DA, 1995. The estimation of genetic correlations from phenotypic correlations: a test of Cheverud's conjecture. *Heredity* 74:481–490.
- Roff DA, 1996. The evolution of genetic correlations: an analysis of patterns. *Evolution* 50:1392–1403.
- Roff DA, 1997. *Evolutionary quantitative genetics*, vol 1. Montreal: Chapman & Hall.
- Sakamoto T, Danzmann RG, Gharb K, Howard P, Ozaki A, Khoo SK, Worman RA, Okamoto N, Ferguson MM, Holm L-E, Guyomard R, and Hoyheim B, 2000. A microsatellite linkage map of rainbow trout (*Oncorhynchus mykiss*) characterized by large sex-specific differences in recombination rates. *Genetics* 155:1331–1345.
- Sanchez JA, Clabby C, Ramos D, Blanco G, Flavin F, Vazquez E, and Powell R, 1996. Protein and microsatellite single locus variability in *Salmo salar* L. (Atlantic salmon). *Heredity* 77:423–432.
- Scribner KT, Gust JR, and Fields RL, 1996. Isolation and characterization of novel microsatellite loci: cross-species amplification and population genetic applications. *Can J Fish Aquat Sci* 53:685–693.
- Searle SR, 1961. Phenotypic, genetic and environmental correlations. *Biometrics* 17:474–480.
- Spruell P, Pilgrim KL, Greene BA, Habicht C, Knudsen KL, Lindner KR, Olsen JB, Sage GK, Seeb JE, and Allendorf FW, 1999. Inheritance of nuclear DNA markers in gynogenetic haploid pink salmon. *J Hered* 90: 289–296.
- Weber KE, 1992. How small are the smallest selectable domains of form? *Genetics* 130:345–353.
- Willis JH, Coyne JA, and Kirkpatrick M, 1991. Can one predict the evolution of quantitative characters without genetics? *Evolution* 45:441–444.
- Wright S, 1968. *Evolution and the genetics of populations*. I. Genetic and biometric foundations. Chicago: University of Chicago Press.
- Young HJ, Stanton ML, Ellstrand NC, and Clegg JM, 1994. Temporal and spatial variation in heritability and genetic correlations among floral traits in *Raphanus sativus*, wild radish. *Heredity* 73:298–308.

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