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SHIFTING PATTERNS IN GENETIC CONTROL AT THE EMBRYO-ALEVIN BOUNDARY IN BROOK CHARR

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Abstract.—Maternal inputs to offspring early in development are initially high but the process of development suggests that ontogenetic shifts in the importance of maternal genetic variation relative to other sources should occur. We investigated additive genetic variance and covariance for direct (animal), sire, and maternal effects on embryonic length (EL), yolk sac volume (YSV), and alevin (after yolk sac resorption) length (AL) for 460 embryonic and 460 alevin brook charr (*Salvelinus fontinalis*) in 23 half-sib families (12 sires, 23 dams). There were no additive genetic effects of sires or individual animals on their own phenotype using sire-dam and maternal-animal models for YSV or EL ($h_a^2 < 0.05$). However, at the alevin stage we detected low but significant heritability for AL ($h_a^2 = 0.14 \pm 0.11$). Conversely, maternal genetic effects were high for both embryonic traits ($h_{EL}^2 = 0.61 \pm 0.05$; $h_{YSV}^2 = 0.57 \pm 0.06$) but faded rapidly for postresorption length ($h_{AL}^2 = 0.18 \pm 0.04$). Maternal effects in the sire-dam model corresponded highly with those in the animal-dam model. We did not detect significant genetic covariance between progeny and dams for preresorption traits or between sires and dams for any trait. However, following resorption of the yolk sac, the genetic value of dams for AL was negatively correlated with that of individual progeny ($r_{m,a} = -0.38 \pm 0.13$), suggesting trade-offs and/or stabilizing selection between maternal and animal genetic trait value. This finding was supported by models of dam fecundity on offspring length and dam weight in phenotypic space. Heritability estimates using simple regression of embryo phenotype on adult parental phenotype produced upwardly biased estimates of genetic variance ($h^2 > 1.0$). We propose that development through the embryo-alevin boundary may be a major point in salmonids for ontogenetic changes in the genetic architecture of embryo size from maternal genetic effects to those of the individual organism, and that maternal-offspring conflicts in resource allocation related to size may be partially indicated by negative genetic covariance.

Key words.—Brook charr, direct genetic, heritability, maternal-animal covariance, maternal genetic, stabilizing selection.

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The expression of maternal effects on offspring mirrors that of classical quantitative genetic theory: early offspring phenotype is partially controlled by common-environmental effects of dams and genetic variance for reproductive investment by dams into their offspring (Cheverud and Moore 1994; Falconer and Mackay 1996; Lynch and Walsh 1998; Mousseau and Fox 1998; Heath et al. 1999). Both maternal variance and estimates of maternal genetic effects are generally high (h^2, $m^2 > 0.4$) for production traits in domesticated animals (Koerhuis and Thompson 1997; Chen et al. 2002; Splan et al. 2002; Hartmann et al. 2003; Van Vleck et al. 2003). Maternal genetic and environmental effects are in all likelihood ubiquitous and considerable (Roach and Wulff 1987; Bernardo 1996a; Mazer and Gorchov 1996; Mousseau and Fox 1998; Wolf et al. 1998), and thus the potential evolutionary role of maternal genetic variation may be profound (Byers et al. 1997; Thiede 1998; McAdam et al. 2002).

However, consideration of the evolutionary role of maternal variation (including maternal genetic variance) seems to have been confined largely to the Mammalia (see Cheverud and Moore 1994; Mousseau and Fox 1998; Wolf et al. 1998; comments in McAdam et al. 2002). Maternal genetic effects are probably less complex in oviparous species compared to mammalian uterine development and lactation after parturition (Falconer and Mackay 1996; Lynch and Walsh 1998; Wolf et al. 1998), but there is evidence of significant maternal genetic effects on early size in egg-laying species such as chickens (Koerhuis and Thompson 1997; Hartmann et al. 2003) and salmonid fishes (Osteichthyes: Salmonidae; Heath et al. 1999; Martínez et al. 1999; Pakkasmaa and Jones 2002). The genetics of early morphological development is likely of crucial importance to the fitness of salmonids because of high embryonic mortality (>80%) and the relationship of morphological (i.e., body size) traits to fitness during this period (Elliott 1986; Einum and Fleming 2000a,b, 2002; Svensson and Sinervo 2000). In salmonids, the critical medium for the transmission of maternal genetic variance should be resource deposition in the yolk sac since parental care is essentially nonexistent. Ontogenetic decreases in maternal genetic value coupled with increases in paternal and/or animal-based estimates of genetic variation observed in parturitive species should be most profound for salmonids and other egg-laying fish at the developmental juncture between the embryonic sac fry (which still retains the yolk sac) and the alevin stage (after resorption of this structure). There is some evidence of ontogenetic change in paternal and maternal genetic (Kinghorn 1983; Withler et al. 1987; Hebert et al. 1998; Heath et al. 1999) and environmental effects in salmonid fishes, but specific associations of the embryo/alevin boundary with changes in maternal genetic variance have not been examined (see also Pakkasmaa and Jones 2002). Moreover, many such estimates in this taxon have

used more classical ANOVA- and regression-based methods for the estimation of genetic variance, possibly incorporating environmental variance from parents, particularly on the maternal side, in the estimation of heritability. The separation of maternal environmental variance from maternal genetic effects might provide additional insight into the role of maternal control of embryonic traits.

Maternal variance may also affect progeny phenotype via maternal-animal genetic covariance (covariation between the effects of genes expressed by dams and those expressed within their progeny; Lynch and Walsh 1998; Wolf et al. 1998; Wolf 2000). Evidence for this phenomenon is persistent (i.e., Roff 1997; Notter 1998; El Fadili et al. 2000; Maniatis and Pollott 2003), although the genetic basis of such correlations is still under some debate (i.e., Maniatis and Pollott 2003). Maternal-animal covariance may also impact evolution indirectly via the maintenance of linkage disequilibrium, local adaptation on fitness landscapes, and genetic divergence of subpopulations (Yazdi et al. 1997; Wolf et al. 1998; Wolf 2000; McAdam et al. 2002). Interaction between maternal and (direct) animal genetic effects (termed intergenomic epistasis, IGE; Wolf et al. 1998) is predicted where the partial disassociation of these effects occurs for a trait affected by maternal variance and selected at the embryonic level (Wolf et al. 1998; Wolf 2000; Boujenane and Kansari 2002). Where IGE underlies negative maternal-animal genetic correlations, fitness trade-offs between maternal parents and their offspring should cause stabilizing selection on the relevant trait (Wolf 2000) due to limitations in the allocation of resources (Trivers and Willard 1973; Trivers 1974; Elgar 1990; Sinervo and Licht 1991; Bernardo 1996b; Einum and Fleming 2000a,b). Alternatively, positive genetic correlations between breeding values of dams and offspring (Näsholm and Danell 1996; Yazdi et al. 1997) could occur from synergistic contributions made by dams without compromising their own fitness (see Wolf et al. 1998; McAdam et al. 2002) and should result in directional selection at the embryonic level (Wolf 2000). The latter seems more likely for species with postpartum care, since specific dams may be able to regulate energetic contributions at lactation depending on environmental quality (see Trivers and Willard 1973; Trivers 1974). Negative maternal-animal genetic correlation might be more common in egg-laying species without adult care because total energetic investment is determined prior to oviposition without much ecological recourse to post-hatch investment. However, trends are not readily apparent for the latter group from the limited literature to date (see Koerhuis and Thompson 1997; Martínez et al. 1999; Hartmann et al. 2003).

In this context we examined maternal and genetic variance and covariance for yolk sac volume and length at the embryo and alevin developmental stages in juvenile brook charr (*Salvelinus fontinalis*). We hypothesized that (1) maternal control of embryonic characters would be high early in development compared to variance from sires and from individual animals, and (2) covariance between direct (animal) genetic and maternal genetic effects would be negative. We also established two additional objectives: (1) the modeling of relative fitness values in phenotypic space to determine whether relationships among embryo traits, dam traits, and fecundity would be indicative of stabilizing selection and (2) the evaluation of heritability estimates generated using parent-offspring regression for the identification of potential bias.

Materials and Methods

Breeding Design and Early Rearing

A total of 12 half-sib along (23 full-sib) families of brook charr were bred from 12 sires and 23 dams from mid- November 2002 to the beginning of January 2003. Parental fish were derived from two sources exhibiting alternate life-history strategies. Six sires and 12 dams were collected from the second pure strain generation of a stock maintained at the Station Aquicole de Pointe-au-Père, Université de Québec à Rimouski (Rimouski, Québec, Canada) descended from anadromous (sea migrant) fish collected from the Laval River (Québec, Canada). The other six sires and 11 dams (see Appendix 1) were resident (nonmigratory) fish collected directly from the Adams Brook (a Laval River tributary) in the summers of 2001 and 2002. Six pure anadromous, six pure resident, and 12 resident-anadromous hybrid full-sib families were created from these parents (Appendix 1). The use of these groups permits cursory analysis of genetic architecture while also permitting examinations of the role of specific population (resident or anadromous) on the control of quantitative traits. Sire and dam weight were collected at spawning. Most of the crosses were arranged so that one dam was a second-generation anadromous (Laval River) individual and the other a wild-caught resident (Adams Brook) within each of the half-sib crosses. Families were reared at 8°C on a 12:12-h light:dark photoperiod during the experiment.

A sample of 20 individuals from each pure strain and hybrid full-sib family ($n = 460$) were measured post-hatch at 100% eclosion by family, at a maximum of 24 h post-hatch (see Appendix 1). For each individual, embryonic length (distance from the snout tip to the tip of the caudal fin, EL; mm), yolk sac length (YSL; mm), and yolk sac diameter (mm) were measured to the nearest 0.1 mm using a binocular dissecting microscope. The standard cylindrical relationship of $\text{YSV} = \pi \times \text{YSL} \times r^2$ was used as an estimate of yolk sac volume (YSV; mm^3), where r represents the yolk sac radius. An additional sample of 20 individuals was taken from each full-sib family following complete (100%) resorption of the yolk sac for the entire family (alevin stage; $n = 460$). Alevin length (AL; mm) was measured and recorded using standard calipers for each of these 20 individuals.

Data Treatment

Trait normality was tested using the Shapiro-Wilk W function (Shapiro and Wilk 1965). Optimal exponential transformations (those minimizing root mean squared error) were determined by a Box-Cox transformation macro (BoxCox, M. Friendly, York University, Ontario, Canada) and applied where necessary to normalize ($P > 0.05$) traits.

Direct Animal, Maternal, and Maternal-Direct Animal Additive Variance

Maternal-animal and sire-dam models

All estimates of genetic variance were made using the programs PEST3.0 (Parameter Estimation Software; Groeneveld

et al. 1990) and VCE 4.1 (Variance Component Estimator; Groeneveld 1994). PEST was used to initially code trait and pedigree data for determination of first-round estimates of environmental and additive genetic variance by VCE using restricted maximum likelihood (REML). These first-round estimates were used in conjunction with phenotypic measurements to calculate best linear unbiased predictors (BLUP) estimated breeding values (EBVs) using PEST. Individual EBVs and the pedigree were in turn used to create final estimates for genetic and environmental variance in VCE including animal and maternal-based heritability (h_a^2) and maternal-animal genetic covariance ($r_{m,a}$). Stopping points for parameter estimate changes between iterations for PEST/VCE were set at program defaults for convergence (ratio change in the genetic variance parameters). Standard errors for variance components were estimated using VCE 4.1 (Groeneveld 1994).

We estimated additive genetic variance and covariance from direct (animal; genetic effects of animals on their own phenotype) and maternal genetic sources from the perspective of the Falconer (1965)/Kirkpatrick-Lande (Kirkpatrick and Lande 1989) approach of modeling environmental elements of maternal phenotype on progeny phenotype (see also Willham 1963, 1972; Cheverud 1984; Roff 1997). Unfortunately, parental phenotype for the embryonic traits measured here was not available because of logistical and biological limitations of early rearing for salmonid embryos. However, the critical role of dam weight with spawning period, progeny size, and fecundity is well known in the Salmonidae (Crandell and Gall 1993; Quinn et al. 1995; Einum and Fleming 2000a,b; Hendry et al. 2001). We therefore included dam weight as a maternal covariate associated with the embryonic characters in the model

$$\sigma_p^2 = \sigma_{a,a}^2 + \sigma_{a,m}^2 + \sigma_a(o,m) + \sigma_m^2 + \sigma_e^2,$$

where σ_p^2 is the phenotypic variance of the offspring individuals, $\sigma_{a,a}^2$ represents phenotypic variance explained by direct additive animal genetic effects of genes carried by offspring individuals, $\sigma_{a,m}^2$ is additive maternal genetic variance resulting from genes carried by dams, $\sigma_a(o,m)$ is the additive covariance between offspring and maternal genetic effects, σ_m^2 represents that proportion of phenotypic variance controlled by nongenetic effects of maternal phenotype (dam weight), and σ_e^2 represents error variance including that experienced by the maternal parent (σ_c^2) and variance derived from other environmental sources (σ_e^2). This model might thus be classified as a ''case 2'' maternal-animal model as described by Roff (1997), in which progeny phenotype is affected by maternal variation in other associated characters. In matrix notation, this model was

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Z_a a_a} + \mathbf{Z_m a_m} + \mathbf{Z_c c} + \mathbf{e},$$

where for trait *i*, **y** represents the vector of phenotypic observations, **X** is the incidence matrix of fixed effects, **b** is the vector of fixed effects, $\mathbf{Z_a}$ is the incidence matrix of (direct) random genetic animal effects, $\mathbf{a_a}$ is the vector of (direct) random animal genetic effects, $\mathbf{Z_m}$ is the incidence matrix of random maternal genetic effects, $\mathbf{a_m}$ is the vector of random maternal genetic effects, $\mathbf{Z_c}$ is the incidence matrix of common permanent maternal environmental effects, **c** is the vector of common permanent maternal environmental effects, and **e** represents random error. Genetic models were fitted for each of the traits (EL, YSV, and AL) individually. Model intercepts were fitted as fixed effects. We also included degree-days as a regressive term for YSV and AL because degree-days age was associated with these two traits in stepwise determination of covariates ($P < 0.001$), but not for EL since no detectable effect of degree-days of age was seen during multivariate analysis ($P > 0.2$). Embryo length may not have been specifically affected by age at this point.

Theory predicts that negative genetic covariance between maternal and animal phenotype should be mediated by intergenomic epistasis (Wolf et al. 1998; Wolf and Brodie 1998; Wolf 2000). Stabilizing selection should be evident in cases where such antagonistic interactions occur, creating structure in adaptive phenotypic landscapes, particularly during juvenile life stages because of early mortality (Wolf and Brodie 1998; Wolf 2000). Since our findings suggested the likelihood of negative maternal-animal genetic covariance (see Results), we explored the landscape of relative fitness-phenotype associations (Wright 1932; Lande 1979; Provine 1986) in these families using reproductive output as a surrogate for fitness (see also Hendry and Day 2003). Fecundity (number of eggs produced; FEC) and effective fecundity (number of eggs fertilized; FERT) were not normally distributed ($P < 0.05$) and so both traits were transformed ($FEC^{0.2}$, $FERT^{0.2}$) using Box-Cox regression (Box-Cox macro, M. Friendly, York University, Ontario, Canada). Transformed fecundity and fertility are represented in the text by FEC and FERT from this point forward. We determined exponential transforms up to the cube exponent (x^3) for dam weight and mean EL and AL by full-sib family (dam). The significance of the association between each measurement of fecundity and transformed embryonic and parental values was evaluated using backwards selection starting with a completely saturated model (PROC REG; SAS 1998; for retention $P < 0.05$). Regression coefficients were identified for each related term from the final model identified as above for each measure of fecundity (PROC REG; SAS 1998). Model coefficients were then submitted into SigmaPlot (2000) to produce a three dimensional landscape of dam fecundity, mean embryo/alevin length by family and dam weight.

Regression-based heritability estimates

We also evaluated the applicability of parent-offspring phenotypic regression (Roff 1997; Lynch and Walsh 1998) for the estimation of heritability in our embryonic brook charr families. Regression-based heritability for EL, YSV, and AL were estimated using individual regression on weight and length measurements from sires and dams. Untransformed progeny and parental phenotype were standardized ($\mu = 0$, $\sigma^2 = 1$) prior to analysis to normalize data distributions by trait and to correct for potential differences in variance between the embryonic and parental characters due to phenotypic scaling (see Heath et al. 1999). Heritability was then estimated as twice the regression coefficient for the specific analysis (β; Falconer and Mackay 1996; Lynch and Walsh 1998). Standard error of the estimates was considered to be twice the estimate of the regression slope (Roff 1997).

TABLE 1. Arithmetic mean (μ), standard deviation (σ), minimum and maximum for embryonic length (EL), yolk sac volume (YSV), and alevin length (AL) in half-sib juvenile brook charr. EL is expressed in $mm^{1.4}$, AL in $mm^{0.5}$, and YSV as $(mm^3)^{0.4}$.

Trait	μ	σ	σ_p^2	Min	Max
EL	53.8	7.03	49.4	39.0	66.3
YSV	4.38	0.830	0.690	2.51	6.17
AL	5.11	0.321	0.103	3.39	7.46

RESULTS

Data Treatment Preanalysis

EL, YSV, and AL were non-normal initially ($P < 0.001$). Box-Cox testing identified that the exponential transformations of $EL^{1.4}$, $YSV^{0.4}$ and $AL^{0.5}$ as minimizing root mean squared error. All analysis was carried out on the transformed data, with the exception of the regression of progeny phenotype on maternal weight (see below). Phenotypic variance relative to the mean was highest for EL and lowest for AL (Table 1).

The identification of covariates for inclusion in analysis of heritability for the traits was performed with backwards stepwise regression (for acceptance $P < 0.10$, single trait removal per regression analysis) using several available traits including dam weight, degree-days at measurement, and degree-days at 100% eye-up by family. Dam weight (g) was strongly and positively associated with all three traits ($P < 0.0001$, Fig. 1). Degree-days of age post-hatch were also associated with both YSV and EL. Dam weight was much more strongly associated with EL ($r^2 = 0.67$; $t_{1,459} = 30.7$) and YSV ($r^2 = 0.74$; $t_{1,459} = 36.0$) than with AL ($r^2 = 0.17$; $t_{1,459} = 9.49$; Fig. 1). The coefficient of variation around the regression function with body weight was highest for YSV (CV = 9.70), intermediate for EL (CV = 7.49), and lowest for AL (CV = 5.75).

Direct Animal, Maternal, and Maternal-Direct Animal Additive Variance

All REML-based analyses in VCE 4.1 finished with status 1 at convergence (stop for burn-in point = 0.001) within 40 iterations of the program start and at log-likelihood ratios for total model acceptance in excess of $L > 250$. Animal-based estimates of h_a^2 were effectively zero for EL and YSV (Table 2), with all variance being assigned to residual and maternal sources. At the same time, estimates of maternal genetic variation were high for both EL ($h_a^2 = 0.61 \pm 0.05$) and YSV ($h_a^2 = 0.57 \pm 0.06$). Following resorption of the yolk sac, we detected low but positive animal-based heritability for AL ($h_{a,AL}^2 = 0.14 \pm 0.11$), whereas maternal genetic variance was low ($h_{a,AL}^2 = 0.18 \pm 0.04$). Maternal-animal genetic covariance was moderate-high for AL ($r_{m,a} = -0.38 \pm 0.13$) but essentially zero for the two embryonic traits (Table 2). Estimates of maternal genetic variance for the embryonic traits in the sire-dam model were also high ($h_a^2 > 0.5$) and not different from those in the maternal-animal model (Tables 2, 3). As in the maternal-animal model (above), maternal variance for length fell sharply following resorption of the yolk sac ($h_{a,AL}^2 = 0.15 \pm 0.05$). Also as in the maternal-animal models, genetic effects of sires on their offspring approached zero for both EL and YSV. The estimate of sire genetic variance for AL was not significantly different from zero (Table 3). There was no evidence of significant genetic covariance

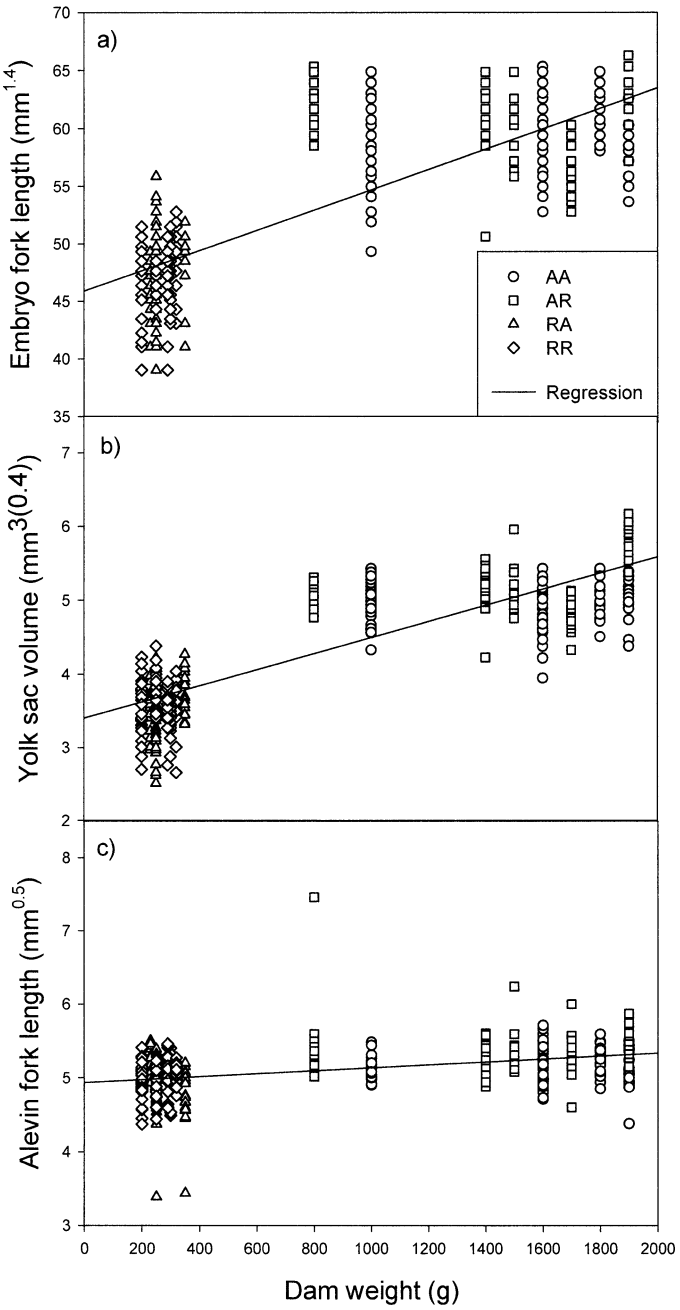

FIG. 1. Association of dam weight (DW;g) with (a) embryonic length (EL; $mm^{1.4}$) (b) yolk sac (YSV; $mm^{3(0.4)}$), and (c) alevin length (AL; $mm^{0.5}$) in posteclosion brook charr. Specific datapoints are given by cross type; circles, pure anadromous (AA), squares, anadromous female × resident male (AR); triangles, resident female × anadromous male; diamonds; pure resident (RR). Regression functions were as follows: (a) EL on DW, $y_{EL} = 0.00882 \times DW$ (± 0.000287) + 45.2(± 0.318); $r^2 = 0.673$. (b) YSV on DW, $y_{YSV} = 0.00109 \times DW$ (± 0.0000303) + 3.40 (± 0.0335); $r^2 = 0.739$. (c) AL on DW, $y_{AL} = 0.000199 \times DW$ (± 0.000210) + 4.94(± 0.0232); $r^2 = 0.165$.

TABLE 2. Direct and maternal genetic (co)variance for embryonic length (EL), yolk sac volume (YSV), and alevin length (AL) in juvenile brook charr. Direct genetic (animal) and maternal genetic estimates of narrow-sense heritability (h_a^2) are given on the diagonal for each trait, with estimates of direct genetic effects given in the upper left at the intersection of Direct and maternal genetic effects (h_a^2) in the lower right (the intersection of Maternal). Maternal-direct genetic correlation ($r_{m,a}$) is given by trait in the upper right. Additive genetic variances and covariances are given immediately below each estimate of h_a^2 and $r_{m,a}$.

Trait	σ_R^2	Direct	Maternal
EL			
Direct	6.67	0.000 ± 0.000 0.000	0.045 ± 0.068 9.96×10^{-6}
Maternal			0.610 ± 0.052 10.405
YSV			
Direct	0.0821	0.000 ± 0.000 0.000	0.041 ± 0.068 2.70×10^{-8}
Maternal			0.573 ± 0.057 0.110
AL			
Direct	0.0573	0.139 ± 0.110 0.00998	−0.379 ± 0.127 −0.00435
Maternal			0.184 ± 0.044 0.0132

TABLE 3. Sire-dam model estimates of quantitative genetic variation for embryonic length (EL), yolk sac volume (YSV), and alevin length (AL) in juvenile brook charr. Narrow-sense heritabilities (h_a^2) are given on the diagonals by trait, with sire-based estimates in the upper left (at the intersection of Sire) and dam estimates in the lower right (at the intersection of Dam). Genetic covariances between sire- and dam-based estimates of breeding values (r_a) are given to the upper right of the diagonal for each trait. Additive genetic variances and covariances are given immediately below h_a^2 and r_a estimates.

Trait	σ_R^2	Sire	Dam
EL			
Sire	6.67	0.000 ± 0.000 3.00×10^{-9}	0.000 ± 0.069 -1.10×10^{-8}
Dam			0.610 ± 0.058 10.405
YSV			
Sire	0.0821	0.000 ± 0.000 0.000	−0.004 ± 0.108 −0.001
Dam			0.573 ± 0.056 0.110
AL			
Sire	0.0623	0.033 ± 0.047 0.00249	0.029 ± 0.043 0.000154
Dam			0.149 ± 0.051 0.0113

between paternal and maternal genetic value for any of the traits (Table 3). However, an additional post-hoc analysis of genetic control of EL and YSV using a maternal-only model (since only maternal relatedness appeared to have any significant genetic effect on the traits at this stage) indicated high additive genetic correlation between these two traits ($r_a = 0.822 \pm 0.039$). Estimates of maternal genetic variance for this latter analysis were similar to those from the previous maternal-animal and sire-dam REML analyses ($h^2_{a,EL} = 0.583 \pm 0.059$; $h^2_{a,YSV} = 0.588 \pm 0.061$; $L = 209$ at 60 iterations).

Modeling of genetic variance using regression of offspring on adult parental phenotype was similar to above trends in genetic variation in that estimates of maternal variance were higher for the preeclosion traits than for AL (Table 4). However, estimates of maternal variance using regression were greatly in excess of acceptable values for the two embryonic characters ($h_a^2 \gg 1.0$), although maternal heritability for AL was low ($h_a^2 < 0.10$) whether weight or length was used as the independent variable (Table 4). Progeny AL was negatively associated with sire weight, producing a moderately negative estimate of sire heritability at this period (Table 4).

Multivariate Modeling of Fecundity, Embryo Size, and Dam Weight

Model fit was high for all four multivariate regression models ($r^2 > 0.85$; Figs. 2, 3). Mathematical models for FEC and FERT based on mean EL were roughly similar in terms of the multivariate model and the landscape of the three-dimensional relative fitness diagrams although there was still considerable scatter evident for mean EL-FEC/FERT-dam weight associations with datapoints at intermediate EL being higher than the estimated curve (Fig. 2A,B; see also Appendix 2). Peak height at intermediate EL may thus have been underestimated, although it was difficult to be certain given the relatively small number of datapoints available and the model structure. The relationship of dam weight to fecundity was simple for both FEC and FERT with mean EL as the covariate. However, quadratic and cubic structure for EL (positive quadratic and negative cubic correlation of mean full-sib family EL with fecundity) was detected for both fecundity measures (Fig. 2A,B). Both graphs using mean EL indicated superior fecundity for all dam sizes at an intermediate embryo size, although maxima may also have occurred at smaller than the maximum (Fig. 2A). Maximum average fecundity occurred at similar intermediate EL for both FEC and FERT (approximately 17.4 mm and 17.1 mm, respectively). However, associations between mean AL by family, dam weight, and fecundity were not clearly supportive of maximal fecundity at intermediate size (Fig. 3A,B). For FEC modeled using AL, there was a minor quadratic component for dam weight resulting in a limited downward curvature from an FEC of 5.4 to 4.9 at an embryo size of 30 mm. Only the quadratic term for AL was significant in this regression (Fig. 3A). There was no evidence of a distinct optimum for embryo number by dam weight and with AL as a covariate for FEC (Fig. 3A). The curvature of the multivariate fitness model was greater for FERT than for FEC with AL as a covariate (Fig. 3B). In this model, significant effects of both quadratic and cubic expressions of mean AL on the number of fertilized eggs were detected, although in this case the association of quadratic mean AL with FERT was negative whereas that with the cubic term was positive. FERT was low at small (<22 mm) mean alevin size by family. There was some curvature in the AL-FERT relationship at mean embryo sizes greater than 24 mm with FERT declining slightly to approximately 5.9 at 26 mm and increasing again to 6.4 at 30 mm,

TABLE 4. Sire- and dam-based estimates (h^2_{sire}, h^2_{dam}) of narrow-sense heritability using regression of offspring phenotype on parental (maternal and paternal) adult phenotype (PROC REG; SAS 1998). PT (parental trait) refers to the independent variable on which the embryonic trait is being regressed: log parental weight (log WT, log(g)) or fork length (log length from the tip of the rostrum to the fork of the caudal fin; log FL, log(mm)). The significance (P_{sire}, P_{dam}) of and variance associated with the regression are indicated by parent (*$P < 0.05$; **$P < 0.01$; ***$P < 0.001$; ****$P < 0.0001$). Correlation coefficients (r^2) and regression coefficients (β_{reg}) with associated standard errors are given for each parent-progeny trait combination.

Trait	PT	P_{sire}	P_{dam}	β_{sire}	β_{dam}	r_{sire}	r_{dam}	h^2_{sire}	h^2_{dam}
EL	log FL	0.3180	<0.0001***	−0.0467 ± 0.0467	0.905 ± 0.0240	0.002	0.773	−0.0934	1.81
	log WT	0.1468	<0.0001****	0.0678 ± 0.0466	0.820 ± 0.0267	0.0046	0.673	0.136	1.64
YSV	log FL	0.8659	<0.0001****	−0.00579 ± 0.0343	0.908 ± 0.0383	0.466	0.780	−0.0116	1.82
	log WT	0.2133	<0.0001****	0.0425 ± 0.0341	0.813 ± 0.0371	0.468	0.739	0.0851	1.63
AL	log FL	0.0002***	0.0874†	−0.148 ± 0.0400	0.0260 ± 0.0751	0.291	0.306	−0.296	0.0520
	log WT	0.0015**	0.5388	−0.127 ± 0.0391	0.0362 ± 0.0589	0.286	0.271	−0.254	0.0724

FIG. 2. Multifactorial association of (A) total maternal fecundity (number of eggs produced; $FEC^{0.2}$) and (B) effective fecundity (number of eggs fertilized; $FERT^{0.2}$) with dam weight (DW;g) and family mean embryonic length (EL_μ). Independent terms in the model (regressive parameters) were not transformed prior to analysis. Relevant terms for the regressive model were identified by stepwise backward regression (PROC REG; SAS 1998) Parameters for the final models were: (A) $y_{FEC,EL} = 438 + (0.0004 \times DW) - (79.0 \times EL_\mu) + (4.75 \times EL_\mu^2) - (0.095 \times EL_\mu^3)$, $r^2 = 0.957$, $n_{obs} = 23$; (B) $y_{FERT,\,EL} = -12.1 + (0.0006 \times DW) + (0.15 \times EL_\mu^2 - (0.006 \times EL_\mu^3)$, $r^2 = 0.896$, $n_{obs} = 23$. The term n_{obs} refers to the number of families used to generate the regression equations. Individual filled circles indicate specific full-sib family values used in model calculation.

but curvature in the intervening space was relatively shallow and may not have been significantly different from the local maximum achieved at 24 mm.

Discussion

Maternal Genetic Effects on Juvenile Morphology

In mammals, maternal genetic and environmental variance for embryonic phenotype results from a series of uterine, postpartum weaning, developmental, and cytoplasmic effects (Atchley and Zhu 1997; Lynch and Walsh 1998; Mousseau and Fox 1998; Van Vleck et al. 2003). Estimates of maternal genetic variation for size and growth traits tend correspondingly to be moderate-high (Chen et al. 2002; Splan et al. 2002; Van Vleck et al. 2003). We detected high additive maternal genetic control of embryonic size and yolk sac volume ($h_a^2 > 0.5$). Maternal genetic effects on body length at the alevin stage (following complete yolk sac resorption and the commencement of external feeding) were considerably lower than for either of the embryonic characters ($h_a^2 < 0.2$). A low but significant expression of genetic effects of animals on their own phenotype was also detected during the alevin period ($h_a^2 > 0.1$). Although declines in maternal effects (environmental and genetic) during ontogeny are very likely ubiquitous (Fox 1994; Montalvo and Shaw 1994; Bernardo 1996b; Fox et al. 1999; Heath et al. 1999; Lynch and Walsh 1998), our study suggests that the resorption of the yolk sac in particular is a major contrast point for the replacement of dam genetic contributions by those of individual progeny in the ontogeny of genetic effects on offspring size. However, we note that the traits used in this analysis constitute a relatively small subset of total phenotype even for size-related characters; furthermore, post-hoc REML analyses using a dam model also indicated relatively high maternal genetic correlation between EL and YSV, suggesting similarity in their genetic control in conjunction with high independent maternal genetic effects on each trait alone. Thus, our results may not be completely representative of ontogenetic changes in genetic control in toto during juvenile development; embryo length and yolk sac volume may have been part of the same modular suite (see Wagner 1996). It is likely that patterns of genetic control for other, possibly more independent morphological characters (i.e., the head and/or fins), would be different than that for general size traits. However, similar general ontogenetic trends have been detected for overall size in other salmonids (i.e., Heath et al. 1999) and thus it also appears likely that similar shifting patterns of genetic variance for size traits should occur relatively frequently within this group. High maternal genetic control for early traits likely reflects their greater energetic investment during this period. Declining maternal genetic variance would be primarily mediated through the gradual disappearance of the yolk sac, since this is effectively the only medium for the expression of maternal genetic variance in salmonid fish.

Aside from energetic considerations, sex- and age-based changes in genetic control might be necessary for the adaptedness of early ecology and reproduction in salmonid fish. In this case, the predominantly maternal genetic control of offspring size could be required to ensure appropriate local adaptation of egg size to the physical and chemical characteristics of their natal spawning habitats, to which they return with high fidelity (see Quinn et al. 1995; Einum et al. 2002; Hendry and Day 2003). This might be an underlying genetic rationale for dam-environmental correlation or phenotype/

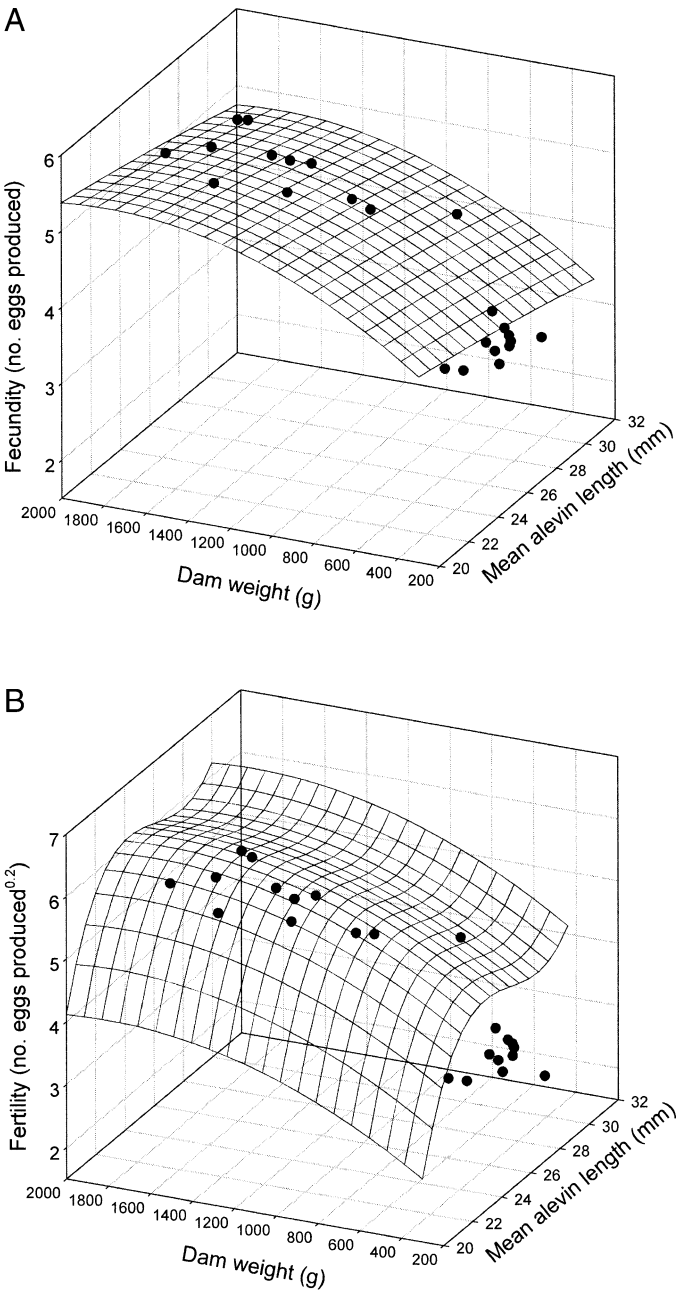

FIG. 3. Multiple regression of associations of dam weight (DW) and mean alevin length (AL_μ) by full-sib family with (A) total maternal fecundity (number of eggs produced; $FEC^{0.2}$) and (B) effective fecundity (number of eggs fertilized; $FERT^{0.2}$). In both instances, the final regression model was determined via stepwise backward regression. These were: (A) $y_{FEC,\,AL} = 3.66 + (0.0024 \times DW) - (6.70 \times 10^{-7} \times DW^2) - (0.001 \times EL_\mu^2)$, $r^2 = 0.947$, $n_{obs} = 23$; (B) $y_{FERT,\,AL} = -191 + (0.0026 \times DW) - (7.2 \times 10^{-7} \times DW^2) + (22.6 \times EL_\mu) - (0.870 \times EL_\mu^2) + (0.111 \times EL_\mu^3)$, $r^2 = 0.940$, $n_{obs} = 22$. The term n_{obs} refers to the number of families used in the regression analysis. Filled circles indicate full-sib family values used for curve construction.

habitat matching (Hendry et al. 2001) where this affects maternal reproductive success. In a similar manner, the general lack of paternal genetic effects at this stage might help avoid the breakup of local adaptation since male straying is relatively common compared to females and since male assortment to specific spawning habitat seems less likely. Additionally, large egg/embryo size at all points in development does not appear to be integrally advantageous due to trade-offs in oxygen absorbance during prehatch development and/or contrasting fitness functions before and after hatching (Hendry et al. 2001; Einum et al. 2002; Hendry and Day 2003). Coincident changes in genetic covariance between age categories in the brook charr thus may not be purely at random if fitness structure in the alevin environment is composed of quite different elements compared to that of the redd (embryonic) environment (i.e., predation avoidance and territorial selection compared to oxygen uptake; Grant 1990; Reznick 1991; Abrams and Rowe 1996; Hale 1999). Individual genetic control of postresorption size might be more appropriate since the majority of indirect maternal genetic variance has been exhausted, causing ontogenetic shifts in the value of different sources of genetic variance (maternal vs. animal) according to the nature of potential selective pressure.

Regression-Based Heritability Analysis

Although the trend in the relative magnitude of sire and dam effects using linear regression-based estimation was similar to the REML results, maternal heritability for the two embryonic characters was higher than the acceptable range of values for genetic variance ratio parameters ($h^2_{dams} > 1.0$). There appeared to be no difference between regressions using parental weight or length, indicating that neither parental trait was a very acceptable proxy for analyses of this form. Although these estimates might be broadly indicative of relative maternal and paternal genetic contributions, regression on dam phenotype in particular likely incorporated considerable bias from maternal environmental variance in our dataset despite normalization of progeny and parental phenotype. Regression-based heritability estimates of sire genetic variance for the embryonic traits were low ($h^2_a < 0.2$), again suggesting relatively minor sire effects at the embryonic level. Heritability for AL was moderately negative for sires, which has been observed for embryonic size in chinook salmon (*Oncorhynchus tshawytscha*; Heath et al. 1999). Negative heritability may occur from the sum effect of random deviations below the limit of zero variance (particularly likely at low h^2_a; Searle et al. 1992; Lynch and Walsh 1998), although the lower limit of this estimable range of heritability values for a paternal half-sib approach based on random error given our sample size was $-4/(n - 1) = -8.71 \times 10^{-3}$ (Lynch and Walsh 1998).

Maternal-Animal Genetic Covariance

Another salient finding of this study was the detection of moderate negative genetic correlation ($r_{m,a} < -0.35$) between animal and maternal genetic effects on AL. Animal-maternal genetic covariance may be fairly common in agricultural species (Koerhuis and Thompson 1997; Martínez et al. 1999; Boujenane and Kansari 2002; Chen et al. 2002; Splan et al. 2002; Maniatis and Pollott 2003; Van Vleck et al. 2003) but has been variably ascribed to variance among maternal and progeny environment (negative maternal-environmental correlation) or to limitations of data structure (Gerstmayer 1992; Meyer 1992; Martínez et al. 1999; Maniatis and Pollott 2003). However, even explorations of data structure and environmental covariance fail to eliminate negative animal-maternal covariance (Martínez et al. 1999; Maniatis and Pollott 2003). Negative genetic correlation may be acting as an evolutionary trade-off for investment relative to adult size, with stabilizing selection resulting from negative genetic covariance partially optimizing offspring versus individual survival (see Trivers 1974; Bernardo 1996b; Einum and Fleming 2000a,b). If this is the case, such trade-offs might be more prevalent in iteroparously reproducing species (in which individuals reproduce more than once; i.e., charrs [*Salvelinus* spp.]) where fitness costs of present reproductive output could be balanced against that likely to occur later in life history (see Merilä and Sheldon 2000; Crespi and Teo 2002; for a thorough description of salmonid life cycles see also Pennell and Barton 1996). Egg size optima do occur in *S. fontinalis*, an iteroparous breeder (Hutchings 1991). Conversely, semelparous salmonid fish (single reproduction followed by mortality; i.e., trouts and salmons, *Oncorhynchus* and *Salmo* spp.) appear to favor the production of larger eggs, possibly for the maximization of net reproductive output at their single spawning point or for selective advantages for larger eggs in certain local environments (see Quinn et al. 1995; Einum and Fleming 2000a,b; Crespi and Teo 2002).

If negative genetic correlation between the genetic value of dams and that of their progeny represents fitness trade-offs via intergenomic antagonism, stabilizing selection should be apparent (Wolf et al. 1998; Wolf 2000; see also Einum and Fleming 2000a,b, 2002) and results suggestive of this have been observed (Hutchings 1991; Réale et al. 2003). Fecundity/fertility peaks at intermediate embryonic progeny sizes in our surrogate fitness surface models suggest stabilizing selection on embryo size in this population, although the peak height in these graphs may have been partially underestimated. However, at the alevin stage, higher fertility appeared to be associated with higher mean alevin length despite negative genetic correlation, suggesting that stabilizing selection (if present) for juvenile length occurred during the previous stage. Differences in fitness functions based on offspring size during pre- and post-hatch development (see Grant 1990; Reznick 1991; Abrams and Rowe 1996; Oyanguren et al. 1996; Hale 1999; Einum and Fleming 2000a,b, 2002; Einum et al. 2002) might provoke dam strategies to maximize the size of progeny at independence while reducing overall costs of increasing embryo size (Lloyd 1987; see also Hutchings 1991). In this case, a potential evolutionary response to changes in size-related fitness could use alterations in developmental trajectories so that individuals of an initially intermediate size (with proportionately less investment by dams) would achieve larger size by the alevin stage (with proportionately higher fitness benefits). Where trait value is initially limited for energetic or ecological reasons but is advantageous later in development (such as size), negative correlation should occur between them (Glazier 2002). In our specific case, genetic correlation between early (embryonic)

and late (alevin) size should deviate considerably from unity. We tested this using a post-hoc analysis of correlation between BLUP dam EBVs (see Fishback et al. 2002) for AL and EL output from VCE. AL-EL correlation from maternal EBV was essentially nonexistent ($F_{1,34} = 0.00$, $p = 0.9806$, adjusted $r^2 = -0.0303$; PROC REG, SAS 1998), indicating genetic independence between the two traits. This is supported by work in brown trout (*Salmo trutta*) that indicates similar lack of correlation between early and late embryonic weight within dam lines (Vandeputte et al. 2002). This seems indicative of maternal limitations on juvenile phenotype, although we cannot completely discount an ecological base for such changes in genetic variance.

Maternal variation may play a critical role in several components of fitness and evolution, including lags to selection response and maladaptation (Cheverud and Moore 1994; Mousseau and Fox 1998), at a point that may constitute a critical node for the fitness of individuals in salmonid species (see Elliott 1986; Reznick 1991; Oyanguren et al. 1996; Hale 1999; Einum and Fleming 2002; Einum et al. 2002). Early maternal genetic contributions to fitness via life history and offspring size may be elusive because of the steady replacement of maternal effects by animal-based genetic variance during development. Changes in the relevance of specific genetic sources of variance and contrasts between maternally and individually expressed genetic variance for size traits may require a greater recognition of the importance of ontogeny in evolution and specifically suggests the need for a more dichotomous perspective on early adaptive fitness in salmonids.

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APPENDIX 1

Embryonic and alevin families of brook charr bred from anadromous (A) and resident (R) parents, November 2002–January 2003. Type indicates the specific form of the cross: pure anadromous (AA), pure resident (RR), anadromous male × resident female (AR), or resident male × anadromous female (RA). Families are thus given as resident (R) (i.e., R1), hybrid (H), or anadromous (A). The second family of full-sib progeny from sire J066 did not survive early development, most likely due to egg inviability.

Sire	Dam	Family	Type
J043 (A)	J055 (A)	A1	AA
	J090 (R)	H11	AR
J049 (A)	B014 (A)	A4	AA
	J077 (R)	H7	AR
J050 (A)	J058 (A)	A6	AA
	J075 (A)	A9	AA
J052 (R)	J060 (A)	H3	RA
	J084 (R)	R2	RR
J053 (A)	B052 (A)	A5	AA
	J088 (R)	H10	AR
J054 (A)	J056 (A)	A2	AA
	J070 (R)	H9	AR
J061 (R)	J067 (A)	H5	RA
	J082 (R)	R4	RR
J065 (R)	J051 (A)	H4	RA
	J073 (R)	R6	RR
J066 (A)	J079 (R)	H8	AR
J068 (R)	J062 (A)	H2	RA
	J086 (R)	RR	RR
J069 (R)	J072 (A)	H1	RA
	J087 (R)	R1	RR
J071 (R)	J059 (A)	H6	RA
	J076 (R)	R5	RR

APPENDIX 2

Transformed fecundity (FEC; $n^{0.2}$), fertility (FERT; $n^{0.2}$) mean dam weight (μ DW; g) and mean progeny length for fish post-hatch (embryonic length, EL; mm) and postresorption of the yolk sac (alevin length, AL; mm) in 23 full-sib families of brook trout (indicated by maternal parent). Data is sorted by EL.

Dam	FEC ($n^{0.2}$)	FERT ($n^{0.2}$)	DW (g)	μ EL (mm)	μ AL (mm)
J082	3.49	3.49	200	15.2	24.1
J079	3.78	3.78	250	15.3	23.9
J077	3.30	2.76	230	15.4	27.4
J084	3.61	3.61	200	15.5	24.8
J088	3.66	3.66	250	15.6	22.4
J086	3.51	3.51	290	15.7	26.0
J073	3.38	3.38	300	15.8	26.3
J076	3.57	3.57	250	16.0	24.5
J090	3.61	3.61	350	16.0	22.5
J070	3.77	3.77	250	16.2	25.2
J087	3.93	3.88	320	16.3	25.3
B014	5.04	4.95	1000	17.4	25.5
J055	5.09	5.08	1600	18.0	24.7
J075	5.18	5.18	1900	18.0	28.5
J059	5.27	5.18	1700	18.0	25.7
J060	4.84	4.63	1500	18.6	28.5
J058	4.82	4.71	1600	18.7	28.7
J062	4.73	4.65	1400	18.8	26.8
J067	4.85	4.74	1400	18.8	28.5
B052	4.70	4.69	1000	19.0	26.8
J056	5.04	5.00	1800	19.0	27.4
J072	4.25	4.16	800	19.2	29.9
J051	4.84	4.75	1900	19.2	30.6