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Quantitative genetic and translocation experiments reveal genotype-by-environment effects on juvenile life-history traits in two populations of Chinook salmon (*Oncorhynchus tshawytscha*)

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Abstract

Understanding the genetic architecture of phenotypic plasticity is required to assess how populations might respond to heterogeneous or changing environments. Although several studies have examined population-level patterns in environmental heterogeneity and plasticity, few studies have examined individual-level variation in plasticity. Here, we use the North Carolina II breeding design and translocation experiments between two populations of Chinook salmon to detail the genetic architecture and plasticity of offspring survival and growth. We followed the survival of 50 800 offspring through the larval stage and used parentage analysis to examine survival and growth through freshwater rearing. In one population, we found that additive genetic, nonadditive genetic and maternal effects explained 25%, 34% and 55% of the variance in larvae survival, respectively. In the second population, these effects explained 0%, 24% and 61% of the variance in larvae survival. In contrast, fry survival was regulated primarily by additive genetic effects, which indicates a shift from maternal to genetic effects as development proceeds. Fry growth also showed strong additive genetic effects. Translocations between populations revealed that offspring survival and growth varied between environments, the degree of which differed among families. These results indicate genetic differences among individuals in their degree of plasticity and consequently their ability to respond to environmental variation.

Introduction

Many species occur across heterogeneous and changing environments, a situation that is increasing in frequency as anthropogenic changes alter the environmental conditions faced by natural populations (Stenseth *et al.*, 2002; Walther *et al.*, 2002). It is thought that the expression of differential phenotypes attributed to a change in the environment, or 'plasticity', should evolve when there is spatial or temporal environmental variation, because it allows individuals to cope with the varying environment (e.g. Charmantier *et al.*, 2008; see de Jong, 1995; Via *et al.*, 1995; Pigliucci, 2001). Indeed, many studies have reported covariation between the degree of environmental variation and plasticity in phenotypes across populations (e.g. Robinson *et al.*, 2009; see Via *et al.*, 1995 and Nussey *et al.*, 2007 for reviews). Furthermore, the importance of understanding variation in plasticity at the level of the individual is increasingly being recognized as it allows for a more detailed examination of the consequences of environmental change on the evolution of genetic and phenotypic variation within populations (Nussey *et al.*, 2007).

Several recent studies have reported individual-level variation in phenotypic plasticity in response to environmental variation. For example, Brommer *et al.* (2005) reported variation in female Collared Flycatcher (*Ficedula*

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albicollis) laying date in response to variation in local temperature, but individual females also showed significant variation in their ability to alter their laying date in response to temperature. Similarly, a study of female Red deer (*Cervus elaphus*) indicated that calving date varies in response to rainfall levels, but females also showed variation in their response to the rainfall (Nussey *et al.*, 2005a). Such variation in plasticity can have significant fitness consequences for individuals. In a study of great tits (*Parus major*), females that exhibited greater phenotypic plasticity in laying date in response to warming spring temperatures had higher fitness than females that were constant in their laying dates (Nussey *et al.*, 2005b). Thus, phenotypic plasticity can be quite complex, and both individual- and population-level analyses of phenotypic responses to environmental change are required to fully understand the fitness consequences of phenotypic plasticity.

Genetic variation for plasticity in fitness-related traits will determine the potential for plasticity to evolve (Kruuk *et al.*, 2008). The genetic basis of plasticity in response to environmental variation can be evaluated using reaction norms and the analysis of genotype-by-environment (G × E) effects. A reaction norm is a function that describes the change in a genotype's phenotype across environments (Via *et al.*, 1995). As the vast majority of traits are likely to be influenced by many genes, a quantitative genetics approach is necessary to detail the genetic architecture underlying these traits and to establish genetic relationships among individuals (Kruuk *et al.*, 2008). Quantitative genetic breeding designs such as the North Carolina II can be used to partition variation in quantitative traits to additive and nonadditive genetic effects as well as maternal effects (Lynch & Walsh, 1998, p. 598; Neff & Pitcher, 2005). Previous studies have successfully utilized this method in fishes to examine the genetic architecture of important fitness-related traits. For example, Pitcher & Neff (2007) conducted a North Carolina II cross on Lake Ontario Chinook salmon (*Oncorhynchus tshawytscha*) and demonstrated that upwards of 50% of the variation in larvae survival could be explained by each of additive and nonadditive genetic effects. The authors also found nonadditive genetic effects on larvae growth. Similarly, Wedekind *et al.* (2008) found that high levels of variance in whitefish (*Coregonus* sp.) embryo survival could be explained by genetic effects, but the sources of variance appeared to vary by the time of year that individuals spawned, suggesting a role for G × E effects.

The genetic architecture of traits has been shown to change throughout ontogeny, which may have implications for our understanding of trait response to selection (Wilson & Réale, 2006; Kruuk *et al.*, 2008). In particular, additive genetic and maternal effects have been shown to vary across different developmental stages. For example, in laboratory rats (*Rattus norvegicus*), maternal effects on offspring mass and tail length decline during juvenile development, whereas additive genetic effects on these two traits either remain constant or increase during the same period (Cheverud *et al.*, 1983). More recently, studies in wild populations have also shown that additive genetic effects vary with age. In mute swans (*Cygnus olor*), the contribution of additive genetic variance to timing of breeding changes with age, with strong additive genetic effects in young and old swans but not in swans of intermediate age (Charmantier *et al.*, 2006). Similar results were reported by Réale *et al.* (1999) for body mass in Bighorn sheep (*Ovis canadensis*). Thus, growing evidence indicates that the genetic architecture of traits varies with age (see Wilson & Réale, 2006 for a review).

Salmonid species represent an ideal system in which to examine variation in the genetic architecture and plasticity of important fitness-related traits, because populations face significant spatial and temporal variation in selection pressures (Taylor, 1991; Adkison, 1995). For example, recent studies have suggested that salmonid populations are the subjects of differential spatial and temporal selection imposed by pathogens (Dionne *et al.*, 2009; Evans & Neff, 2009). Dionne *et al.* (2009) demonstrated that Atlantic salmon (*Salmo salar*) juveniles face spatial and temporal variation in the levels of bacterial and Myxozoan infections. Similarly, Evans & Neff (2009) demonstrated that Chinook salmon juveniles face differing levels of bacterial infections and divergent bacterial parasite communities across populations. Nevertheless, despite widespread interest in the investigation of adaptations in populations of salmonids, relatively little is known about the genetic architecture of fitness and the extent to which genetically determined fitness-related traits show G × E effects across environmental gradients.

In this study, we employ the North Carolina II approach to investigate genetic variation underlying juvenile Chinook salmon survival and growth in two populations. We also investigate plasticity and the importance of G × E effects on these traits across environments using reciprocal translocation experiments. Our study focuses on the Quinsam River and Big Qualicum River populations that are located on Vancouver Island in British Columbia, Canada, and are separated by approximately 100 km. We first investigate how offspring survival and growth varies among family groups in each population. Furthermore, we investigate variation in the contribution of maternal and additive and nonadditive genetic effects to survival across the larval and fry developmental stages, which has not previously been accomplished in multiple wild populations. We then partition phenotypic variance in survival and growth using full-sibs that were reared either in their natal population or reciprocally translocated to the non-natal population. Individuals were reared in a common garden experiment within a hatchery environment, so survival and growth were not affected by predation or

limited by food availability. However, the families were exposed to untreated river water and thus any waterborne pathogens and natural fluctuations in water temperature or water chemistry (Amos & Thomas, 2002). We also used microsatellite loci to examine how relatedness between parents influences juvenile survival. Finally, we examine how sire or dam length relate to the growth of their offspring as one potential phenotypic correlate of performance. Given that many Pacific salmonid populations have faced unprecedented declines and even extirpation over the last 50 years (Nehlsen *et al.*, 1991; Levin & Schiewe, 2001; Fraser, 2008), knowledge of the genetic architecture of fitness throughout juvenile development and the abilities of salmonid juveniles to respond to different environments will be critical for conservation and management planning.

Materials and methods

Collection of Chinook salmon

Wild Chinook salmon were collected from the Big Qualicum River using diversion channels located at the Fisheries and Oceans Canada Big Qualicum salmon hatchery. At the Quinsam River, wild adults were collected by seine netting from natural holding ponds during the migration to the spawning grounds. Crosses were conducted on 8 October 2007 at the Big Qualicum River hatchery located near Qualicum Beach, B.C., and on 15 October 2007 at the Quinsam River hatchery located near Campbell River, B.C. Males and females were euthanized prior to gamete collection using carbon dioxide and were measured for post-orbital hypural body length. Milt was collected from males by applying pressure to the male's abdomen, and eggs were collected by dissecting the abdomen of the female and removing the eggs. Eggs and milt were stored in dry buckets or Whirl-pak bags (Nasco Plastics, Newmarket, Canada), respectively, until the crosses were conducted. We also obtained a tissue sample from the adults for later genetic analysis. The tissue samples were stored in 95% ethanol.

Crosses and translocation

All possible crosses were conducted between eight males and eight females in each population, resulting in 64 unique family groups within each population. Each cross was replicated four times ($N = 256$ family groups), and each replicate consisted of 100 eggs. Eggs were stored under cool conditions until fertilization could take place (< 2 h) and were fertilized in the order in which they were collected. Families were stored in individual egg tubes (Dynamic Aqua-supply Ltd, Surrey, Canada) and were randomly allocated to a location within a Heath incubation tray system located at each hatchery. Fertilized eggs were exposed to natural river water for the duration of their development.

To examine whether juvenile performance varied between rearing sites, we transplanted half of the replicates from each family group (two replicates per family group; $N = 128$ families) to the reciprocal hatchery (i.e. Quinsam to Big Qualicum and Big Qualicum to Quinsam). Early on in salmon egg development, mortality can be high if the developing eggs are moved or exposed to light (Stead & Laird, 2002). Salmon egg development rate is dependent on water temperature, and the development can be tracked by measuring accumulated thermal units (ATUs). At approximately 250–280 ATUs (based on °C), Chinook salmon eggs become 'eyed' and are no longer sensitive to light exposure and movement (Groves *et al.*, 2008). Prior to becoming eyed, the mean water temperature that developing embryos were exposed to was 8.6 °C for the Big Qualicum River and 10.8 °C water at the Quinsam River (the river water temperature). Embryos became eyed in both populations on 10 November 2007, following 33 days of development in the Big Qualicum population and 26 days of development in the Quinsam population. Thus, the translocation experiment took place on 19 November 2007, once the family groups in each population had been exposed to at least 280 ATUs. Family groups that remained in the natal hatchery were also handled and then relocated so as to replicate the movement experienced by the translocation groups. The location of each egg tube within the heath stack system was noted before and after translocation as it may influence the survival of juveniles (e.g. Pitcher & Neff, 2007).

Larvae survival

Family groups were monitored weekly for juvenile survival through egg and larvae development. Survival to the end of the larval stage within each family group was represented by the proportion of survivors out of 100. For comparisons between control and translocation family groups, survival from the time of translocation to the end of the larvae stage was used. This survival estimate was calculated as # survivors at the end of the larval stage/# survivors at translocation.

Fry survival

We continued to rear all family groups during the fry stage; however, it was necessary to pool all 64 family groups within a replicate because of the logistical constraints of keeping each family group separate within hatchery rearing tanks. Individual replicates from each family were reared separately, and all juveniles within a replicate were reared in Capilano troughs until the end of the fry stage. Fry were exposed to natural, untreated river water and fed EWOS feed (EWOS Canada Ltd, Surrey, B.C., Canada) *ad libitum*. Chinook salmon from both populations are ocean-type meaning that fry

typically begin migration to the ocean in the spring (typically mid April–May). Thus, on 2 April 2008, 134 days after translocation, we terminated the experiment and randomly sampled 200 fry from each pooled replicate. Sampled fry were anaesthetized using MS-222 (tricaine methanesulphonate), measured for body length (post-orbital hypural), and a tissue sample was taken from the adipose fin for parentage analysis. The tissue samples were stored in 95% ethanol.

Parentage analysis

DNA was extracted from the tissue of parents and offspring using a DNA Wizard Extraction kit (Promega Corp., Madison, WI, USA). To obtain an estimate of fry survival for each family group, we used microsatellite parentage analysis. Chinook salmon parents and fry were genotyped at eight microsatellite loci (Table 1). Loci were amplified using polymerase chain reaction (PCR) following the protocol outlined in Heath *et al.* (2006). Each forward primer was tagged with a fluorescent dye that enabled the PCR products to be compared against size standards using the Li-Cor 4300 DNA analyser (Li-Cor Biosciences, Licoln, NE, USA). For each locus, we calculated the number of alleles and the expected (H_E) and observed (H_O) heterozygosity using Genepop v. 3.4 (Raymond & Rousset, 1995). We assigned parentage using a maximum likelihood procedure implemented in the program CERVUS v. 3.0 (Kalinowski *et al.*, 2007). Only offspring that were successfully genotyped at five or more loci were included in the analysis.

Survival through the fry stage was extrapolated for each family group by estimating the number of offspring assigned to a parental pair at 80% or higher confidence. We were unable to enumerate all of the fry in the tanks at the end of the experiment, so we calculated and index of 'relative fry survival' for each family group by dividing the proportion of genotyped offspring identified to a family group by the proportion of offspring from that family group that were introduced into the tank. Thus, a value of < 1 would indicate a family with lower than average survival, and a value of > 1 would indicate a family with higher than average survival. The assigned fry were also used to determine a mean body length for each family. We calculated a standardized estimate of length within each replicate to account for replicate-specific effects on growth (e.g. tank effects). This standardized estimate was calculated for each family group as (mean fry length within a family group − the mean length within that replicate)/standard deviation of the replicate mean length (Lipsey & Wilson, 2001). A value > 0 would indicate above average body size relative to individuals within the replicate, whereas a value < 0 would indicate below average size.

Relatedness

We used the microsatellite data to estimate the relatedness (RI) between parents within each population using the method described by Ritland (1996). Relatedness was estimated in GeneAlEx v. 6.2 (Peakall & Smouse, 2006). A negative relationship between parental relatedness and

Table 1 Microsatellite loci used to assess parentage in Chinook salmon (*Oncorhynchus tshawytscha*) fry. For each microsatellite, forward and reverse primer sequences, PCR annealing temperatures (T_m), observed (H_O) and expected heterozygosity (H_E) and number of alleles found in the 16 parents from the Big Qualicum and Quinsam populations are indicated.

			Big Qualicum			Quinsam		
Locus	Primer sequence*	T_m (°C)	H_O	H_E	A	H_O	H_E	A
Ots107	F-ACAGACCAGACCTCAACA R-ATAGAGACCTGAATCGGTA	58	16	15.0	14	16	14.6	11
Ots104	F-GCACTGTATCCACCAGTA R-GTAGGAGTTTCATTTGAATC	64–49†	15	14.9	16	13	13.6	12
OtsG311	F-GCGGTGCTCAAAGTGATCTCAGTCA R-TCCATCCCTCCCCCATCCATTGT	64–49†	15	14.0	18	16	15.2	16
OtsG249	F-TCTCAGAGGGTAAAATCTCAGTAAG R-GTACAACCCCTCTCACCTACCC	64–49†	16	15.1	15	15	15.3	18
Omy325	F-TGTGAGACTGTCAGATTTTGC R-CGGAGTCCGTATCCTTCCC	56	13	13.2	8	10	11.4	6
OtsG83B	F-TAGCCCTGCACTAAAATACAGTTC R-CATTAATCTAGGCTTGTCAGCAGT	60	15	15.1	15	16	14.9	13
OtsG68	F-CATGTACGTGGCGAAGCCCTC R-CATGTCGGCTGCTCAATGTA	55	16	15.3	16	13	14.1	14
OtsG432	F-TGAAAAGTAGGGGAAACACATACG R-TAAAGCCCATTGAATTGAATAGAA	64–49†	13	11.9	12	8	10.9	10

*Primer sequences published in Nelson & Beacham (1999), Williamson *et al.* (2002) and O'Connell *et al.* (1997).
†Touchdown PCR: annealing temperature set at 64 °C for initial cycle, then dropped by 1 °C in each subsequent cycle until a final annealing temperature of 49 °C was reached, which was continued through the remaining cycles.

juvenile survival would indicate inbreeding depression, whereas a positive relationship would indicate outbreeding depression. Furthermore, if the populations show adaptive divergence, we predicted that translocation families that performed relatively well (i.e. higher survival) would be more related to individuals from the translocation site than translocation families than performed less well.

Statistical analysis

We first used a two-way ANOVA to assess the potential that tray location before and after the eyed-egg stage (i.e. when we moved the eggs) influenced larvae survival. For control replicates, a two-way restricted maximum likelihood (REML)-based ANOVA was then used to partition variance in larvae survival, relative fry survival and fry length to Sire and Dam identity and the interaction between Dam and Sire (Dam × Sire). For the survival analyses, we used mean survival within a family replicate. However, for fry length, we used individual estimates of length so as to account for within-family variation in growth rate (see Puurtinen *et al.*, 2009). Following Lynch & Walsh (1998, p. 598), the contribution of additive genetic effects to survival and length was estimated as four times the Sire contribution to variance. Nonadditive genetic effects were estimated as four times the Dam × Sire contribution to variance. Finally, maternal effects were calculated as the difference between the variances associated with Dams and Sires.

To examine variation in larvae survival following translocation, we used a REML-based ANOVA to partition variance in larvae survival, relative fry survival and standardized fry length within each treatment to Dam, Sire, Dam × Sire, Dam × Treatment, Sire × Treatment and Dam × Sire × Treatment effects. Treatment had two levels coded as ''natal'' (for families that remained exposed to their natal river water) and ''translocation'' (for families that were exposed to non-natal river water). We were unable to compare overall fry survival and fry growth among natal and translocation treatments, as each of these measures was standardized; hence, treatment on its own was included only in the analysis of larvae survival. Dam and Sire were treated as random factors, and Treatment was treated as a fixed factor within the models.

We examined the relationships between parental male and female length and mean invenile length, and relatedness between parents and offspring survival using linear regression with more than one value of Y per value of X (Sokal & Rohlf, 1995, p. 476). We examined the relationship between parental lengths and mean juvenile length and survival of juveniles in control and translocation replicates in separate regression analyses. Finally, we also used linear regression to examine the relationship between the mean relatedness between a parent and the parents in the translocation population and the survival of the parent's translocated offspring. All statistical analyses were run in SPSS v.17. We used a significance value of $\alpha = 0.05$, and all means are reported ± 1 SD.

Results

Quantitative genetics

Larvae survival

The initial two-way ANOVA revealed that tray location did not have a significant effect on larvae survival in the Quinsam population (before relocation: $F_{13,242} = 1.02$, $P = 0.44$; after relocation: $F_{12,243} = 1.02$, $P = 0.44$) or in the Big Qualicum population (before relocation: $F_{13,242} = 0.73$, $P = 0.72$; after relocation: $F_{12,238} = 0.81$, $P = 0.82$). Larvae survival averaged 73 ± 27% (range = 0–100%) for the Big Qualicum natal family groups. In the Quinsam River natal family groups, larvae survival averaged 94 ± 7% (range = 67–100%). The two-way ANOVA for the Big Qualicum population revealed that Dam, Sire and Dam × Sire effects were significantly associated with larvae survival and explained 62%, 6% and 9% of the variation in survival, respectively (Table 2). From the variance components, we estimated that nonadditive genetic effects represented 34% of the total phenotypic variance in survival. Additive genetic effects represented 25% of the total phenotypic variance in survival, and maternal effects represented 55% of the phenotypic variance in survival. The total phenotypic variance explained by these effects exceeded 100%, which could indicate epistatic interactions (Lynch & Walsh, 1998, p. 601; also see Pitcher & Neff, 2007; Wedekind *et al.*, 2008), or that heritability is overestimated as a result of using family mean survival (see Puurtinen *et al.*, 2009). In the Quinsam population, only Dam effects were significantly associated with larvae survival, although Dam × Sire effects were near-significant (Table 2). Maternal effects represented 61% of the phenotypic variance in larvae survival, and although only near-significant, nonadditive genetic effects explained 24% of the phenotypic variance in larvae survival.

Relative fry survival

A total of 337 natal fry from the Big Qualicum River and 344 natal fry from the Quinsam River were identified to a parent pair. The relative fry survival in Big Qualicum family groups ranged between 0 and 9.74. For the Quinsam River fry, relative survival ranged between 0 and 3.44. Female 8 was removed from the two-way ANOVA of relative fry survival in the Big Qualicum population because of low juvenile survival during the previous developmental stage (mean survival = 22%). For the Big Qualicum population, the two-way ANOVA revealed that Dam and Sire effects explained 17% and 13% of the variation in relative fry survival, respectively, whereas the Dam × Sire interaction was not significant (Table 2). From these variance components, it was estimated that additive genetic effects accounted for

Table 2 Summary of two-way ANOVA results for larvae survival, relative fry survival and fry length in Big Qualicum and Quinsam river Chinook salmon (*Oncorhynchus tshawytscha*). The source of variation, degrees of freedom (DF), sum of squares (SS), F statistic, *P*-value, variance component (σ^2) and the per cent of total variance (% total var) explained by each source are indicated. In addition, on the right side of the table, the contribution of maternal effects, and additive and nonadditive genetic effects to phenotypic variance (% phenotypic var) is indicated. Significant values ($P < 0.05$) are bolded.

	DF	SS	*F*	*P*	σ^2 (% total var)	% phenotypic var	
Larvae survival							
Big Qualicum							
Dam	7	57247.1	25.47	**< 0.001**	491.1 (62)	Maternal	55
Sire	7	7939.0	3.53	**0.004**	50.8 (6)	Additive	25
Dam × Sire	49	15732.4	1.74	**0.019**	68.4 (9)	Nonadditive	34
Residual					184.3 (23)		
Quinsam							
Dam	7	3480.6	22.21	**< 0.001**	29.7 (62)	Maternal	61
Sire	7	0.0	0.00	1.000	0.0 (0)	Additive	0
Dam × Sire	49	1097.2	1.42	0.092	28.9 (6)	Nonadditive	24
Residual					15.7 (32)		
Relative fry survival							
Big Qualicum							
Dam	6	7.5	4.18	**0.002**	7.2×10^{-2} (17)	Maternal	4
Sire	7	5.6	2.67	**0.022**	5.5×10^{-2} (13)	Additive	52
Dam × Sire	42	0.0	0.00	1.000	0.0 (0)	Nonadditive	0
Residual					29.9×10^{-2} (70)		
Quinsam							
Dam	7	0.6	0.23	0.975	1.1×10^{-2} (2)	Maternal	0
Sire	7	21.7	8.75	**< 0.001**	16.6×10^{-2} (31)	Additive	125
Dam × Sire	49	0.2	0.01	1.000	0.2×10^{-2} (0)	Nonadditive	2
Residual					35.5×10^{-2} (67)		
Fry length							
Big Qualicum							
Dam	6	86.4	18.17	**< 0.001**	47.4×10^{-2} (33)	Maternal	23
Sire	7	26.8	4.83	**< 0.001**	14.4×10^{-2} (10)	Additive	39
Dam × Sire	42	11.2	0.33	1.000	4.5×10^{-2} (3)	Nonadditive	13
Residual					79.2×10^{-2} (54)		
Quinsam							
Dam	7	572.8	4.19	**0.001**	2.1 (9)	Maternal	1
Sire	7	576.6	4.22	**0.001**	2.0 (8)	Additive	33
Dam × Sire	49	8.34	0.01	1.000	3.2×10^{-2} (0)	Nonadditive	0
Residual					19.5 (83)		

52% of the total phenotypic variance in relative fry survival, and maternal effects accounted for an additional 4% of the phenotypic variance. In the Quinsam population, we also found that Sire identity was significantly associated with relative fry survival, but Dam identity and the Dam × Sire interaction were not significant (Table 2). Sire effects explained 31% of the variance in relative fry survival, and the additive genetic effects on relative survival represented 125% of the total phenotypic variance. Again, the total phenotypic variance explained by genetic effects exceeded 100%, suggesting that epistatic interactions may be an important aspect of fry survival.

Fry length

Big Qualicum fry averaged 33.2 ± 0.9 mm (range = 31.0–36.0 mm) in length. Quinsam River fry averaged 58.2 ± 3.3 mm (range = 50.5–66.0 mm) in length. The difference in mean lengths between locations was likely the result of hatching and rearing time differences within each population (after hatch rearing time: Big Qualicum = 54 days; Quinsam = 72 days). In the Big Qualicum population, the two-way ANOVA revealed that Dam identity, Sire identity and Dam × Sire effects explained 33%, 10% and 3% of the variation in standardized juvenile length, respectively, although only Dam and Sire effects were significantly associated with length (Table 2). Additive genetic effects represented 39% of the total phenotypic variance in fry length, whereas maternal effects explained 23% of the variance. In the Quinsam population, we found Dam and Sire identity were significantly associated with fry length. As in the Big Qualicum population, Dam × Sire effects were not significant (Table 2). Sire and Dam effects explained 9% and 8% of the variance in fry length, respectively, and additive genetic effects represented 33% of the total

phenotypic variance in fry length, and maternal effects represented an additional 1% of the variance (Table 2).

Survival and length following translocation

Larvae survival

After translocation, survival averaged 98 ± 5% (range = 70–100%) for the Big Qualicum natal family groups and 97 ± 10% (range = 0–100%) for the Big Qualicum translocation family groups (Fig. 1a). In the Quinsam River natal family groups, survival after translocation averaged 99 ± 1% (range = 94–100%), whereas for the Quinsam River translocation family groups, survival averaged 99 ± 3% (range = 71–100%; Fig. 1b). In the Big Qualicum population, Dam identity, Dam × Sire and Sire × Treatment effects were significantly associated with variation in larvae survival (Table 3). Mean survival for the half-sib family groups of three of the males decreased when reared in the non-natal Quinsam environment, whereas offspring from the remaining five males exhibited similar survival in both environments (Fig. 1a). Thus, these results provide evidence of G × E effects and two discrete reaction norms exhibited by males in response to the translocation. Males that exhibited lower larvae survival in their translocation family groups relative to natal family groups were not

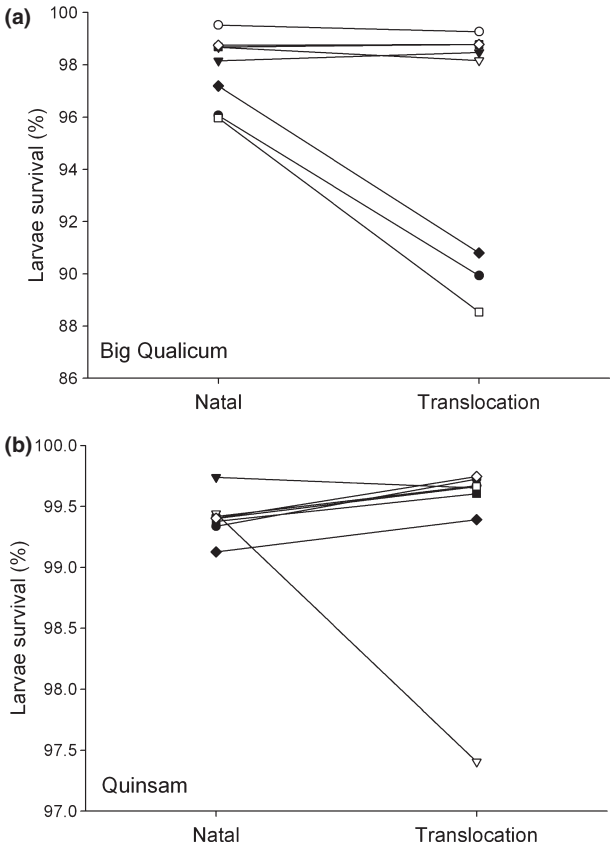

Fig. 1 Norms of reaction for larvae survival in Chinook salmon (*Oncorhynchus tshawytscha*). The norms of reaction are shown for (a) Big Qualicum larvae and (b) Quinsam larvae. Like symbols represent means from paternal half-sib families reared in a hatchery located either on their natal river or reciprocally translocated to the foreign hatchery. Phenotypic plasticity is indicated by nonhorizontal lines, and a genotype-by-environment interaction is indicated by nonparallel lines. Note that the scales of the *y*-axes differ.

Table 3 Summary of three-way ANOVA results for larvae survival, relative fry survival and fry length in Big Qualicum and Quinsam river Chinook salmon (*Oncorhynchus tshawytscha*). Families were reared in their natal population or reciprocally translocated to the non-natal population. Sources of variation, degrees of freedom (DF), sum of squares (SS), F statistic and the *P*-value are indicated. Significant values ($P < 0.05$) are bolded.

Population	Source of variation	DF	SS	*F*	*P*
Larvae survival					
Big	Dam	7	89168.1	23.04	**< 0.001**
Qualicum	Sire	7	7416.1	1.36	0.261
	Treatment	1	157.9	0.37	0.702
	Dam × Sire	49	27512.7	6.20	**< 0.001**
	Dam × Treatment	7	604.8	0.96	0.472
	Sire × Treatment	7	2219.1	3.52	**0.004**
	Dam × Sire × Treatment	48	4321.5	0.62	0.970
Quinsam	Dam	7	10497.2	16.69	**< 0.001**
	Sire	7	296.2	0.76	0.628
	Treatment	1	64.0	0.90	0.368
	Dam × Sire	49	2154.7	3.38	**< 0.001**
	Dam × Treatment	7	412.3	4.53	**0.001**
	Sire × Treatment	7	174.4	1.92	0.087
	Dam × Sire × Treatment	49	636.8	0.86	0.681
Relative fry survival					
Big	Dam	6	12.2	2.08	0.190
Qualicum	Sire	7	5.5	0.72	0.664
	Dam × Sire	42	11.1	1.19	0.287
	Dam × Treatment	6	5.6	4.21	**0.002**
	Sire × Treatment	7	7.3	4.71	**0.001**
	Dam × Sire × Treatment	42	9.3	0.62	0.958
Quinsam	Dam	7	6.3	1.12	0.448
	Sire	7	21.8	1.77	0.237
	Dam × Sire	49	16.9	0.99	0.510
	Dam × Treatment	7	5.7	2.33	**0.039**
	Sire × Treatment	7	12.4	5.10	**< 0.001**
	Dam × Sire × Treatment	49	16.9	0.89	0.676
Fry length					
Big	Dam	6	53.8	2.74	0.090
Qualicum	Sire	7	24.5	1.87	0.161
	Dam × Sire	42	44.3	2.14	**0.009**
	Dam × Treatment	6	16.1	5.48	**< 0.001**
	Sire × Treatment	7	9.4	2.73	**0.020**
	Dam × Sire × Treatment	40	19.7	1.36	0.118
Quinsam	Dam	7	50.5	4.65	**0.050**
	Sire	7	12.6	1.87	0.273
	Dam × Sire	48	21.9	0.68	0.907
	Dam × Treatment	7	12.2	2.58	**0.024**
	Sire × Treatment	7	8.1	1.72	0.126
	Dam × Sire × Treatment	46	30.9	0.89	0.661

significantly less related to individuals in the Quinsam population (RI = −0.13) than individuals with similar survival between treatments (RI = −0.05; $P = 0.27$). In the Quinsam population, we found that only Dam identity, Dam × Sire and Dam × Treatment were significantly associated with fry survival (Table 3). As predicted under the adaptive divergence hypothesis, some families exhibited higher survival in their natal hatchery. However, paternal half-sib family groups for six of the Quinsam males exhibited slightly higher mean survival in the Big Qualicum environment than in their natal environment, whereas the half-sib family groups for the other two Quinsam males exhibited lower mean survival when reared in the Big Qualicum environment (Fig. 1b). Although not significant, this pattern suggests G × E effects on larvae survival (Table 3).

Relative fry survival

The relative fry survival ranged from 0 to 5.57 for fry in Big Qualicum translocation family groups, and for Quinsam River translocation family groups, relative survival ranged between 0 and 3.22. In both populations, only Dam × Treatment and Sire × Treatment significantly explained variation in relative fry survival. These interaction effects were not simple in direction. Approximately half of the paternal half-sib family groups exhibited higher relative fry survival when translocated, whereas the other half-sib family groups exhibited lower survival (Fig. 2). Thus, G × E effects appear to play a significant role in fry survival.

Fry length

Translocated Big Qualicum fry averaged 58.5 ± 3.5 mm (range = 43.3–73.0 mm), and translocated Quinsam River fry averaged 33.1 ± 1.5 mm (range = 25.5–36.0 mm) in length. Dam × Sire, Dam × Treatment and Sire × Treatment were significantly associated with fry length for Big Qualicum fish, whereas only Dam × Treatment and Dam identity were significantly associated with juvenile length for Quinsam fish (Table 3). However, as was found for variance in fry survival, treatment affected fry length inconsistently across family groups. In both populations, half of the paternal half-sib family groups exhibited decreased growth in the translocated environment, whereas the other paternal half-sib family groups exhibited increased growth in the translocated environment (Fig. 3). These results again indicate G × E effects.

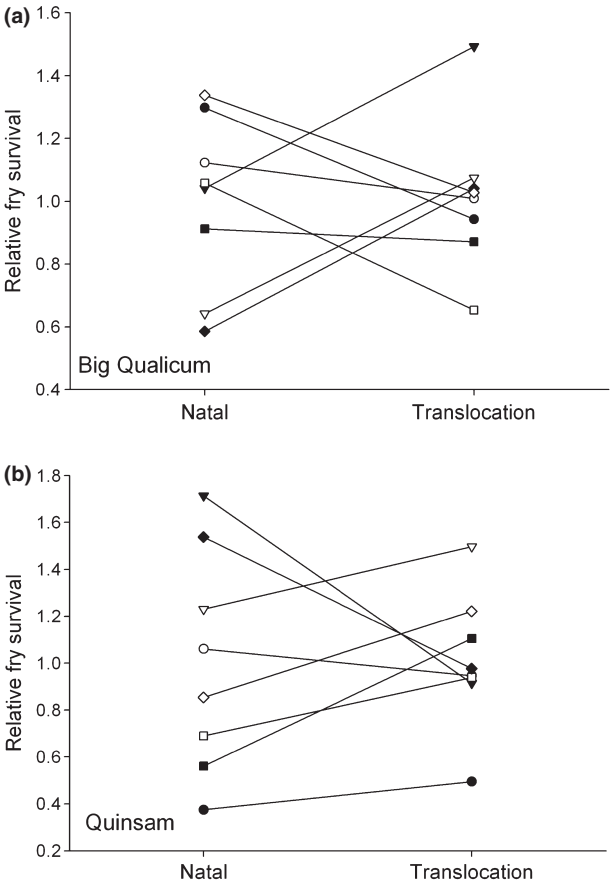

Fig. 2 Norms of reaction for relative fry survival in Chinook salmon (*Oncorhynchus tshawytscha*). The norms of reaction are shown for (a) Big Qualicum fry and (b) Quinsam fry. Like symbols represent means from paternal half-sib families reared in a hatchery located either on their natal river or reciprocally translocated to the foreign hatchery. Phenotypic plasticity is indicated by nonhorizontal lines, and a genotype-by-environment interaction is indicated by nonparallel lines. Relative fry survival was calculated from % genotyped juveniles identified to a family group/% of juveniles from the family group that were introduced into each rearing tank. A value of < 1 indicates a family with lower than average survival, and a value of greater than one indicates a family with higher than average survival. The estimates of survival are based on 681 fry from the Big Qualicum River and 688 fry from the Quinsam River. Note that the scales on the *y*-axes differ.

Relatedness and survival

Mean relatedness between parents in the Big Qualicum population was −0.013 ± 0.036 (range = −0.06–0.07). In the Quinsam population, mean relatedness was −0.01 ± 0.034 (range = −0.06–0.08). We did not find that relatedness between parents was significantly associated with larvae survival in either natal or translocation family groups in the Quinsam population (natal: $r^2 = 0.007$, $F_{1,62} = 0.86$, $P = 0.36$, translocation: $r^2 = 0.011$, $F_{1,62} = 1.46$, $P = 0.23$) or in Big Qualicum translocation family groups ($r^2 = 0.011$, $F_{1,62} = 1.35$, $P = 0.25$). However, we found a positive relationship between relatedness and larvae survival in the Big Qualicum natal family groups ($r^2 = 0.029$, $F_{1,62} = 3.82$, $P = 0.055$), albeit the relationship was not significant and captured only a small per cent of the variation in survival. Relative fry survival in natal and translocation family groups was not associated with relatedness

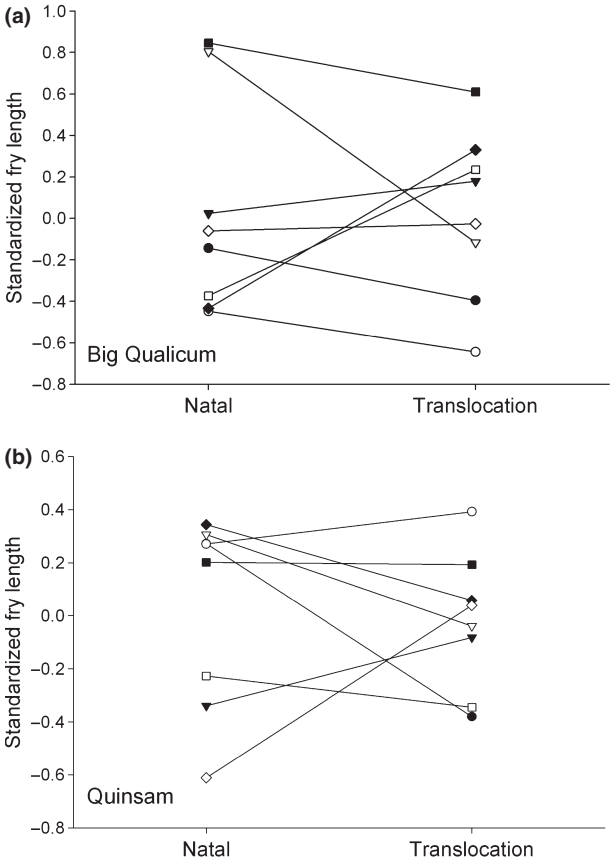

Fig. 3 Norms of reaction for standardized fry length in Chinook salmon (*Oncorhynchus tshawytscha*). The norms of reaction are shown for (a) Big Qualicum fry and (b) Quinsam fry. Like symbols represent means from paternal half-sib families reared in a hatchery located either on their natal river or reciprocally translocated to the foreign hatchery. Phenotypic plasticity is indicated by nonhorizontal lines, and a genotype-by-environment interaction is indicated by nonparallel lines. Standardized fry length was calculated as (mean fry length within a family group − the mean length within the replicate)/standard deviation of the replicate mean length. A value of < 0 indicates a family with lower than average growth, and a value > 0 indicates a family with higher than average growth. The estimates of length are based on 681 fry from the Big Qualicum River and 688 fry from the Quinsam River. Note that the scales on the *y*-axes differ.

between parents in either population (Big Qualicum natal: $r^2 = 0.004$, $F_{1,60} = 0.008$, $P = 0.93$, Big Qualicum translocation: $r^2 = 0.006$, $F_{1,59} = 0.79$, $P = 0.38$; Quinsam natal: $r^2 = 0.004$, $F_{1,62} = 0.53$, $P = 0.47$, Quinsam translocation: $r^2 = 0.003$, $F_{1,62} = 0.42$, $P = 0.52$). Finally, across all adults, we did not detect a significant relationship between mean relatedness of a parent to individuals from the translocation population and juvenile survival during the larvae ($r^2 = 0.003$, $F_{1,30} = 0.079$, $P = 0.78$) or fry stage ($r^2 = 0.005$, $F_{1,30} = 0.15$, $P = 0.70$) when reared in the translocation environment. Thus, straying of adults is not likely to explain the observed variation in family performance.

Parent and offspring length

In the Big Qualicum population, male parents averaged 81.8 ± 6.3 cm, and females averaged 80.3 ± 3.5 cm. Quinsam River male parents averaged 84.6 ± 3.8 cm, and female parents averaged 80.8 ± 5.0 cm. For females in the Big Qualicum and Quinsam populations, the regression analysis did not reveal a significant relationship between female length and fry length (Big Qualicum: $r^2 = 0.013$, $F_{1,6} = 0.33$, $P = 0.59$; Quinsam: $r^2 = 0.002$, $F_{1,6} = 0.18$, $P = 0.69$). Similarly, we found no relationship between length of males and juvenile length in either population (Big Qualicum: $r^2 < 0.001$, $F_{1,6} = 0.003$, $P = 0.96$; Quinsam: $r^2 = 0.012$, $F_{1,6} = 0.39$, $P = 0.56$).

Discussion

In fishes, growing evidence indicates that genetic and maternal effects play an important role in survival and growth, two life-history traits that are tightly linked to fitness throughout development. Our study took an unprecedented step in following the survival of Chinook salmon family groups derived from a full factorial breeding design throughout the freshwater stages of development to examine the contribution of genetic and maternal effects. We found that additive genetic, nonadditive genetic and maternal effects played important roles in survival and growth. However, the magnitude of these effects varied between populations and across developmental stages. Such variation across development has been reported in other fishes, albeit only across the early stages of embryo and larvae development (Wedekind *et al.*, 2001, 2008). Here, we found that maternal effects played a primary role in survival during the larvae stage. These maternal effects could result from differences in egg quality among females (Heath *et al.*, 1999) or even differences in maternally transferred innate or adaptive immune substances to the egg (Lovoll *et al.*, 2007; Mulero *et al.*, 2007). During the fry stage, we found that maternal effects had no influence on survival, but instead found that additive genetic effects were important to survival. Thus, there was a shift from maternally derived factors to genetic effects. Previous studies in other taxa, particularly mammals, have found analogous results (e.g. Cheverud *et al.*, 1983; see Wilson & Réale, 2006 for a review). It is also possible that the shift we observed in Chinook salmon reflects a G × E interaction across different ages, in which some genes have a greater effect on survival in the environment associated with fry vs. larvae development. Regardless of the mechanism underlying the shift, our results are the first to follow survival through development in multiple wild fish populations and show a clear switch from

maternal effects to additive genetic effects as development proceeds.

Enhanced growth of juveniles may play an important role in individual survival (Roff, 1992), and thus, understanding the genetic architecture of growth can be important for understanding variation in fitness among individuals. We found that the growth of fry, as measured by body length, was influenced by additive genetic effects in both populations, albeit the effect was marginally nonsignificant in the Quinsam population. Perhaps surprisingly, the effect did not translate into a significant relationship between parental and juvenile length. The lack of this correlation may reflect the low variance in size among our dams and sires, which may have limited our statistical power. Heath *et al.* (1999) found that additive genetic effects explain 57% of the variation in growth in Chinook salmon larvae, compared to a mean of 36% of the variation in length explained by additive genetic effects in our populations. Similarly, Withler *et al.* (1987) reported that growth of Chinook salmon smolts exhibited a heritable component, although the degree of heritability varied by population. In Brook charr (*Salvelinus fontinalis*), 14% of the variation in alevin length was related to additive genetic effects (Perry *et al.*, 2004). These results collectively indicate that additive genetic effects play an important role in salmon growth. Furthermore, studies of other fishes implicate additive genetic effects on growth (Gjerde & Schaeffer, 1989; Hershberger *et al.*, 1990; Winkelman & Peterson, 1994), suggesting that growth is highly heritable in many fish populations.

When populations are faced with fluctuating environments, they may evolve plasticity in important fitness traits to cope with environmental heterogeneity (Via *et al.*, 1995). However, correspondence between the scale of environmental variation and the scale of plasticity in fitness traits is poorly understood for most species (Hutchings *et al.*, 2007), particularly when plasticity is considered at the level of the individual (Nussey *et al.*, 2007). We found extensive variation in the survival and growth of a given male or female's offspring to the rearing environment, especially during the fry stage of development. Indeed, for some males' half-sib families, survival and growth was considerably higher or lower in the translocation replicates than the natal-reared replicates, whereas other half-sib families exhibited virtually no change in survival or growth between the two environments. The observed half-sib family group response to environmental variation suggests significant individual-level genetic variation in plasticity. These results also suggest that Chinook salmon offspring survival is not necessarily expressed at its optima in the natal environment, which is in contrast to the prediction that salmonid natal philopatry is an adaptation that enhances offspring survival (Hendry *et al.*, 2004). Our observation of potential suboptimal survival and growth within natal environments for some families is unlikely to be influenced by parents that had strayed, because we did not find a relationship between survival in translocation family groups and the relatedness of their parents to individuals within the translocation population. Thus, our results, instead, support a role for plasticity in Chinook salmon offspring survival and growth across the two environments examined in this study and demonstrate significant individual-level variation in ability to respond to differing environments.

Within population variation in reaction norms (i.e. G × E effects) will determine the potential for plasticity to evolve (Pigliucci, 2001; Nussey *et al.*, 2007). Our results support a role for G × E effects on offspring survival and growth through the larval and fry stages. In a recent study of Atlantic salmon, the survival and growth of wild and farmed juveniles similarly showed G × E effects in response to temperature variation (Darwish & Hutchings, 2009). In contrast, a study in great tits found no evidence for G × E effects on laying date, clutch size and egg mass in response to differing temperature regimes experienced across years (Garant *et al.*, 2008; but see Nussey *et al.*, 2005b). In our study, we did not identify the specific sources of environmental variation potentially influencing phenotype expression between populations. However, because our families were reared in a hatchery environment, it is likely that only a few environmental factors varied. Our family groups were exposed to natural, untreated river water, which can be an important source of selection through pathogen exposure or water quality differences between populations (Amos & Thomas, 2002). For example, a study of Atlantic salmon juveniles showed significant temporal variation in bacterial infections within populations and also found evidence of spatial variation in infections across populations (Dionne *et al.*, 2009). Thus, it is possible that spatial or temporal variation in the pathogen community could be driving the maintenance of apparently suboptimal genotypes among family groups. It is also possible that the selective advantage of the various genotypes changes during later stages of development such as during the ocean phase of life. Further research is necessary to identify the specific environmental and genetic factors underlying the G × E interactions observed here and the evolutionary forces maintaining the genetic variation for plasticity in offspring survival and growth within the populations.

In conclusion, our results demonstrate a clear role for genetic and maternal effects on offspring survival and growth in Chinook salmon. However, we also found evidence that the expression of the genotypes contributing to survival and growth respond to environmental variation. The G × E effects varied among families, and thus, presumably selection could operate on the various reaction norms. It is also possible that spatial or temporal variation in selection maintains the genetic variation in survival and growth through changing directional selection. Consideration of such individual-level differences in

the ability to respond to environmental variation will have important implications for salmonid population recovery and conservation.

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