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Ontogentic shifts in genetic and maternal effects on length and survival in Chinook salmon (*Oncorhynchus tshawytscha*)

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ABSTRACT

Understanding how the interplay between genetic and environmental factors changes over the lifetime of a species is critical when selecting broodstock to optimize production at each life stage and reduce bottlenecks in the production chain. We analyzed changes in environmental, additive genetic, and non-additive genetic contributions in growth and saltwater survival across multiple life stages of Chinook salmon (*Oncorhynchus tshawytscha*) in an aquaculture facility to assess the importance of each factor throughout life. We used a full-factorial breeding design and followed fish for their entire 3-year life cycle to quantify the dam and sire effect on growth and survival and partition these effects into the respective environmental and genetic components. We show for body size that maternal and non-additive effects are the most important drivers of larval size, explaining a total of 87% of total larval phenotypic variation, but decline with age. Additive genetic effects peak during the juvenile stage, explaining a maximum of 15% of the total variation in size, but are much less important at earlier and later life stages. We saw a similar pattern to saltwater survival—with non-additive effects high (42% of total variance explained) at the earliest stage measured and decreasing in later stages and additive effects playing little role. Unlike growth, there was little maternal influence on survival. Taken together, our results show that maternal and non-additive effects are important drivers of larval size but that additive effects may play a more important role with age. Non-additive effects explained the most phenotypic variance observed for survival, playing a much larger role than maternal or additive effects. Our results add to a growing body of literature suggesting careful crosses of select lines can lead to enhanced growth and survival and that these effects can be tailored to the life stages of most concern for a given system. In this way hatchery managers can best develop breeding lines for specific systems and these lines can have stable effects over many generations.

Statement of relevance: The enclosed manuscript is directly relevant to commercial aquaculture. The study was conducted in a commercial aquaculture facility and the questions we are asking relate to the effects of genetic and non-genetic (maternal) factors in Chinook salmon growth and survival in an aquaculture facility.

1. Introduction

In aquaculture, growth and survival of fish are influenced by a complex interplay of genetic and environmental factors (Garcia de Leaniz et al., 2007; Sonesson, 2007; Gutierrez et al., 2012) that may have different effects at different life stages in the species of interest (Heath and Blouw, 1998; Evans et al., 2010; Bougas et al., 2013; Clark et al., 2014). When creating new offspring for each year-class, it is therefore vital to understand how much variation can be attributed to genetic and environmental influences and how much can be attributed to stochastic variation (Gjedrem et al., 2012; Yàñez et al., 2014). Complicating this question is the role of non-genetic maternal influences on fish growth and survival which can significantly influence the growth and survival of many fish species and, at least early in life, can override the genetic influences of the parents (Heath et al., 1999; Aykanat et al., 2012; Bougas et al., 2013). Partitioning parental influences into maternal environmental, additive genetic and non-additive genetic variance components is therefore necessary to enhance growth and survival throughout the life of commercially important species.

Fishes in the family Salmonidae are especially critical for assessment of genetic and non-genetic factors in growth and survival due to their importance as a commercial aquaculture species (Asche et al., 2013), as well as their non-resource-based mating systems and ease of external fertilization (Neff and Pitcher, 2005; Pitcher and Neff, 2007; Wedekind et al., 2008). In resource-based mating systems, offspring receive not only genes but additional parental care such as food, shelter and protection from predators, which can also affect offspring characteristics (Neff and Pitcher, 2005). Non-resource-based mating systems such as in the Salmonidae are therefore better suited for investigating the effects of

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genetic quality on a desired trait in offspring, as other confounding factors can be avoided (Neff and Pitcher, 2005; Pitcher and Neff, 2006, 2007; Wedekind et al., 2008; Hettyey et al., 2010; Houde et al., 2015). In salmonids, maternal effects significantly influence offspring size early in life (Heath et al., 1999; Debes et al., 2013; Houde et al., 2013) and can drive differences in survival both within and between populations, at least throughout the fry stage (Aykanat et al., 2012; Debes et al., 2013; Houde et al., 2013). Both additive and non-additive genetic components also explain growth and survival parameters in salmonids during at least some life stages (Wedekind et al., 2001; Wedekind et al., 2008; Huuskonen et al., 2009; Jacob et al., 2010; Falica and Higgs, 2013; Houde et al., 2015) but it is unclear precisely how the role of each of these three factors (maternal, additive and non-additive) changes throughout life in these species.

In the current study, we used Chinook salmon (*Oncorhynchus tshawytscha*), in a fully-factorial breeding design (Lynch and Walsh, 1998) using 7 dams and 7 sires to examine dam, sire, and interactive effects on offspring length and survival. Chinook is a major species of aquaculture interest and as of 2014 represented 10,840 t per year of harvest worldwide (FAO Factsheet, www.fao.org) We followed those two performance-related traits in the offspring for 3 years (from hatching through to the adult stage) and estimated the contributions of additive genetic effects, non-additive genetic effects, and maternal effects (non-genetic) on offspring length and survival at several time points throughout ontogeny. In this manner we were able to assess the use of a genetic-based approach to developing broodstock for aquaculture as well as increase our understanding of changes in genetic architecture of Chinook salmon through development.

2. Methods

2.1. Study species and breeding design

In the fall of 2008, we haphazardly selected 7 female and 7 male sexually mature (4 year old) Chinook salmon from a total population of 400–600 individuals to create 49 half- and full-sib families in a North Carolina Design II, which crosses the gametes of all dams and sires in every pair-wise combination (Lynch and Walsh, 1998). The dams and sires used in the current study were 7th-generation descendants originating from crosses between wild females taken from the Robertson Creek Hatchery (Port Alberni, British Columbia) and wild males taken from Big Qualicum River Hatchery (Qualicum Beach, B.C.) in 1985, and raised at the Yellow Island Aquaculture Ltd. (YIAL) hatchery and netcage site on Quadra Island, B.C. In 1997, YIAL began a marker-assisted broodstock selection program creating two different survival lines (termed a 'high-survival line' and a 'low-survival line') based on variation in growth and survival related gene markers (Docker and Heath, 2002). The descendants used in the current study were also from these two lines. The dams and sires for the current study were haphazardly selected until 3 of each were from the high-survival line and 4 were from the low-survival line, with identity established from previously implanted coded wire tags inserted into the nose of each fish. Broodstock were fed on Taplow certified organic Chinook feed (Taplow Ventures Ltd.) and were kept in salt water until September, when they are moved into freshwater tanks in preparation for spawning. All procedures were approved by the University of Windsor Animal Use and Care Committee.

2.2. Rearing conditions

The selected adult salmon were sacrificed via cerebral concussion, and gametes were extracted for artificial fertilizations, where an approximately equal amount of eggs from each female were fertilized by each male. We split fertilized eggs from each family into two cells of 100 eggs each in vertical stack incubation trays to account for location effects, therefore requiring 98 cells (2 per family). During incubation the trays were exposed to natural, untreated fresh water (from an artesian well) that ranged from 7 °C to 9 °C (pH = 7.15, hardness = 37 mg L^{-1}, alkalinity = 44.2 mg L^{-1}). UV-treated salt water (28 ppt) was pumped through the trays three times per week for 45 min to reduce fungus growth until hatching. The incubation trays were checked every other day until the end of the endogenous feeding stage to remove and record all unfertilized eggs and dead offspring.

At the end of the endogenous feeding stage in March 2009, up to 150 larvae from each family were transferred to a 200 L barrel, therefore requiring 49 barrels. Heath et al. (1999), which used a similar rearing design, did not find a correlation between rearing density (which could be different due to mortality differences among families) and growth. All barrels were cared for equally with flow-through fresh water ranging from 7 °C to 10.5 °C, aeration, and light from 7 am–5 pm. Fish care consisted of feeding the offspring daily with EWOS commercial salmon feed (EWOS Canada Ltd.), siphoning waste from the barrels every 5 days, and removing any dead offspring.

In our system Chinook smolt in May so in June 2009 a sample of 30 smolt (unless there were fewer remaining individuals) from each family were anaesthetized with clove oil and injected with Passive Integrated Transponder (PIT) tags to allow permanent individual identification ($n = 1379$). All tagged offspring from every family were then transferred to one 15 × 15 × 20 ft. (L × W × D) netpen at YIAL in the Pacific Ocean near Quadra Island B.C. Offspring were reared to adulthood, where in June 2010 all individuals were transferred to a bigger netpen 15 × 30 × 30 ft., and then later transferred once again to a new netpen (15 × 30 × 30 ft) in June 2011. In November 2010, any males that had become 'Jacks' (precocious sexually mature males) were removed from the netpen. During ocean life, fish were fed twice a day (Taplow Grower, Taplow Ventures Ltd.). Any mortalities were retrieved and scanned for their PIT tag to identify their dam and sire.

2.3. Body size measurements

We measured fork length and wet weight as metrics of body size five times throughout ontogeny as follows: '*Date 1*' = March 2009/End of larval stage; '*Date 2*' = June 2009/smolt stage; '*Date 3*' = November 2009/Juvenile stage; '*Date 4*' = June 2010/Adult stage; and '*Date 5*' = June 2011/Adult stage. For *Date 1*, we measured a sample of 20 fish per family. For *Date 2* we measured all PIT-tagged fish, which was 30 fish per family unless there were fewer remaining individuals. For *Dates 3*, *4* and *5* we measured all PIT-tagged fish that were still alive at the sample date.

2.4. Survival measurements

Survival was monitored at three time points after PIT tagging and saltwater entry, including '*Date 3*' = November 2009/Juvenile stage; '*Date 4*' = June 2010/Adult stage; and '*Date 5*' = June 2011/Adult stage. Survival was determined by the presence of PIT tagged individuals during sampling, and any individual PIT tags that were not detected during sampling were considered mortalities.

2.5. Statistical analysis

We were able to collect fork length data at all sample dates, but were not able to collect weight data at Date 4 when the fish were in netpens in the ocean due to rough sampling conditions. Thus, we chose to analyze only the length data as the length and weight were highly correlated (Spearman's correlation $r = 0.99$, $p < 0.001$). When testing the fork length data for normality, the Kolmogorov-Smirnov (K-S) test indicated that the data were statistically not normal and transformation of data often failed to improve normality. However, when sample sizes are large such as ours, small deviations from normality will frequently result in a significant result for the K-S test (Field, 2009, pg. 144). Thus, a significant K-S test does not necessarily mean that 'deviation' from

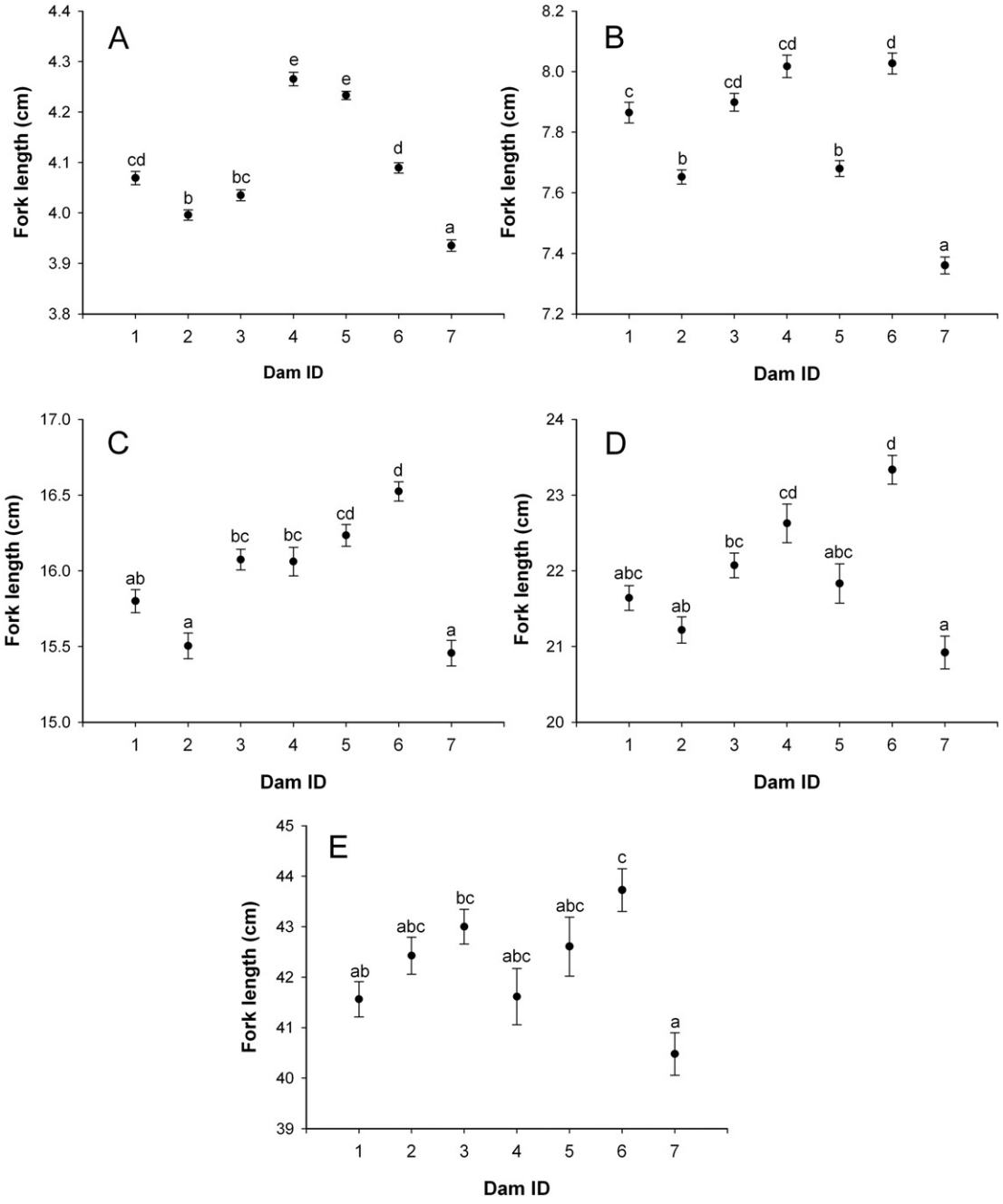

Fig. 1. Dam identity vs. mean fork length (mean ± 1 S.E.) for a) *Date 1*: March 2009, b) *Date 2*: June 2009, c) *Date 3*: November 2009, d) *Date 4*: June 2010, and e) *Date 5*: June 2011. Different letters for the homogenous subsets denote significant differences between dams ($p < 0.01$).

normality will bias the results when analyzing the data and therefore we instead chose to examine the normality plots (histograms and Q-Q plots) to view the scope of any non-normality (Field, 2009). For fork length data, histograms showed bell-shaped curves and Q-Q plots revealed observed values that fell exactly along the straight line (except for a only a few points at the ends) indicating that the data were near normal.

Data were analyzed using the lme4 package (Bates et al., 2009) in R software (R Development Core Team, 2011). Linear mixed models were used to determine whether dam, sire and their interaction significantly contributed to the phenotypic variance observed for body size. First, we partitioned phenotypic variance using the model (Lynch and Walsh, 1998):

$$\text{model}: \quad z_{ijk} = \mu + d_i + s_j + I_{ij} + e_{ijk}$$

where z_{ijk} is the phenotypic value of the k^{th} offspring from the i^{th} dam and j^{th} sire, and μ is the mean phenotypic value of the sample. In the model, dam (d), sire (s) and their interaction (I) were regarded as random effects and e denotes the residual error. To examine the contribution of dam (d), sire (s) and their interaction (I) to the phenotypic variance for body size, we contrasted the fit of models in a stepwise manner by removing the term and refitting the model. Models were fit using maximum likelihood, and the fit of the models were compared using log-likelihood tests. Variance component estimates from the models were used to calculate the total phenotypic variance to determine maternal, additive and non-additive phenotypic variance. The contribution of additive genetic effects to offspring body size was calculated from four times the sire component of variance, the non-additive genetic effects were calculated from four times the interaction (dam × sire) component of variance, and the maternal effects were

Table 1
Summary of linear mixed models examining the contribution of dam, sire and their interaction to the phenotypic variance of fork length at five sampling dates. The percent of phenotypic variance (% phenotypic var) for maternal effects, and additive and non-additive genetic effects are also included. Significant effects are indicated in bold with asterisk ($p < 0.01$).

Trait	Source of variation	σ^2 (% total var)			σ^2 (% phenotypic var)	
Fork Length						
Date 1 (larval stage)	Dam	**0.0115***	(40.0)	Maternal	0.0114	(40.0)
	Sire	0	(0)	Additive	0	(0)
	Interaction	**0.0034***	(11.8)	Non-additive	0.0135	(47.0)
	Residual	0.0138	(48.3)			
	Total	0.0287				
Date 2 (smolt stage)	Dam	**0.0450***	(19.7)	Maternal	0.0398	(17.5)
	Sire	**0.0052***	(2.3)	Additive	0.0210	(9.2)
	Interaction	**0.0120***	(5.3)	Non-additive	0.0480	(21.1)
	Residual	0.1656	(72.7)			
	Total	0.2279				
Date 3 (juvenile stage)	Dam	**0.1242***	(11.7)	Maternal	0.0851	(8.0)
	Sire	**0.0391***	(3.7)	Additive	0.1564	(14.8)
	Interaction	0.0176	(1.7)	Non-additive	0.0705	(6.7)
	Residual	0.8776	(82.9)			
	Total	1.0586				
Date 4 (early adult stage)	Dam	**0.5452***	(10.1)	Maternal	0.5137	(9.5)
	Sire	0.0315	(0.6)	Additive	0.1260	(2.3)
	Interaction	0	(0)	Non-additive	0	(0)
	Residual	4.832	(89.3)			
	Total	5.409				
Date 5 (later adult stage)	Dam	**0.8052***	(5.5)	Maternal	0.4683	(3.2)
	Sire	0.3369	(2.3)	Additive	1.3476	(9.1)
	Interaction	0	(0)	Non-additive	0	(0)
	Residual	13.602	(92.3)			
	Total	14.745				

calculated from the difference between dam and sire components of variance (reviewed in Neff and Pitcher, 2005). The alpha level was adjusted to 0.01 (0.05/5) for all dates to account for the same individuals being measured throughout this study. When a significant random effect of dam and/or sire was detected, a two-way ANOVA with Tukey's post-hoc test was performed to determine which dams and sires differed significantly ($p < 0.01$) in offspring body size.

Saltwater survival data were analyzed using generalized linear mixed effect models for binomial data. Offspring that survived were coded as "1" and mortalities were coded as "0". Data were analyzed in R with the "glmer" function, where models were fitted and evaluated in a manner similar to that outlined above. The alpha level was adjusted to 0.017 (0.05/3) for all dates to account for the same individuals being measured throughout this study. When a significant random effect of dam and/or sire was detected, logistic regressions with post-hoc Tukey tests were used to determine which dams and sires differed significantly ($p < 0.017$) from one another in offspring survival.

3. Results

3.1. Body size

Dam had a significant effect on offspring fork length throughout all life stages (Fig. 1), where the percentage of the total variance in fork length attributed to dam declined from the larval stage (40% total variance) to the later adult stage (6% total variance) (Table 1). Consequently, maternal effects on fork length variance declined from 40% at the larval stage to only 3% at the later adult stage (Table 1). Sire effects only contributed significantly to the variation in fork length at two sampling dates, which included the smolt stage and juvenile stage (Fig. 2). The total variation in fork length attributed to sire effects was <4% throughout all stages (Table 1). Additive genetic effects on the phenotype ranged from 0% at the larval stage to 15% at the juvenile stage, and at all other stages additive effects accounted for <9% of the variance. The interaction of dam and sire had a significant effect on fork length during the larval stage and smolt stage, where the interaction effect accounted for 12% and 5% of the total phenotypic variance, respectively (Table 1). However, it should be noted that at the smolt stage, interaction (family) effects could not be separated from barrel effects, as each family was reared in separate barrels. At later stages, the interaction effect represented <2% of the phenotypic variance. Non-additive genetic effects were therefore also largest during early development (47% at the larval stage) and declined throughout life (0% at later adult stage).

3.2. Saltwater survival

Maternal effects accounted for <3% of the total variance in saltwater survival at three sampling dates (Table 2), with significant dam effects only at the latter two dates (Table 2, Fig. 3). Sire effects contributed significantly to the variance in saltwater survival during the juvenile stage but not later stages (Fig. 4), and at all dates sire effects represented <1% of the total variance in survival (Table 2). Similarly, additive genetic effects were highest at the juvenile stage (5%) and represented 0% and 2% of the variance at early adult and later adult stages, respectively. The interaction of dam and sire had a significant influence on saltwater survival at the juvenile stage (10% of total variance) and early adult stage (3% of total variance), but not at the later adult stage (<1% of total variance) (Table 2). Non-additive genetic effects contributed to 42% of the variance in saltwater survival at the juvenile stage but declined to 13% and 3% at the early and later adult stages, respectively (Table 2).

4. Discussion

The current study presents the first investigation to our knowledge to follow the contributions of additive and non-additive genetic effects and maternal effects to variation in phenotypic traits in salmon from larval stages through to adulthood. In previous studies using species with non-resource-based mating systems to determine all three contributions (additive, non-additive and maternal effects) to offspring size and survival, the results among studies were inconsistent. Previous studies examining species with non-resource-based mating systems have shown effects varying from significant maternal and additive effects (in Chinook salmon fry, Evans et al., 2010), to non-additive and maternal effects but little or no additive effects (in larval Lake Ontario Chinook salmon, Pitcher and Neff, 2007 and in juvenile Atlantic salmon,

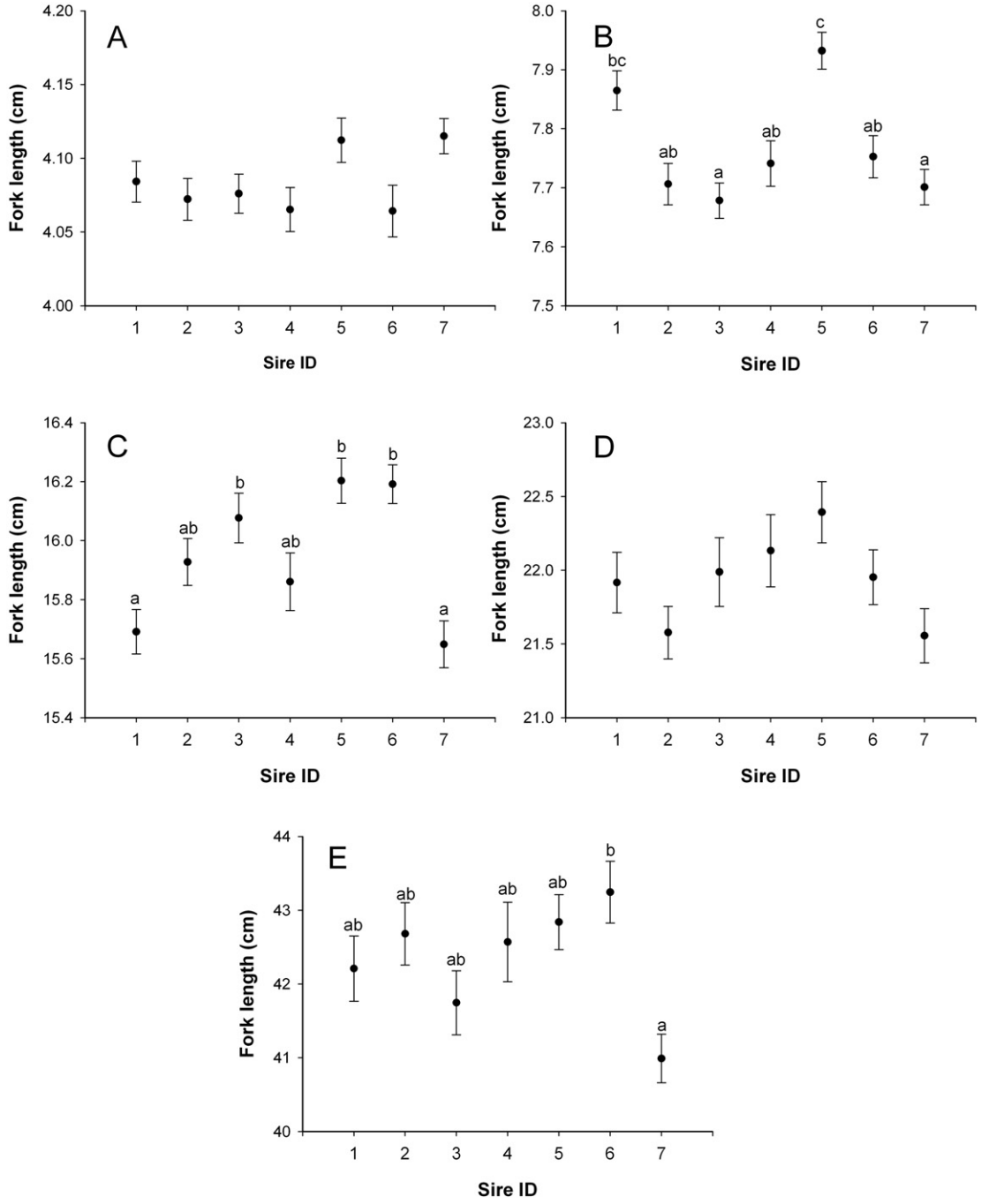

Fig. 2. Sire identity vs. mean fork length (mean ± 1 S.E.) for a) *Date 1*: March 2009, b) *Date 2*: June 2009, c) *Date 3*: November 2009, d) *Date 4*: June 2010, and e) *Date 5*: June 2011. Different letters for the homogenous subsets denote significant differences between sires ($p < 0.01$).

Salmo salar, Houde et al., 2015), to additive effects on length but not on weight, showing variation due to what measure of size was used (in larval Atlantic herring, *Clupea harengus*, Bang et al., 2006). Similarly, significant effects on offspring survival ranged from non-additive and maternal effects (in embryonic sea lamprey, *Petromyzon marinus*, Rodriguez-Munoz and Tregenza, 2009 and in Atlantic salmon fry, Houde et al., 2015), to all three (additive, non-additive and maternal) (in larval Lake Ontario Chinook salmon, Pitcher and Neff, 2007). The differences among previous studies are likely due to environmental variation, species differences in genetic architecture, and especially developmental stage.

In the current study, we found that results varied across development, with the genetic and maternal factors explaining variation in length generally decreasing over time. Interestingly, the dam component of variance remained significant on offspring length for all five dates measured. However, when the dam component was separated and the true maternal effects examined, maternal effects went from representing 40% of the phenotypic variation at the larval stage to 17.5% at the smolt stage, and continued to decrease to only 3.2% by the adult stages. The fact that we found higher maternal effects early in development supports the established concept that maternal effects decrease over time, due to other factors such as offspring genome and environmental quality increasing in their influence (e.g. Heath et al., 1999; reviewed in Heath and Blouw, 1998; and in Green, 2008). The non-additive (dam x sire) effects on length in the current study also decreased over time, suggesting that genetic compatibility does affect

Table 2
Summary of generalized linear mixed models examining the contribution of dam, sire and their interaction to the phenotypic variance of survival for three sampling dates. The percent of phenotypic variance (% phenotypic var) for maternal effects, and additive and non-additive genetic effects are also included. Significant effects are indicated in bold with asterisk ($p < 0.017$).

Trait	Source of variation	σ^2 (% total var)			σ^2 (% phenotypic var)	
Saltwater survival						
Date 3 (juvenile stage)	Dam	6.0×10^{-9}	(0)	Maternal	−0.0418	(0)
	Sire	**0.0418***	(1.1)	Additive	0.1672	(4.5)
	Interaction	**0.3853***	(10.3)	Non-additive	1.5412	(41.4)
	Residual	$\pi^2/3$	(88.5)			
	Total	3.7271				
Date 4 (early adult stage)	Dam	**0.0307***	(0.9)	Maternal	0.0307	(0.9)
	Sire	0	(0)	Additive	0	(0)
	Interaction	**0.1097***	(3.2)	Non-additive	0.4388	(12.8)
	Residual	$\pi^2/3$	(95.9)			
	Total	3.4404				
Date 5 (later adult stage)	Dam	**0.1185***	(3.4)	Maternal	0.0994	(2.9)
	Sire	0.0191	(0.6)	Additive	0.0764	(2.2)
	Interaction	0.0271	(0.8)	Non-additive	0.1086	(3.1)
	Residual	$\pi^2/3$	(95.2)			
	Total	3.4648				

Note: Generalized linear mixed models for binomial data with logit link function have an underlying residual variance of $\pi^2/3$ (see Nakagawa and Schielzeth, 2010; Johnson and Brockmann, 2013).

length but the effects are life-stage specific. Similarly, in a previous study on Chinook salmon, maternal and non-additive effects contributed to larval growth (comparable to our Date 1 measurement), which represented 11% and 73%, respectively, of the phenotypic variation (Pitcher and Neff, 2007). In the current study, we found that non-additive effects were higher than maternal effects (by 7%) for larval length, which was also seen in Pitcher and Neff (2007), although their non-additive effects were much larger than the maternal effects.

In our study additive effects represented a small, but at smolt and juvenile stages significant, portion of phenotypic variance. Additive effects on length have been previously reported at the fry stage in Chinook salmon (Evans et al., 2010); however their estimates were much larger—representing 39% for one population of Chinook salmon, and 33% for a second population of Chinook. That we found no additive effects on larval length is also consistent with previous work on larval Lake Ontario Chinook salmon (Pitcher and Neff, 2007) and in Atlantic salmon (Houde et al., 2015). However, sire effects have been demonstrated to contribute significantly to offspring swimming ability in Chinook salmon (Falica and Higgs, 2013), but only in older juveniles, and the contribution of additive genetic effects to phenotypic variation increased from 26% (at 15 weeks post-hatch) to 100% (at 18 weeks post-hatch). Additionally, previous studies have shown that additive genetic effects are important for body size in fish and explain for example, 14% of the variation in alevin length in brook charr (*Salvelinus fontinalis*) (Perry et al., 2004); 57% of the variation in larval size in Chinook salmon (Heath et al., 1999); and 65% of the variation in larval standard length in Atlantic herring (Bang et al., 2006). Overall, the current study shows changes in contributions of additive, non-additive and maternal effects throughout developmental stages. Combining our results with the findings of previous studies, it seems maternal effects and non-additive effects contribute to larval length, which switches to additive effects playing a role when older, demonstrating an ontogenetic shift in the importance of these effects. Applying our results in an aquaculture setting will allow managers to fine tune breeding strategies to maximize different stages of growth relevant for their particular operational needs, for example when selecting for fish that can be transferred to salt water earlier versus those that might achieve maximal adult size.

For survival, we also found that the factors explaining variation generally decreased over time, except for maternal effects which increased slightly over time. Non-additive effects explained the most phenotypic variance observed for survival, playing a much larger role than maternal or additive effects. Similarly in sea lamprey, non-additive effects play a much larger role than maternal effects, with non-additive effects representing 65.5% whereas maternal effects represent only 14.8% of the phenotypic variation (Rodriguez-Munoz and Tregenza, 2009). Although we did not find significant additive genetic effects later in life this may be due to the fact that we used hatchery fish, as Evans et al. (2010) found additive effects in late-stage wild populations which may be expected to have greater genetic diversity than our hatchery fish.

In the current study, the offspring were reared in a common environment, given the same amount of food, where predators were absent and where other factors that normally influence survival (e.g. competition for resources) were likely minimal due to the aquaculture setting. This was done to minimize confounding factors and test applicability to aquaculture settings, so any differences in size or survival seen among the offspring could be attributed to differences in genetic quality (and maternal effects). It is possible that had the offspring been reared in the wild where selection pressures (e.g. due to risk of starvation or predation) are higher, differences in size and/or survival among the offspring may have been more pronounced. Large genotype x environment interactions have been shown in many species of fish (e.g. Heath et al., 1993; Devlin et al., 2004; Donelson et al., 2009; Darwish and Hutchings, 2009; Evans et al., 2010) so it is important to note that if genotype x environment interactions exists, the different genotypes in the current study may respond differently to changes in the environment (Lynch and Walsh, 1998).

Although the proportion of variance in offspring length attributable to among dam and among sire effects is small, it is well known that the size of offspring, especially in the early stages of development is a major factor influencing survival and recruitment (e.g. Jenkins and King, 2006; Fontes et al., 2011; reviewed in Chambers and Leggett, 1996). For instance, being larger at hatching offers several benefits such as having more time to find food sources before starvation (Miller et al., 1988), being too large for smaller predators to handle and consume (Bailey, 1984), and having sense organs and swimming ability more developed thus assisting in predator detection and escape (Bailey, 1984; Bailey and Batty, 1984; Fuiman et al., 2004). In Pacific salmon specifically, larger smolts also possess several advantages including better escape from predators and ability to catch prey due to enhanced swimming ability, and ultimately greater survival when migrating to and entering the sea (Beckman et al., 2003). In steelhead trout (*O. mykiss*), smolt-to-adult survival had a positive relationship with length (Ward et al., 1989). Thus, it would be interesting to see if the differences in offspring length among dams and sires seen in the current study would influence the fitness of the offspring if they were in the wild.

In conclusion, our study adds to growing evidence that genetic architecture of performance traits varies among individuals and across

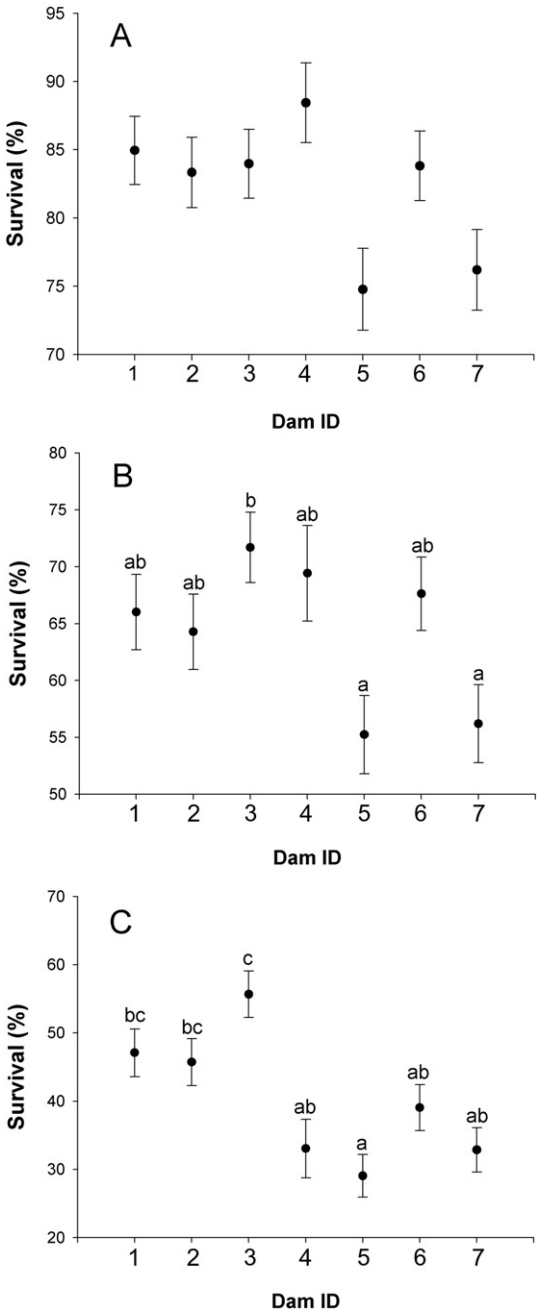

Fig. 3. Dam identity vs. mean (±1 S.E.) percent survival a) *Date 3:* November 2009 b) *Date 4*: June 2010, c) *Date 5*: June 2011. Different letters for the homogenous subsets denote significant differences between dams ($p < 0.017$).

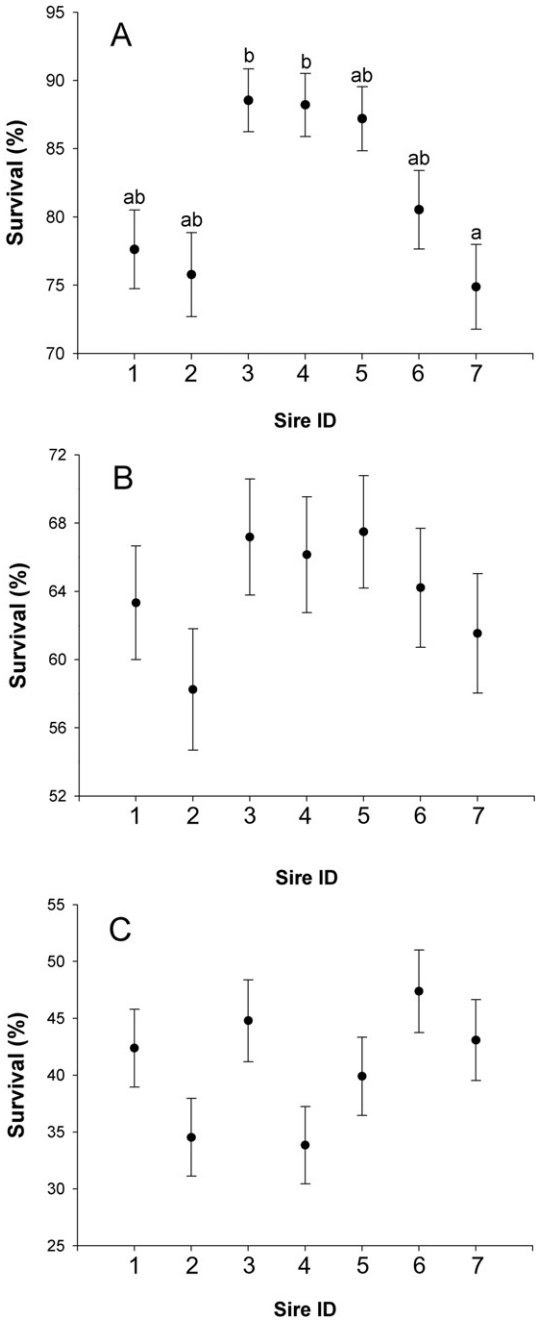

Fig. 4. Sire identity vs. mean (±1 S.E.) percent survival a) *Date 3:* November 2009 b) *Date 4*: June 2010, c) *Date 5*: June 2011. Different letters for the homogenous subsets denote significant differences between sires ($p < 0.017$).

development. No other study to our knowledge has partitioned phenotypic variance components from larval to adult stages. We found that genetic and maternal effects play an important role in larval length and survival, and that these effects decrease with age, possibly due to environmental variation masking genetic effects. Our study also adds to evidence of non-additive genetic effects (i.e. genetic compatibility) affecting larval length, suggesting that 'compatible genes' can play very important roles in larval survival. This also suggests that individuals who get to choose their mates may benefit by having offspring with higher fitness if they find these more genetically compatible individuals, thus future breeding programs that mate individuals randomly may want to consider this to optimize growth and survival in aquaculture systems.

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