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Environment-specific heritabilities and maternal effects for body size, morphology and survival in juvenile Atlantic salmon (*Salmo salar*): evidence from a field experiment

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Abstract Environmental heterogeneity may strongly influence the amount of heritable variation in phenotypic traits and thus affect evolutionary responses to natural selection. However, the question of whether heritabilities change across environmental gradients has received little empirical attention, particularly for wild vertebrates. We tested whether levels of heritable variation in body size, morphology and survival of juvenile Atlantic salmon (*Salmo salar*) differed between water flow regimes. We exposed individuals of known genetic relationships to rearing habitats characterized by slow and rapid water flows in a field experiment. We found that the additive genetic variation in body size tended to be higher for individuals reared under rapid water flows. By contrast, the heritabilities of other morphological traits were not consistently higher in either water flow. We also found that salmon grew faster under rapid water flows but also suffered high mortality rates with little heritable variation explaining the variation in survival. However, part of the variation in survival in the rapid water flow was explained by maternal effects. Our results suggest a strong tendency for heritable variation, particularly in body size to be revealed only under specific environmental conditions, such as those that allow for rapid growth. We provide support for the hypothesis that genotype by environment interactions have important effects on the adaptive potential of phenotypes in nature.

Keywords Additive genetic variation · Water flow · Rearing habitats · Juvenile salmon · G × E interactions

Introduction

The principal concept associated with phenotypic plasticity is that environmental conditions experienced by genotypes can, to some extent, generate phenotypic variation (DeWitt et al. 2004; Bradshaw 1965). Reaction norms are the functions that describe the change in traits across environments (Schlichting and Pigliucci 1998). Previous studies have shown that selection can shape reaction norms by favoring optimal mean trait values in different environments, as long as the conditions of sufficient additive genetic variation and selection intensity are met (McGuigan and Sgró 2009;

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Gomulkiewicz and Kirkpatrick 1992; Via and Lande 1985). Some evidence from natural systems further suggest that both genetic variability and the strength of natural selection depend strongly on environmental conditions (Dingemanse et al. 2009; Wilson et al. 2006; Merilä 1997). However, most of this evidence is limited to a few well-studied species for which long-term ecological and phenotypic data are available. For non-model wild organisms, we still know little about how environmental heterogeneity affects the heritable variation of phenotypes (but see Wood and Brodie 2015).

Understanding the processes that shape trait variation under different environmental conditions is also important to predict evolutionary responses of wild organisms in space and time. In particular, many eco-evolutionary models are built to allow for changing strengths of selection, but often fix additive genetic variation to specific values based on estimates obtained from laboratory and field experiments (but see Thériault et al. 2008). Yet, model predictions may change dramatically if genetic variance changes in space or time (Shaw and Shaw 2014). Obtaining data on how genetic variance changes across different environments in wild organisms will therefore be invaluable to better understand the role that adaptive responses in plastic traits play under future climatic scenarios (e.g. Wilson et al. 2006; Charmantier and Garant 2005; Hoffmann and Merilä et al. 1999; Merilä et al. 1999).

For many salmonids, including the Atlantic salmon (*Salmo salar* L.), rapid and slow water flow habitats are respectively thought to represent rearing environments that favor high and low juvenile growth (Fausch and White 1981; Fausch 1984; Allan 1995). Although rapid water flows impose high energetic demands on fish movement through elevated drag forces (Pettersson and Hedenström 2000), these habitats are also associated with a greater abundance of invertebrate drift (i.e. food) (Nislow et al. 1998). Rapid water flow is characteristic of stream habitats which often produce juveniles of greater body sizes for a given age (Aubin-Horth et al. 2005; Páez et al. 2010). Conversely, slow water flows, while minimizing drag forces on fish movement, also carry less invertebrates per unit time (Fausch 1984; Nislow et al. 1998). Such habitats are a feature of larger river sections, which on average, produce juvenile fish with smaller body sizes (e.g. Aubin-Horth et al. 2005; Páez et al. 2010). Variation in body size and morphology has also been documented to occur across different spatial scales in other species of salmonids (Beacham and Murray 1985; 1987; Bailey and Kinnison 2010)

Even though juvenile salmon are highly territorial (Metcalfe and Thorpe 1992; Metcalfe et al. 1995), fish also move about and exploit a range of water flow velocities (Roy et al. 2013; Hedger et al. 2005). Nevertheless, previous studies have found that individuals reared in habitats with rapid water flow differ in size and morphology compared to fish found in habitats with slow water flows (Godin and Rangeley 1989; Pakkasmaa and Piironen 2000; Páez et al. 2008).

Under laboratory conditions, morphological differences can be explained by phenotypic plasticity (Pakkasmaa and Piironen 2000). However, specifically for newly emerged salmon, density dependant regulation (Elliott 1989), territorial disputes (Metcalfe et al. 1989), and size-selective mortality (Good et al. 2001; Einum and Fleming 2000; Sogard 1997) may also contribute to generate variation in survival, body size and morphology across rearing habitats. Previous work has further shown that size selective mortality acts strongly on juvenile salmon (Elliott 1989; Good et al. 2001; Bailey and Kinnison 2010), having profound effects on subsequent growth, survival, and recruitment of migrant and resident life-history tactics (Aubin-Horth et al. 2005). Moreover, recent evidence suggests that the patterns of size-selective mortality differ among and within river systems (Bailey and Kinnison 2010; Aubin-Horth et al. 2005). This implies that the intensity of selection on juvenile body size varies even across small spatial scales. However, the adaptive consequences of size-selective mortality are not fully understood because we do not know how genetic variation in survival and body size differs between the different rearing habitats experienced by newly emerged salmon (but see Garant et al. 2003). Such information, however, is crucial for understanding the mechanisms generating phenotypic diveristy in salmonids and assessing potential conservation strategies (Carlson and Satterthwaite 2011; Watters et al. 2003).

Here, we test for the effects of environmental heterogeneity on the causal sources of phenotypic variation by conducting quantitative genetic experiments under conditions that closely mimic natural conditions. Specifically, we test whether body size, morphology and survival measured on juvenile

salmon harbour different levels of heritable variation when reared in environments characterized by rapid or slow water flows. We also test whether survival is related to body size to identify potential mechanisms generating the diversity in juvenile body size observed in nature (Aubin-Horth et al. 2005; Páez et al. 2010).

Methods

Mating design and embryonic rearing

We captured six females and twelve males (six anadromous and six mature parr) in late August 2006 from the Sainte-Marguerite River, Québec, Canada (48° 20'N, 70° 00'W) to use as progenitors in our breeding design. We use both resident and anadromous sires in our experiment because previous preliminary evidence suggested that the sire's sexual tactic may affect juvenile growth (Garant et al. 2002). For each female, we fertilized approximately 660 eggs with the sperm of six males (that is, three anadromous and three residents) to produce maternal and paternal half-sib families for a total of 36 full-sib families, as depicted in Table 1. We allowed egg development to occur under temperatures characteristic of the Sainte-Marguerite River and recorded embryonic mortality on a weekly basis. Alevin emergence, which is the onset of juvenile life, was visually assessed by noting juvenile swimming behaviour and occurred between June 16 and 18, 2007. Because fertilization success and embryonic mortality varied across families, we had a variable number of juveniles per family available for our experiments (Table 1).

Field experiment

Fish allocation to the experimental treatments

On June 18 2007 we randomly sampled 8 individuals per full-sib family to use as initial measures for growth rate calculations. We then allocated the remaining individuals to experimental channels installed in a river site containing slow or rapid water flows. Specifically, juvenile fish from each one of the 36 full-sib families were separated into two equally-sized groups and allocated to either the slow or rapid water flow treatment (Table 1). To assure that we could track individual family identity, we installed at total of 72 experimental channels along a 400 m river segment

Table 1 Mating design for field experiment

Sire	Dam					
	1	2	3	4	5	6
1P	76, 0.76, 0.68				100, 0.72, 1	96, 0.56, 0.79
2A	117, 0.74, 0.88				81, 0.4, 0.63	97, 0.69, 0.67
3P	108, 0.78, 0.87	96, 0.29,0.58				100, 0.7, 0.98
4A	50, 0.2, 0.88	53, 0.35, 0.56				88, 0.34, 0.91
5P	51, 0.44, 1	47, 0.43, 0.67	29, 0, 0.79			
6A	67, 0.59, 0.79	41, 0.4, 0.62	52,0.12, 0.58			
7P		46, 0.3, 0.83	41, 0.5, 0.62	87, 0.02, 0.95		
8A		86, 0.79, 1	38, 0.21, 0.63	72, 0.58, 0.89		
9P			66, 0.36, 0.48	78, 0.62, 0.56	99, 0.71, 1	
10A			82, 0.63, 0.85	84, 0.43, 0.74	96, 0.58, 1	
11P				85, 0.67, 0.88	82, 0.63, 0.98	91, 0.29, 0.98
12A				77, 0.61, 0.97	86, 0.28, 1	83, 0.39, 1

Note: Values indicate the total number of newly emerged Atlantic salmon introduced to the field experimental channels (i.e. available at emergence), followed by the percent mortality in the slow and the rapid channels, respectively. Crosses for dams with ids 1-3, 4-5, and 6 were performed on 25th, 31st October 2006, and 11th November 2006, respectively. P = precocious resident male sire, A = anadromous male sire. All females were anadromous

previously known as a productive area for juvenile growth. On average, juvenile density was 32 fish per m^2 (range: 20-56), which is within the densities observed in suitable rearing habitats in nature (Atkinson et al. 2004).

Channel construction, placement and maintenance

Channels were constructed using plywood and were 2 m long × 0.6 m wide × 0.6 m deep. The upstream and downstream ends of the channels were sealed with a screen that impeded fish from escaping, while allowing the natural flow of small invertebrates (further details in Blanchet et al. 2008) (Fig. S1). To recreate the substrate structure characteristic of salmon habitats, we lined the floor of each channel with river pebbles. In addition, we added six large rocks within each channel to provide additional fish shelter. To avoid avian predation, we covered each experimental channel with a screen that had little effects on light conditions.

Channel placement in the river site was based on water speed readings using an ultrasonic flow meter and on previous familiarity with the site Paéz et al 2008; 2010). Water temperature and velocity was recorded at three additional times during the course of the experiment (Table S1). On average, water flows were 3.4 times faster in the rapid experimental channels (Table S1) and were characteristic of water flows where we had previously captured juvenile salmon (Hedger et al. 2005).

We installed channels within the river in a zigzag pattern, assuring that downstream channels were at least 2 m downstream from upstream channels and that their upstream ends did not overlap with the downstream ends of other upstream channels (Fig. S1). We conducted channel maintenance on a daily basis, recording any mortality. On July 10, 2007 unfavorable weather conditions, which risked flooding the experimental channels, forced us to halt the experiment.

Data collection

We extracted individuals from their experimental channels with nets in preparation for morphological measurements. All fish were sacrificed the same day when retrieved from channels. Across the 36 families, we recovered 228 and 665 individuals from the rapid and slow water flow treatments, respectively. Each individual was then pinned by the fins to a grid and photographed on the right side of its body. We then used Image J (Schneider et al. 2012) to measure five morphological traits that are commonly measured to capture the variation in fish morphology (Pakkasmaa and Piironen 2000; Páez et al. 2008). Measures of distance between landmarks were calibrated using the same ruler for all images. The traits measured were: 1) standard body length (most anterior point of the head to the most posterior point of the hypural plate), or BL; 2) head depth (the highest dorsal point of the head to the lowest ventral point of the head), or HD; 3) body depth (origin of dorsal fin to the most ventral point in a straight line), or BD; 4) caudal peduncle depth (most dorsal point of the hypural plate to the most ventral point of the hypural plate), or CP; and 5) caudal peduncle length (insertion of the anal fin to most posterior point of the hypural plate), or CAUD (Fig. 2). Individual survival was recorded as a binary trait based on initial minus final fish counts.

Statistical analyses

Testing for growth differences between rearing treaments

To show that the rearing habitats had an effect on growth which then translated to other differences in phenotypic variation, we calculated specific growth rates using $\frac{ln(x_T)-ln(x_t)}{T-t} \times 100$, where x_T and x_t are the mean values for a given trait, x, before (at time t) and after (at time T) exposure to the water flow treatments. The growth statistic was calculated at the water flow treatment level because juveniles sampled at emergence had to be sacrificed due to logistical considerations. We tested whether rearing habitats lead to significant growth differences using a bootstrapping resampling procedure that allowed us to account for the variability in the data rather than only considering the mean values. Briefly, the data for a given trait were sampled with replacement 10^4 times. At each sampling event we re-calculated the trait mean before and after the experiment and then calculated the growth statistic. This allowed us to generate two distributions of growth associated with the two water flow treatments. We then considered that growth differed between treatments if the 95 percentiles of these two distributions did not overlap.

Power and sensitivity analyses on the pedigree design

To determine if additive genetic variance could be estimated from the sample size resulting from the experiment, we conducted a power and sensitivity analysis of the models used to estimate heritability. For simplicity, we treated the pedigrees (Table 1) from each water flow independently and simulated Gaussian phenotypic records with different levels of additive genetic variation and maternal effects.

We simulated phenotypes with heritabilities ranging from 0.02 to 0.5 at 0.02 intervals and maternal effects equal to 0, 0.15, 0.30 and 0.50. For each combination of heritability and maternal effects, we generated phenotypic records for the individuals in the pedigree 10^3 times, using the software Pedantics (Morrissey et al. 2007). For each simulation we then fit a mixed effects model known as an "animal model" to estimate heritability and variation due to maternal effects. In this model we included a global intercept as a fixed effect and both the animal and the maternal identity as random effects. However, when no maternal effects were used to generate phenotypes (i.e. maternal effects = 0), the random structure of the animal model only included animal effects.

For all scenarios of heritability and maternal effects, we then fit a second model which omitted the random animal effect. Next, we used a log-likelihood ratio test to determine whether including the animal effect significantly increased the likelihood of the data based on the probability threshold value of 0.05 under a Chi-square distribution (Lynch and Walsh 1998). To quantify the range of heritability values that could be detected with confidence from our pedigree designs, we defined power as the fraction of significant log-likelihood ratio tests over the total number of tests (Morrissey et al. 2007). We also monitored biases in heritability estimates for each of the heritability/maternal effects scenarios (Fig. S2). Variance component estimation in these simulations was performed using Wombat (Meyer 2007).

Variance component estimation for the measured traits

We estimated heritabilities and maternal effects using the "raw" measurements taken from individual photographs. However, a complication with estimating variance components on "raw" values is that trait variation could be partly attributed to effects of body size (Reist 1985). By removing the effect of size from traits, however, we were able to analyze variation that was unique to each trait, which we interpreted as morphological variation (Reist 1985). To correct for body size, we therefore extracted residuals from a linear regression between each of the four traits and body length (which measures body size). We then used these trait residuals as size-corrected traits to also estimate the heritability and the variation due to maternal effects of morphology.

Variance component estimation was conducted using animal models. In these models, the explanatory variables used as fixed effects were the water flow treatment and the sire's sexual tactic (i.e. whether the sire was anadromous or resident). We included the sire's sexual tactic as a non-genetic source of paternal effects because some previous work suggested that offspring produced by the alternative male tactics may grow at different rates (Garant et al. 2002), although the non-genetic mechanism for this process is unclear.

The random structure of the animal model included the offspring and dam identity, a channel effect to account for common rearing environments, and additional residual effects. To obtain an estimate of additive genetic variation associated with each treatment, we also fit categorical random interactions (Hadfield 2010) between the animal effect and the water flow treatment. Similarly, to obtain an estimate of maternal sources of variation specific to each environment, we also specified a categorical random interaction between the maternal identity and the water flow treatment. Notice that including channel effects in our models allowed us to account for variation arising through differences among experimental channels, which are often named common environmental effects (Kruuk 2004; Wilson 2008). In our experiment, these effects could have captured differences in channel rearing densities and other micro-climatic conditions. All traits, except survival, were verified to be normally distributed by visual examination of the data, and this allowed us to assume Gaussian errors in the animal models.

By contrast, animal models using survival data failed to converge. To circumvent this problem, we instead based the variance partitioning of survival on models that considered the variation across paternal half-sibling families (Falconer and Mackay 1996)

rather than among individuals. For this, we used a mixed effects model in which the sire (N = 12), dam (N = 6) and channel identity (N = 72) were included as random effects. Because survival was a binary trait, we also used a logit-link function and binomial errors. To then estimate variation in survival that was specific to the water flow treatments we specified categorical random interactions between (i) the sire identity and water flow treatment and (ii) the dam identity and the water flow treatment. Heritability was then estimated using $h^2 = 4 \times \frac{V_A}{V_P}$, which is standard for half-sib designs (Falconer and Mackay 1996).

In all of our analyses we used Markov-chain Monte Carlo (MCMC) tools for parameter estimation (Hadfield 2010). We discarded the first 15000 steps of our MCMC chains, and ran a total of 2×10^6 iterations. The chains were then thinned by using only 1 in every 1000 steps, which assured a negligible (< 0.1) autocorrelation between posterior samples. We used informative proper priors to estimate genetic and maternal effects. Specifically, prior variances were specified by multiplying the observed phenotypic variation of the trait by the heritability or the estimated maternal effect obtained in Páez et al. (2010). For all other parameters we used uninformative priors. We also verified that prior specification did not affect the posterior distribution of parameters by running all analyses with uninformative priors. We assessed significance of the estimated parameters by examining the 95 % credible intervals obtained from posterior distributions. In these analyses we used the package MCMCglmm (Hadfield 2010) available in R (R Core Team 2012).

Effects of survival on body size

Lastly, to test whether differences in survival across treatments were related to juvenile body length, we used a linear mixed effects model and a bootstrapping procedure. In this model, the mean juvenile body length of a family was a function of linear and quadratic effects of the family-percent survival. We also included the maternal identity as a random effect because different families could have the same dam. We then bootstrapped this model to account for the variation around body length means. Specifically, we first recalculated family body length means by randomly resampling individuals within families 10^4 times. We then re-fit this model and used the generated distribution of the model coefficients to determine if the relation between body size and survival was statistically significant.

Results

Effects of rearing treatment on fish growth and trait variation

We found higher specific growth rates for all traits in the rapid water flow treatment. These results were statistically significant based on non-overlapping 95 percentiles of the bootstrapped distributions (Fig. 1). We did not, however, find any evidence for trait differences between salmon sired by anadromous or resident males, suggesting that non-genetic paternal effects have little influence on juvenile phenotypic variation (Fig. S3).

The effects of water flow treatment on fish growth also resulted in phenotypic differences for the

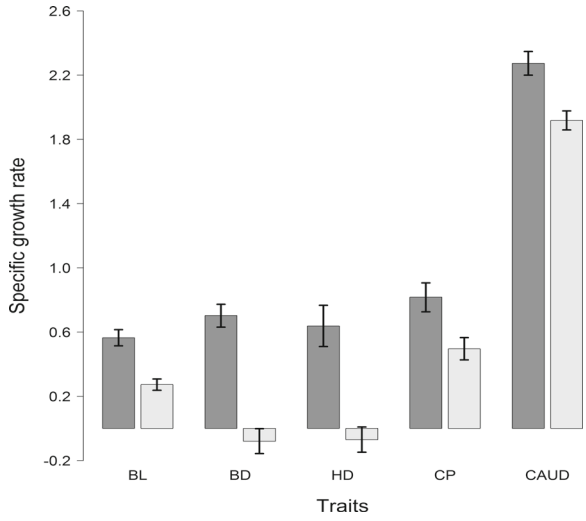

Fig. 1 Specific growth rates for the 5 traits measured on juvenile Atlantic salmon that were reared under rapid (dark bars) and slow (light bars) water flow treatments. Specific growth rates were calculated as $100 \times \left[\frac{ln(x_T)-ln(x_t)}{T-t}\right]$, where x_t and x_T are the mean trait values at emergence and at the end of the experiment, respectively. Error bars are 95 percentiles generated from a bootstrapping resampling procedure. BL is standard length, BD is body depth, HD is head depth, CP is the caudal peduncle depth and CAUD is the caudal peduncle length

measured traits (Fig. 2). Although we found greater mean values under the rapid water flow treatment for all traits (Table S2), there was considerable variation in the response across families, as depicted in Fig. 2 (B-F), indeed suggesting that part of this variation is genetically based.

Power and sensitivity of the pedigreee design

Based on the sample size obtained from the slow water treatment, our power analyses show that we were able to detect heritabilities as low as 0.09 at least 80 % of the time using animal models (Fig. 3). By contrast, for the rapid water flow treatment, such resolution is only achieved for heritability $\geq$ 0.27. We also show that different levels of maternal effects can have important effects on the capacity to estimate heritability particularly for small sample sizes (Fig. 3). However, a discussion about why this is the case is beyond the scope of this work.

Nevertheless, these results show that our sample sizes and the pedigree design are sufficient to detect small to moderate levels of additive genetic variation with little bias (see also Fig. S2). We show that even under the worse case scenario of a weak maternal effect in the slow water flow, animal models should detect heritabilities $\geq$ 0.13, at least 80 % of the time (see red lines in Fig. 3).

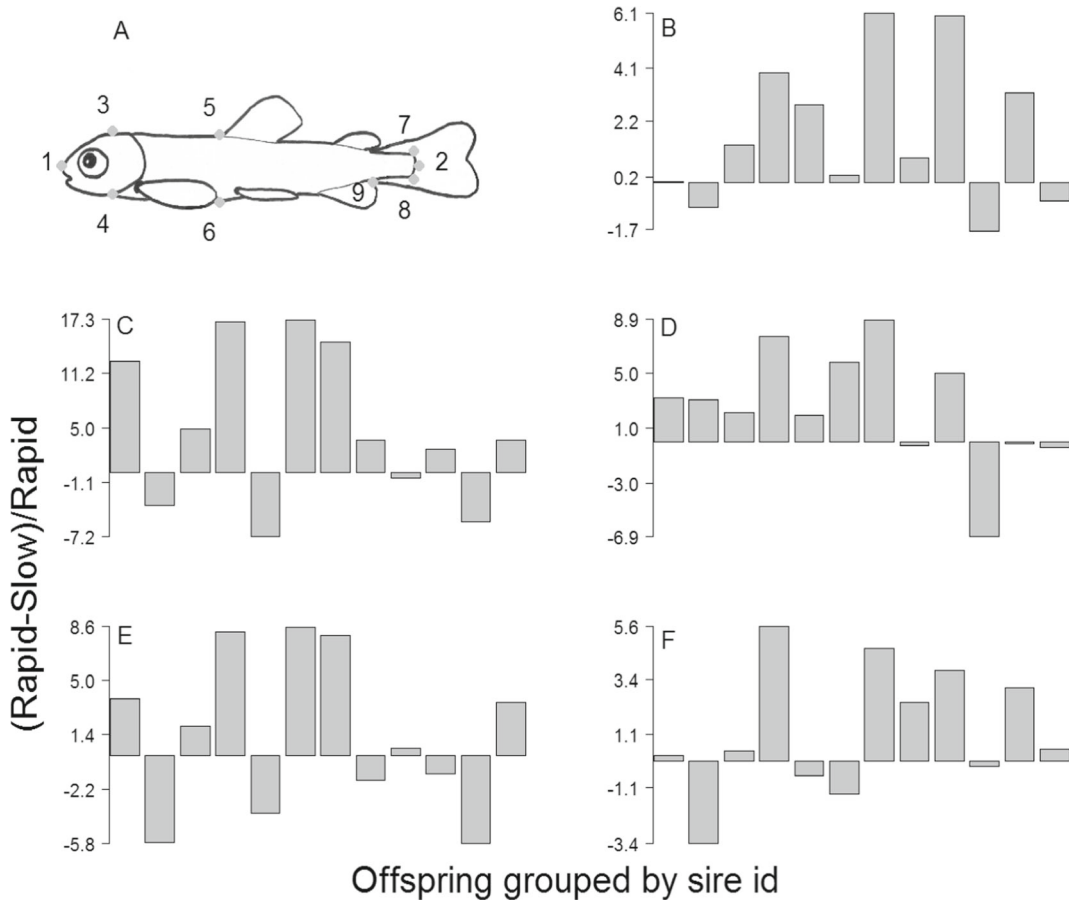

Fig. 2 Phenotypic differences between juvenile Atlantic salmon reared in rapid and slow water flow treatments. Figure **A** shows the measurements made on each individual. Points represent landmarks from which linear measurements were obtained: Body length (BL) = 1-2, head depth (HD) = 3-4, body depth (BD) = 5-6, caudal peduncle depth (CP) = 7-8, and caudal peduncle length (CAUD) = 9-2. All other figures show the percent difference in mean trait values across rapid and slow water flow conditions for all the offspring of each sire (i.e. across all dams). Panel **B** is body length, **C** is head depth, **D** is body depth, **E** is caudal peduncle depth and **F** is caudal peduncle length

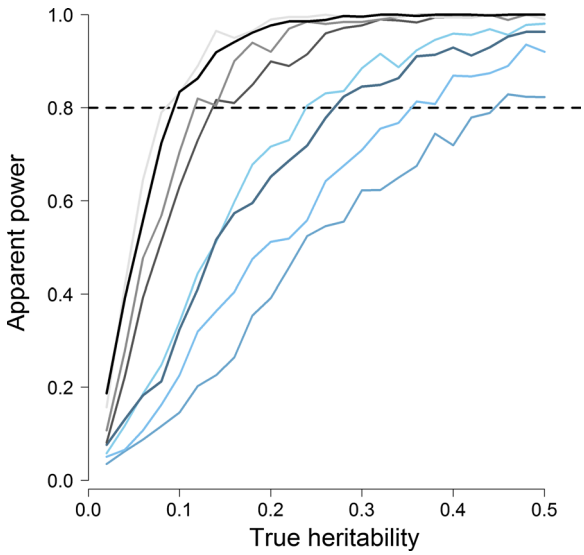

Fig. 3 Aparent power to detect heritability from the mating designs of the rapid (*slate blue lines*) and slow (*black-grey lines*) water flows. Line color gradients from dark to light, are different assumptions of maternal effects, with the darkest line assuming 0 maternal effects and the lightest line assuming maternal effects = 0.50 (Intermediate values = 0.15 and 0.30). Notice that our mixed effects "animal models" have greater power to detect heritability from the slow flow treatment. Therefore, in the case of no maternal effects (darkest red and blue lines), we can detect $h^2 = 0.09$ or greater more than 80 % of the time (dotted line) in the slow water flow, whereas this is achieved in the rapid water flow when $h^2 = 0.27$ or greater

Variance components for body size, morphology and survival

We found that levels of phenotypic variation in body size were similar across environments, based on phenotypic coefficients of variation which scale variation to mean trait values (Fig. 4). Yet, we found that additive genetic variation and the heritability of body size was detected with greater certainty in the rapid water flow treatment (Fig. 4, Table 2). Indeed we found that the heritability of body length in the rapid water flow had a point estimate of 0.55, whereas the corresponding point estimate in the slow water flow was close to 0. Furthermore, by using the "raw" measures for each trait, we found that heritability estimates were higher and estimated with greater certainty in the rapid water flow, with the exception being body depth (Fig. 4, Table S2). Interestingly, we found significant maternal effects in the slow water flow for body depth and head depth (Table S2).

By contrast, when removing the effect of body size to focus on morphological variation, our results showed inconsistent patterns of heritable trait variation across the water flow treatments (Fig. 4). For instance, while the size-corrected heritabilities of body depth and the caudal peduncle depth were detected with higher certainty in the slow water flow, the size-corrected heritability of head depth was higher in the rapid water flow. Furthermore, we did not find any maternal effects on the size-corrected traits (Table 2).

Water flow treatment had strong effects on individual survival, with approximately a three-fold survival increase in the slow water flow treatment (Table 2). The full-sib family mortality range in the rapid water flow varied from 48 to 100 %, whereas that in the slow water flow ranged from 0 to 78 %. However, we also found higher phenotypic variation in survival in the rapid water flow compared to the slow treatment. However, this difference was not explained by differences in heritability because our models did not detect additive genetic variance for this trait in either water flow treatment (Table 2). By contrast, we found that at least part of the variation of survival in the rapid water flow was explained by maternal effects (Fig. 4).

Effects of survival on body size

By comparing family-specific survival and mean body lengths, we found that an increase in survival results in decreased body length (Fig. 5). These results also suggested a significant quadratic effect whereby the decrease in body length with increase survival saturates when survival is approximately $\geq$ 60 %. The average coefficients of the bootstrapped model were $BL = 29.05 - 8.69 \times S + 6.12 \times S^2$, where S is survival.

Discussion

Our results show that environmental heterogeneity affects the causal components of phenotypic variation of body size, trait morphology and the survival of juvenile salmon. We provide among the first estimates of heritability of fish body size under different rearing habitats in semi-natural conditions (but see Garant et al. 2003). This is important because studies conducted under laboratory conditions or with

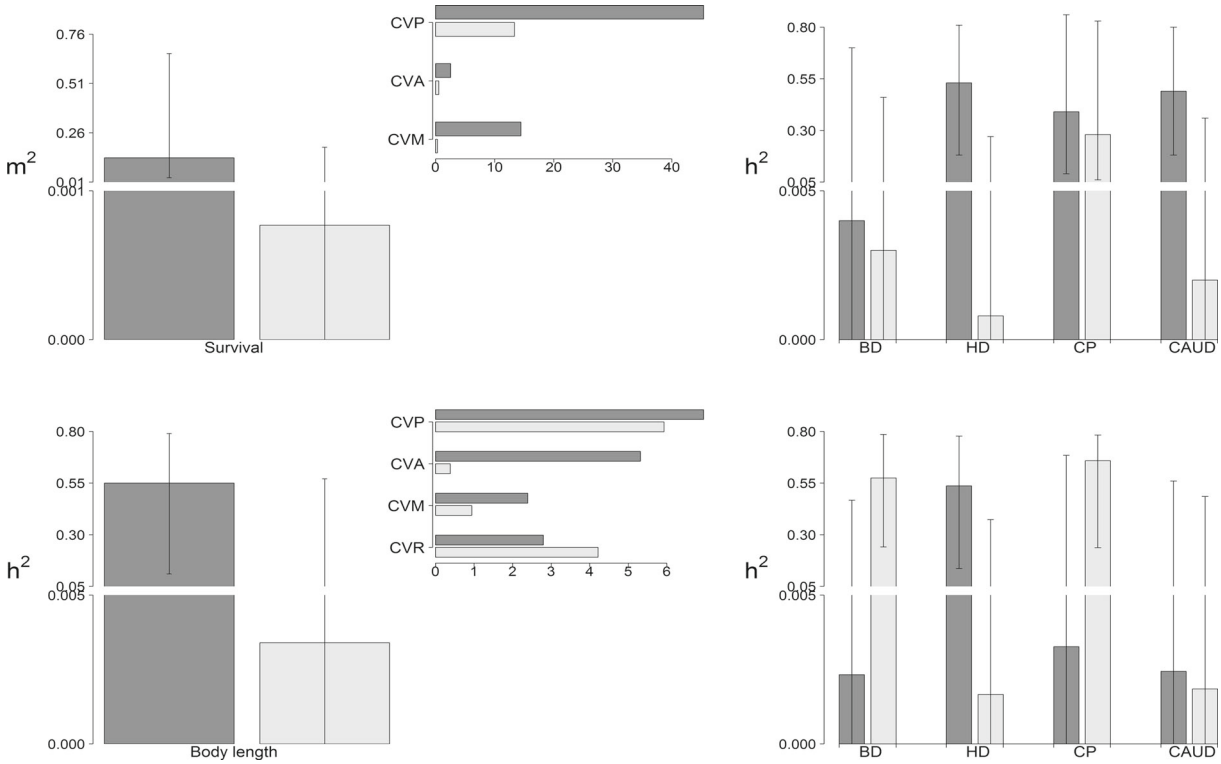

Fig. 4 Estimates of maternal effects of survival (*top left*), and the heritability of body length (*bottom left*) measured on juvenile Atlantic salmon reared in rapid (*dark grey*) and slow (*light grey*) water flows from the field experiment. The *horizontal bar graphs* are corresponding coefficients of variation of survival (*top*) and body length (*bottom*). Coefficients of variation (CV_X) were calculated as $V_X 0.5/\bar{X}$, where V_X is either the phenotypic (P), additive genetic (A), maternal (M), or residual (R) variation, and $\bar{X}$ is the mean estimate of the trait. *Top right* and *bottom right* panels show estimates of heritability for "raw" and size-corrected traits, respectively. All error bars are 95 % high posterior density credible intervals

selected lines of salmon often find moderate to high heritabilities for body size (Withler 1987; Páez et al. 2010). By contrast, we found that the heritability of body size was estimated with confidence only in the rapid water flow, suggesting that different rearing habitats could affect levels of genetic variability and therefore responses to selection. Our results thus provide support for the hypothesis that adaptive dynamics in nature depend on G × E interactions which are often neglected under more controlled settings.

When comparing trait heritabilities between the two water flow treatments we found overlapping credible intervals of the estimates. This made it difficult to conclude that heritabilities were higher in the rapid water flow compared to the slow water flow. However, analyses on the pedigree design suggested that we had sufficient statistical power to detect heritability values as small as 0.15 based on the sample size available in the slow water flow treatment. The large uncertainty in heritability estimates obtained in this water flow treatment may have thus occurred because of a lack of power to detect heritabilities smaller than 0.15 or because of a true lack of additive genetic variation in this treatment. By contrast, trait heritabilities in the rapid water flow were high for all traits except body depth and were estimated with narrower credible bounds.

The observed differences in the heritability of body size between environments agree with previous studies which show increased heritable variation under environmental conditions that are favorable for growth (Wilson et al. 2006; Charmantier and Garant 2005; Merilä and Sheldon 2001). Specifically, reduced growth in the slow water flow may have affected rates of genetic expression and development, leading to lower levels of additive genetic variation (Gebhardt-Henrich and Van Noordwijk 1991).

Table 2 Estimated variance components for traits measured in juvenile Atlantic salmon

	Trait					
A Rapid	Surv	BL	BD*	HD*	CP*	CAUD*
Mean	6.33	27.72	4.58	4.83	2.44	7.73
V_A	$0.02_{(0,4.16)}$	$2.17_{(0.22,3.25)}$	$0.04_{(0,6)}$	$16.2_{(3.6,26.1)}$	$0.02_{(0,3.30)}$	$0.03_{(0,6.02)}$
V_M	$0.83_{(0.01,13.49)}$	$0.44_{(0,4.13)}$	$0.07_{(0,4.7)}$	$0.32_{(0,23.9)}$	$0.09_{(0,7.2)}$	$0.05_{(0,3.39)}$
V_R	–	$0.6_{(0.19,1.55)}$	$4.59_{(1.5,5.7)}$	$10.11_{(5,16.7)}$	$1.66_{(0.20,2)}$	$5.58_{(2.46,6.97)}$
V_P	$8.25_{(5.79,21.49)}$	$3.71_{(2.67,7.83)}$	$9.35_{(6.98,16.19)}$	$29.91_{(20.27,50.28)}$	$4.28_{(2.81,10.8)}$	$7.76_{(5.96,12.4)}$
m^2	$0.13_{(0.03,0.66)}$	$0.002_{(0,0.55)}$	$0.002_{(0,0.33)}$	$0.001_{(0,0.48)}$	$0.004_{(0,0.69)}$	$0.001_{(0,0.29)}$
h^2	$0.007_{(0,1.28)}$	$0.55_{(0.11,0.79)}$	$0.002_{(0,0.47)}$	$0.54_{(0.14,0.78)}$	$0.003_{(0,0.69)}$	$0.002_{(0,0.56)}$
B Slow						
Mean	18	25.99	3.85	4.14	2.27	7.15
V_A	$0.01_{(0,1.40)}$	$0.01_{(0,1.71)}$	$15.72_{(8.70,24.2)}$	$0.01_{(0,2.7)}$	$2.92_{(1.20,3.7)}$	$0.03_{(0,4.55)}$
V_M	$0.003_{(0,1.60)}$	$0.060_{(0,2.71)}$	$0.64_{(0,21.8)}$	$0.03_{(0,2.4)}$	$0.05_{(0,2.9)}$	$0.03_{(0,2.25)}$
V_R	–	$1.20_{(0.38,1.37)}$	$3.56_{(1.8,9.2)}$	$4.11_{(2.8,4.7)}$	$0.53_{(0.2,1.4)}$	$5.25_{(3.29,6.39)}$
V_P	$5.77_{(5.27,8.56)}$	$2.37_{(1.73,4.85)}$	$27.04_{(21.05,47.43)}$	$6.03_{(4.79,8.88)}$	$4.17_{(3.29,7.22)}$	$7.21_{(6.28,10.8)}$
m^2	$0_{(0,0.19)}$	$0.03_{(0,0.56)}$	$0.002_{(0,0.45)}$	$0.002_{(0,0.29)}$	$0.001_{(0,0.42)}$	$0.002_{(0,0.22)}$
h^2	$0.003_{(0,0.75)}$	$0.003_{(0,0.57)}$	$0.58_{(0.24,0.79)}$	$0.002_{(0,0.37)}$	$0.66_{(0.24,0.78)}$	$0.002_{(0,0.49)}$
V_{CE}^1	$1.28_{(0.78,2.44)}$	$0.60_{(0.27,1.19)}$	$3.38_{(2,6.4)}$	$1.02_{(0.4,2.5)}$	$0.65_{(0.4,1.3)}$	$0.89_{(0.503,1.89)}$

Note: All variance components were estimated from mixed effects models. Subscript values in parentheses represent 95 % credible intervals around the estimate. V_A, V_M, V_P, V_{CE}, are, respectively the additive genetic, maternal, phenotypic and common environmental variances. m^2 is the ratio of the maternal variance to the total phenotypic variance. The traits Surv, BL, HD, BD, CP, and CAUD are, respectively, survival, body length, head depth, body depth, caudal peduncle depth, and caudal peduncle length (see, Fig. 2). * Variance components estimated after correcting for body size and for convenience, we multiplied variance components by 100. [1]Common environmental effects were channel specific and therefore not estimated separately in each treatment. Variance components for "raw" variables are presented in the Supplementary material (Table S2)

This result is further supported by the observation that phenotypic variation was partly explained by maternal effects for some traits measured in the slow water flow (i.e. head depth and caudal peduncle length). We suggest that under unfavorable growth conditions for early juvenile stages, maternal provisioning in the egg has long-lasting effects on the phenotypic expression of early life traits (see also Einum and Fleming 1999). Interestingly, under laboratory conditions which usually favor elevated growth, maternal effects were not detected in the measured traits for similarly aged fish (Páez et al. 2010). This suggests that, depending on environmental circumstances, developmental processes are able to draw from different sources of variation to shape phenotypic traits.

When focusing on morphological variation, we found that heritability estimates for the traits were no longer consistently higher in the rapid water flow. For example, we found that the heritability of size-corrected body depth and the caudal peduncle depth was higher in the slow water flow, but that the heritability of size-corrected head depth was higher in the faster water flow. Previous studies have found that juvenile salmonids are phenotypically plastic in response to variable water flow regimes (Godin and Rangeley 1989; Pakkasmaa and Piironen 2000). We show that at least part of the variation in morphology is due to additive genetic variation (see also McCairns and Bernatchez 2012), but that the amount of genetic variation depends on the rearing environment (McGuigan and Sgró 2009; Charmantier and Garant 2005; Sgró and Hoffmann 1998; Hoffmann and Merilä 1999). This result therefore has important implications for our understanding of the evolution of reaction norms and the adaptive potential of phenotypic plasticity in nature (Morrissey 2011). Morphological responses to selection, mediated by the

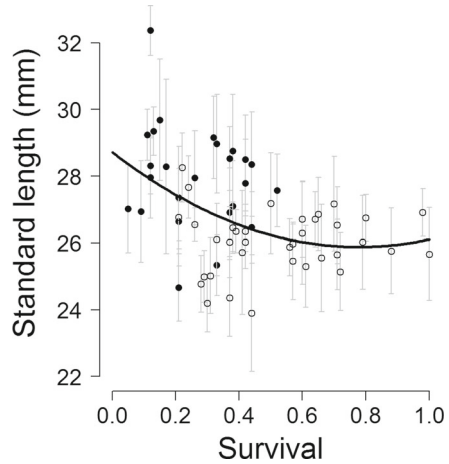

Fig. 5 Effects of differential juvenile Atlantic salmon mortality across rapid (*closed symbols*) and slow (*open symbols*) water flow treatments on standard body length. Each point is the mean trait value for one full-sib family with error bars that represent one standard deviation. Fitted lines were obtained from the mean bootstrapped coefficients from a linear mixed model. We found evidence of non-linearities in the response of body length to survival probability, so we modeled body length as a quadratic function of survival

environmental heterogeneity in different river habitats could also help explain the great variation in morphology observed in fishes and their natural habitats (Peres-Neto and Magnan 2004; Páez et al. 2008).

Lastly, previous studies conducted under controlled settings have found significant heritabilities in survival (Friars and Smith 2010). However, we could not detect additive genetic variation for this trait in either rearing treatment. Instead we found that the variation in survival was influenced by maternal effects only for fish reared in the rapid water flow. We also found that body size was negatively correlated with survival, such that the larger body sizes found in the rapid water flow were also associated with higher mortality rates in this treatment.

We hypothesize that the size differences between rearing habitats may be due to either size-selective mortality (Perez and Munch 2010; Good et al. 2001; Sogard 1997) or density dependence (Elliott 1989). Size-selective mortality favoring large individuals may have occurred from territorial disputes or competition for food occurring in the rapid water flow. However, we cannot rule out density dependent effects (Elliott 1989) because higher mortality may have also favored increased growth of survivors through an increase in per-capita food availability. Further studies are required to differentiate between size-selective mortality and density dependence as sources of juvenile mortality across different rearing habitats.

In conclusion, our results emphasize the importance of conducting quantitative genetic experiments under field conditions when studying the adaptive potential of organisms (e.g. Thériault et al. 2007; Mazé-Guilmo et al. 2014; Morrissey and Ferguson 2011). For example, our previous results obtained under laboratory conditions (Páez et al. 2010) failed to detect differences in heritabilities between individuals originating from upstream and downstream sites of this river system. However, juvenile phenotypic differences, particularly in body size, are readily apparent between sites in this river. Our new results suggest that additive genetic variation may be "hidden" or "revealed" depending on the environment and may thus vary across sites and habitats (McGuigan and Sgró 2009; Charmantier and Garant 2005; Sgró and Hoffmann 1998; Hoffmann and Merilä 1999). Therefore, while laboratory experiments provide crucial information about the causal components of trait variation and of the course of evolution under artificial selection, field studies are required to understand predictions of evolutionary responses in nature. Our results suggest that to understand adaptive processes, both natural selection and additive genetic variation have to be quantified within a context that is as close to natural conditions as logistically permitted (Lynch and Walsh 1998; Falconer and Mackay 1996).

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