



## Genetic variance and covariance for 0+ brook charr (*Salvelinus fontinalis*) weight and survival time of furunculosis (*Aeromonas salmonicida*) exposure

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### Abstract

Furunculosis, caused by the bacterium *Aeromonas salmonicida*, is a serious pathogen of salmonids including the brook charr (*Salvelinus fontinalis*). However, within-stock genetic variance for furunculosis resistance and its genetic association with other economically important traits are not known in brook charr. Our objective was a preliminary identification of the heritability of early furunculosis resistance and body weight (WT) in a commercially important Québec strain of brook charr. Survival was expressed quantitatively as survival time (ST; hours) for analysis. Twenty-three half and full sib families of 0+ brook charr in a mixed pedigree were exposed to a concentration of  $2.86 \times 10^5 \text{ l}^{-1}$  infective particles. Heritability estimates within this strain were high for both ST ( $h_{a,ST}^2 = 0.51 \pm 0.03$ ) and WT ( $h_{a,WT}^2 = 0.57 \pm 0.04$ ). Low positive genetic correlation between ST and WT ( $r_a = 0.15 \pm 0.06$ ) was supported by phenotypic correlation ( $r_p = 0.12$ ). Independent or simultaneous pedigreed selection for these traits within this strain might produce significant genetic gain, although indirect selection for tolerance of furunculosis via genetic correlation with body weight is not likely to be successful.

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## 1. Introduction

The relatively high proportion of extant genetic variation for morphological (production) characters, their direct connection to economic value and ease of measurement have resulted in numerous studies of the genetic control of such traits (i.e. Argue et al., 2002; Fishback et al., 2002; Kühn et al., 2003; Pieramati et al., 2003). Quantitative genetic variation is typically high in salmonid fish (Osteichthyes: Salmonidae) for production traits such as weight-at-age and growth rate (Wangila and Dick, 1996; Bonnet et al., 1999; Fishback et al., 2002; Perry et al., submitted for publication). With sufficient correlation between early and late production traits at different points in ontogeny (see Fishback et al., 2002; Su et al., 2002), juvenile selection on production characters could result in gains via the elimination of costs associated with rearing of sub-optimal individuals.

However, the incorporation of additional traits such as disease resistance in selective indices may also provide additional economic returns (see Henryon et al., 2002) by mitigating losses from pathogens or by reducing disease incidence. Genetic improvement of such indirectly valuable traits may also help ameliorate environmental and legal issues potentially arising from widespread vaccination or post-infective therapeutic treatment (Kerry et al., 1994; Hastings, 1997; Markestad and Grave, 1997; van Muiswinkel et al., 1999; Stear et al., 2001; see also Cipriano et al., 2002). The potential utility of improvement for resistance characters, however, may be limited depending on genetic correlation with production traits (Roff, 1997; Stear et al., 2001), possibly requiring the use of more complex selective indices or alternate approaches such as sequential selection (Gibson and Smith, 1989; Cameron, 1997). Alternatively, advantageous genetic correlations between disease resistance and production traits would obviate the need for expensive, time-consuming and complex progeny testing for disease resistance (see Røed et al., 2002). This issue has been approached from the perspective of the establishment of disease-resistant salmonid strains (Ehlinger, 1964, 1977; Cipriano et al., 2002) but within-population examination of heritability and breeding value of specific sires and dams might also be useful where whole-stock replacement is less desirable; i.e. where previous genetic gain in production traits for already established strains has occurred. Various work indicates heritable variance for resistance to pathogens within salmonid populations (see Chevassus and Dorson, 1990; Rye et al., 1990; Fjalestad et al., 1993; Gjedrem, 2000; Henryon et al., 2002).

Brook charr (*Salvelinius fontinalis*) form a significant component (>50%) of the total value of the Québec aquacultural industry but there has been little or no examination of genetic variation for traits directly and indirectly related to production value in this species despite its high, stable growth and good fertilization rate for this genera (Dumas et al., 1995). Brook charr are particularly susceptible to furunculosis, caused by the Gram-negative bacterium *Aeromonas salmonicida*, manifested as a pattern of serious external lesions (Beaulieu et al., 1990; Cipriano et al., 2002). Our objectives were (i) to estimate the level of genetic control of furunculosis resistance and early body weight in an aquacultural brook trout strain and (ii) to investigate genetic correlation between these characters in this strain.

## 2. Materials and methods

A total of 23 half sib and full sib families were bred using 15 sires and 10 dams (i.e. a partial factorial setup) from a population used in commercial production and descended from wild fish from the Rupert River, Québec, Canada at the Laboratoire Regionale de Sciences Aquatiques (LARSA; Université Laval) on November 30 and December 13, 2001 (Table 1). Fish derived from the Rupert River (Québec, PQ) and associated waterways (i.e. the Assinica) have been noted for particularly rapid growth and large final size (Sutton et al., 2002) suggesting utility for aquacultural use. Fish were reared by full sib family in LARSA under external photoperiod conditions at 8 °C (to eliminate the possibility of accidental occurrence of *A. salmonicida* infections) and were fed at 5.3% feed by body weight below 0.5 g average weight and at 4.5% body weight thereafter marking and transport to the test facility (October 2002). Fish were marked by fin-clips at one of the paired fins (pectoral or pelvic) or the anal fin and divided into two approximately equal replicates of three to five families per tank, 10 tanks total (Table 1). Fish were then immediately transported to the fish pathogen exposure room at the Département de microbiologie et immunologie, Université de Montréal (Montréal, QC) and acclimated at 10 °C for 2–3 days. We used a rapid bacterial infection and morbidity procedure similar to

Table 1

Rupert strain brook trout pedigree, date of fertilisation and number of individuals used to test furunculosis resistance

Fertilisation date	Female	Male	Family	<i>n</i>	Age	
November 30, 2001	126	196	1	144	318/311	
	126	100	2	56	318/311	
	126	162	3	60	318/311	
	193	162	4	59	318/311	
	193	100	5	27	311	
	193	138	6	28	311	
	252	88	7	46	318/311	
	252	249	8	40	318/311	
	252	196	9	22	311	
	217	84	11	60	318/311	
	217	65	12	48	318/311	
	217	250	13	55	318/311	
	242	84	15	53	318/311	
	December 13, 2001	251	177	16	58	305/298
		231	114	17	59	305/298
231		51	18	30	298	
231		247	19	50	305/298	
38		247	20	59	305/298	
38		148	21	53	305/298	
38		177	22	57	305/298	
94		148	24	58	305/298	
135		177	26	49	305/298	
135		235	27	59	305/298	

Age refers to the age at which families were transferred to the Université de Montreal for furunculosis resistance testing.

that described by Lutwyche et al. (1995). Fish were given a small caudal incision to facilitate infection and then were then immersed separately in an *A. salmonicida* suspension ( $2.86 \times 10^5 \text{ l}^{-1}$ ) for 15 min at full aeration. After infection, fishes were immediately stressed by raising the water temperature to 18 °C over 4 h and by decreasing the dissolved oxygen level to 50%. All fish in the first replicate (five families per replicate tank) were exposed on October 7, 2002 with the second replicate initially used as a negative control (uninfected) to assess the survival of fish immediately after the caudal incision and stress exposure. There was no mortality in the controls during this period. The latter in turn were then brought back to 10 °C and 100% oxygen for 2–3 and then processed as for the first experimental group (infection on October 15, 2002). For both infection experiments, exposed fish were checked every 4 h for mortalities until 96 h, which were removed on observation and their survival time (ST; h) and weight (WT; g) recorded. All remaining fish were removed from the experiment at 96-h post-infection and all estimates of survival in remaining individuals censored to that point. Individuals unidentifiable at the end of the experiment (i.e. due to fin erosion) were not included in analysis, leaving an average of 54 fish per full sib family ( $n = 1230$ ; Table 1). Tests of normality indicated that both ST and WT were non-normal in distribution ( $P < 0.05^*$ ; PROC UNIVARIATE; SAS, 1998). Appropriate data transformations were determined for both traits using a SAS (1998) BoxCox macro (M. Friendly, York University, ON, Canada). All analysis was performed using transformed data.

The pedigree of the experimental families (up to the parental generation, as relationships above the level of the parents of the experimental families were not known) and the transformed data values were recoded using the program Parameter Estimation Software 3.0 (PEST; Groeneveld et al., 1990). Restricted maximum likelihood (REML) analysis with this data and the pedigree was used in Variance Component Estimator 4.1 (VCE4.1; Groeneveld, 1994) to provide genetic (co)variance estimates and estimated breeding values (EBV) using an animal model

$$y_i = \mathbf{X}_i \mathbf{b}_i + \mathbf{Z}_i \mathbf{a}_i + e_i$$

or in matrix notation

$$\mathbf{y} = \mathbf{X}\mathbf{b} + \mathbf{Z}\mathbf{a} + \mathbf{e}$$

where  $y_i$  is the phenotypic vector for trait  $i$  (ST and FR),  $\mathbf{X}_i$  is the incidence matrix for trait  $i$ ,  $\mathbf{b}_i$  is the fixed effects incidence vector,  $\mathbf{Z}_i$  is the incidence matrix of random effects (including animal effects),  $\mathbf{a}_i$  is the vector of random animal effects (i.e. breeding values) for trait  $i$  and  $e_i$  represents random error for trait  $i$ . Both traits were fit simultaneously and estimates of animal effects and genetic variances for the two traits were made using Gauss-Seidel iteration via analytical gradients (Mrode, 1996). Although we initially considered several fixed effect vectors including fertilization date, experiment day (start date of pathogen exposure), early rearing tank (i.e. full sib family) and experimental tank, ultimately only early rearing tank (full sib family) and experimental tank were used in the final animal model. Minor age differences between some of the families were not included in analysis since age differences between fish at the point of experimentation were less than 20 days total (see Table 1). Any age-related effects therefore fell into either full sib

family effects and/or experimental tank. Experiment day was similarly not included since tanks were nested within experiment day and the inclusion of experimental tank would have likely resulted in over-parameterisation. This position was supported by Akaike's Information Criterion (AIC) ratios using PROC MIXED in SAS (1998), which indicated a slight superiority of the model including only early rearing tank (full sib family) and experimental tank effects (data not shown).

### 3. Results

Data and/or pedigree information was available from a total of 1255 fish including 1230 progeny and 25 parents (15 sires and 10 dams). All fish with missing records were eliminated. Optimal transformations (minimizing root mean squared error) were determined as  $WT^{0.1}$  and  $ST^{1.4}$ . Mean values for ST and WT were  $75.5 \pm 0.32$  h and  $1.97 \pm 0.031$  g in original format,  $426 \pm 88.9$  h and  $1.07 \pm 0.0514$  g after transformation. Mean weight by full sib family ranged from 1.0 to 1.16  $g^{0.1}$  (Table 2). Mean survivorship per full sib family ranged from 352 to 492  $h^{1.4}$  (censored at 96 h). Estimates of narrow-sense heritability for both characters were high ( $>0.4$ ; see Roff, 1997). Narrow-sense heritability for transformed ST was  $0.51 \pm 0.03$  and our estimate of heritability for WT was also very high ( $0.57 \pm 0.04$ ). Genetic correlation between WT and ST was low but

Table 2  
Mean body weight (WT;  $g^{0.1}$ ) and survival time (ST;  $h^{1.4}$ ) by family for furunculosis-exposed Rupert strain brook trout

Family	Mean WT	Mean ST
1	$1.06 \pm 0.00211$	$405 \pm 6.89$
2	$1.04 \pm 0.00415$	$380 \pm 11.0$
3	$1.10 \pm 0.00363$	$425 \pm 10.7$
4	$1.08 \pm 0.00335$	$397 \pm 8.03$
5	$1.01 \pm 0.00574$	$357 \pm 14.0$
6	$1.02 \pm 0.00500$	$352 \pm 8.60$
7	$1.07 \pm 0.00372$	$454 \pm 14.2$
8	$1.06 \pm 0.00423$	$450 \pm 13.5$
9	$1.05 \pm 0.00554$	$391 \pm 14.6$
11	$1.14 \pm 0.00433$	$478 \pm 11.9$
12	$1.16 \pm 0.00462$	$484 \pm 10.4$
13	$1.11 \pm 0.00404$	$463 \pm 11.1$
15	$1.10 \pm 0.00359$	$426 \pm 11.2$
16	$1.14 \pm 0.00285$	$428 \pm 10.9$
17	$1.05 \pm 0.00345$	$433 \pm 9.56$
18	$1.08 \pm 0.00311$	$405 \pm 10.6$
19	$1.13 \pm 0.00377$	$492 \pm 11.7$
20	$1.10 \pm 0.00282$	$483 \pm 12.2$
21	$1.01 \pm 0.00294$	$402 \pm 13.4$
22	$1.01 \pm 0.00441$	$412 \pm 11.5$
24	$1.05 \pm 0.00315$	$427 \pm 11.2$
26	$1.00 \pm 0.00372$	$384 \pm 11.7$
27	$1.09 \pm 0.00338$	$430 \pm 11.4$

positive ( $0.15 \pm 0.06$ ). The estimate of genetic correlation between ST and WT on the quantitative scale was reflected by phenotypic correlation between these traits ( $r_p = 0.12$ ;  $\beta = 601 \pm 46.3$ ).

#### 4. Discussion

Previous characterizations of phenotype in brook charr have characterized variance from the perspective of phenotypic plasticity (Schlichting and Pigliucci, 1996) rather than genetic variation (Imre et al., 2001, 2002; Pakkasmaa and Piironen, 2001). Results from our modest genetic design suggest high heritability ( $h_a^2 > 0.4$ ) for early (0+ year) body weight and furunculosis resistance in the Rupert strain but should be considered somewhat preliminary owing to the small numbers of parents sampled from the inference population. Our estimate of genetic variances for the two characters falls within the higher end of the range of observed estimates for additive genetic variance for production (Fishback et al., 2002; Henryon et al., 2002; Perry et al., submitted for publication) and disease resistance traits in other salmonids (Withler and Evelyn, 1990; Gjedrem et al., 1991; Yamamoto et al., 1991; Beacham and Evelyn, 1992a, b; Dorson et al., 1995; Hard et al., 1997) (see also Na Nakorn et al., 1994; Wiegertjes et al., 1996 for results from non-salmonid fish) but may have partially resulted from non-additive genetic or non-genetic effects despite the explicit inclusion of model terms to account for such sources of variance (full sib family and experimental tank), or from the relatively small sampling size. Early selection on disease resistance and/or body weight might be useful for genetic improvement assuming sufficient genetic correlation between early and late trait measurements, which appears to be moderate-high for production characters in other salmonid populations (Fishback et al., 2002; Su et al., 2002). Such correlations have not been measured in brook charr, however, nor has the ontogenetic persistence of genetic value for disease resistance been evaluated in salmonid fish. If high-moderate genetic variance within strains is relatively common in this species, selective improvement may be of use to individual aquacultural operations where (i) the genetic constitution of a given stock is already amenable to specific production goals due to previous investment in selection or innate advantages in economic traits, (ii) where economic recourse to other strains is unavailable, or (iii) where the risks associated with introduction of novel brook charr strains outweigh potential advantages.

Indirect selection for disease resistance could eliminate the difficulty and expense of disease testing (see Stear et al., 2001; Røed et al., 2002) providing that the traits used for direct selection have high positive genetic correlation with disease resistance (i.e. where  $r_{1,2} \sqrt{h_y^2} > \sqrt{h_x^2}$ ; see Falconer and Mackay, 1996) and where the economic risk of disease outbreak exceeds the costs of progeny testing or where genetic weights for disease resistance are higher than those for weight (see Gibson and Smith, 1989). However, our results indicated low positive genetic correlation ( $r_a < 0.2$ ) at best between early weight and furunculosis resistance in the Rupert strain. Although these results may be interpreted as somewhat preliminary, they were supported by our estimate of phenotypic correlation between these traits in our families and to varying degrees in other brook charr strains (Hayford and Embody, 1930; Cipriano et al., 2002). Phenotypic correlation appears to be generally indicative of genetic correlation for pairs of morphological (production) traits

(Cheverud, 1988; Mousseau and Roff, 1987; Roff and Mousseau, 1987; Koots and Gibson, 1994, 1996; Roff, 1997) but there seems to be little known about its predictive value for genetic correlation between production and physiological traits (see Roff, 1997).

Where the potential for genetic gain via multi-trait selective approaches incorporating multiple characters with direct and indirect relationships with economic value exists (Henryon et al., 2002), an enlargement of the phenotypic architecture, developmental periods and rearing environments considered in analyses of genetic variance should permit the identification of useful temporal and environmental frames for optimal genetic gain and also allow investigations of genetic covariance between traits early and late in development (see also Fishback et al., 2002). The application of a more complete understanding of phenotypic–genetic relationships to selection indices in aquacultural production may permit substantial gains over even a relatively short term.

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