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

Guy M.L. Perry[a,*], Phillipe Tarte[b], Sebastian Croisetière[b], Pierre Belhumeur[b], Louis Bernatchez[a]

[a] *Département de biologie, Université Laval, Cité Université, Pavillion C.-E. Marchand, Ste-Foy, QC, Canada G1K 7P4*

[b] *Département de microbiologie et immunologie, Université de Montréal, C.P. 6128, succursale Centre-ville Montréal, QC, Canada H3C 3J7*

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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 2.86×10^5 l^{-1} infective particles. Heritability estimates within this strain were high for both ST ($h^2_{a,ST} = 0.51 \pm 0.03$) and WT ($h^2_{a,WT} = 0.57 \pm 0.04$). 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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Keywords: *Salvelinus fontinalis*; Brook charr; Heritability; Furunculosis; *Aeromonas salmonicida*; Body weight; Genetic correlation

* Corresponding author. Tel.: +1-418-656-2131x8455; fax: +1-418-656-7176.
E-mail address: guy.perry@giroq.ulaval.ca (G.M.L. Perry).

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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 (Osteicthyes: 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 establishement 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. salmonicia* 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	*n*	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 (2.86×10^5 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

$$y_i = \mathbf{X}_i b_i + \mathbf{Z}_i a_i + e_i$$

or in matrix notation

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Za} + \mathbf{e}$$

where y_i is the phenotypic vector for trait i (ST and FR), $\mathbf{X}_i$ is the incidence matrix for trait i, b_i is the fixed effects incidence vector, $\mathbf{Z}_i$ is the incidence matrix of random effects (including animal effects), 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 ± 0.32 h and 1.97 ± 0.031 g in original format, 426 ± 88.9 h and 1.07 ± 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 ± 0.03 and our estimate of heritability for WT was also very high (0.57 ± 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 ± 0.00211	405 ± 6.89
2	1.04 ± 0.00415	380 ± 11.0
3	1.10 ± 0.00363	425 ± 10.7
4	1.08 ± 0.00335	397 ± 8.03
5	1.01 ± 0.00574	357 ± 14.0
6	1.02 ± 0.00500	352 ± 8.60
7	1.07 ± 0.00372	454 ± 14.2
8	1.06 ± 0.00423	450 ± 13.5
9	1.05 ± 0.00554	391 ± 14.6
11	1.14 ± 0.00433	478 ± 11.9
12	1.16 ± 0.00462	484 ± 10.4
13	1.11 ± 0.00404	463 ± 11.1
15	1.10 ± 0.00359	426 ± 11.2
16	1.14 ± 0.00285	428 ± 10.9
17	1.05 ± 0.00345	433 ± 9.56
18	1.08 ± 0.00311	405 ± 10.6
19	1.13 ± 0.00377	492 ± 11.7
20	1.10 ± 0.00282	483 ± 12.2
21	1.01 ± 0.00294	402 ± 13.4
22	1.01 ± 0.00441	412 ± 11.5
24	1.05 ± 0.00315	427 ± 11.2
26	1.00 ± 0.00372	384 ± 11.7
27	1.09 ± 0.00338	430 ± 11.4

positive (0.15 ± 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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References

Argue, B.J., Arce, S.M., Lotz, J.M., Moss, S.M., 2002. Selective breeding of Pacific white shrimp (*Litopenaeus vannamei*) for growth and resistance to Taura Syndrome Virus. Aquaculture 204, 447–460.

Beacham, T.D., Evelyn, T.P.T., 1992a. Population and genetic variation in resistance of chinook salmon to vibriosis, furunculosis, and bacterial kidney disease. J. Aquat. Anim. Health 4, 153–167.

Beacham, T.D., Evelyn, T.P.T., 1992b. Genetic variation in disease resistance and growth and growth of chinook, coho, and chum salmon with respect to vibriosis, furunculosis, and bacterial kidney disease. Trans. Am. Fish. Soc. 121, 456–485.

Beaulieu, M., Fréchette, J.L., Gonthier, S., Roy, M., Turgeon, Y., 1990. Principales maladies des poissons au Québec. Ministère du Loisir, de la Chasse et de la Pêche: Direction de la gestion des espèces et des habitats, Québec, Gouvernement du Québec, Canada. 46 pp.

Bonnet, S., Haffray, P., Blanc, J.M., Valleé, F., Vauchez, C., Fauré, A., Fauconneau, B., 1999. Genetic variation in growth parameters until commercial size in diploid and triploid freshwater rainbow trout (*Oncorhynchus mykiss*) and seawater brown trout (*Salmo trutta*). Aquaculture 175, 359–375.

Cameron, N.D., 1997. Selection Indices and Prediction of Genetic Merit in Animal Breeding. CAB International, Wallingford, UK. 203 pp.

Chevassus, B., Dorson, M., 1990. Genetics of resistance to diseases in fish. Aquaculture 85, 83–107.

Cheverud, J.M., 1988. A comparison of genetic and phenotypic correlations. Evolution 42, 958–968.

Cipriano, R.C., Marchant, D., Jones, T.E., Schachte, J.H., 2002. Practical applications of disease resistance: a brook trout fishery selected for resistance to furunculosis. Aquaculture 206, 1–17.

Dorson, M., Quillet, E., Hollebecq, M.G., Torhy, C., Chevassus, B., 1995. Selection of rainbow trout resistant to viral haemorrhagic septicaemia virus and transmission of resistance by gynogenesis. Vet. Res. 26, 361–388.

Dumas, S., Blanc, J.M., Audet, C., de la Noüe, J., 1995. Variation in yolk absorption and early growth of brook charr *Salvelinus fontinalis* (Mitchill), Arctic charr, *Salvelinus alpinus* (L.), and their hybrids. Aquac. Res. 26, 759–764.

Ehlinger, N.F., 1964. Selective breeding of trout for resistance to furunculosis. N. Y. Fish Game J. 11, 78–90.

Ehlinger, N.F., 1977. Selective breeding of trout for resistance to furunculosis. N. Y. Fish Game J. 24, 25–36.

Falconer, D.S., Mackay, T.F.C., 1996. Introduction to Quantitative Genetics, 4th ed. Addison Wesley Longeman, Essex, UK. 464 pp.

Fishback, A.G., Danzmann, R.G., Ferguson, M.M., Gibson, J.P., 2002. Estimates of genetic parameters and genotype by environment interactions for growth traits of rainbow trout (*Oncorhynchus mykiss*) as inferred using molecular pedigrees. Aquaculture 206, 137–150.

Fjalestad, K.T., Gjedrem, T., Gjerde, B., 1993. Genetic improvement of disease resistance in fish: an overview. Aquaculture 111, 65–74.

Gibson, J.P., Smith, C., 1989. The incorporation of biotechnologies into animal breeding strategies. In: Babiuk, L., Phillips, J.P. (Eds.), Comprehensive Biotechnology. Pergamon, Oxford, pp. 203–231.

Gjedrem, T., 2000. Genetic improvement of cold-water fish species. Aquac. Res. 31, 25–33.

Gjedrem, T., Salte, R., Gjoen, H.M., 1991. Genetic variation in susceptibility of Atlantic salmon to furunculosis. Aquaculture 97, 1–6.

Groeneveld, E., 1994. A reparameterization to improve numerical optimization in multivariate REML (co)variance component estimation. Genet. Sel. Evol. 26, 537–545.

Groeneveld, E., Kovac, M., Wang, T., 1990. PEST, a general purpose BLUP package for multivariate prediction and estimation. Proceedings of the 4th World Congress on Genetics Applied to Livestock Production, pp. 488–491. Edinburgh, UK, Garsi, Madrid.

Hard, J.J., Connell, L., Hershberger, W.K., Harrell, L.W., 1997. Genetic variation in mortality of chinook salmon during a bloom of the marine alga *Heterosigma akashiwo*. J. Fish Biol. 56, 1387–1397.

Hastings, T.S., 1997. Chemotherapy of furunculosis. In: Bernoth, E.M., Ellis, A.E., Midtlying, P.J., Olivier, G., Smith, P. (Eds.), Furunculosis: Multidisciplinary Fish Disease Research. Academic Press, San Diego, pp. 423–432. 529 pp.

Hayford, C.O., Embody, G.C., 1930. Further progress in the selective breeding of brook trout at the New Jersey State Hatchery. Trans. Am. Fish Soc. 60, 109–113.

Henryon, M., Jokumsen, A., Berg, P., Lund, I., Pedersen, P.B., Olesen, N.J., Slierendrecht, W.J., 2002. Genetic variation for growth rate, feed conversion efficiency, and disease resistance exists within a farmed population of rainbow trout. Aquaculture 209, 59–76.

Imre, I., McLaughlin, R.L., Noakes, D.L.G., 2001. Temporal persistence of resource polymorphism in brook charr, *Salvelinus fontinalis*. Environ. Biol. Fisches 60, 393–399.

Imre, I., McLaughlin, R.L., Noakes, D.L.G., 2002. Phenotypic plasticity in brook charr: changes in caudal fin induced by water flow. J. Fish Biol. 61, 1171–1181.

Kerry, J., Hiney, M., Coyne, R., Cazabon, D., NicGabhainn, S., Smith, P., 1994. Frequency and distribution of resistance to oxytetracycline in microorganisms isolated from marine fish farm sediments following therapeutic use of oxytetracycline. Aquaculture 123, 43–54.

Koots, K.R., Gibson, J.P., 1994. How precise are genetic correlation estimates? Proc. 5th World Congr. Gen. Appl. Livest. Prod. 18, 353–356.

Koots, K.R., Gibson, J.P., 1996. Realized sampling variances of estimates of genetic parameters and the difference between genetic and phenotypic correlations. Genetics 143, 1409–1416.

Kühn, C., Bennewitz, J., Reinsch, N., Xu, N., Thomsen, H., Looft, C., Brockmann, G.A., Schwerin, M., Weimann, C., Hiendleder, S., Erhardt, G., Medjugorac, I., Förster, M., Brenig, B., Reinhardt, F., Reents, R., Russ, I., Averdunk, G., Blümel, J., Kalm, E., 2003. Quantitative trait loci mapping of functional traits in the German Holstein cattle population. J. Dairy Sci. 86, 360–368.

Lutwyche, P., Exner, M.M., Hancock, R.E., Trust, T.J., 1995. A conserved *Aeromonas salmonicida* porin provides protective immunity to rainbow trout. Infect. Immun. 63, 3137–3142.

Markestad, A., Grave, K., 1997. Reduction of antibacterial drug use in Norwegian fish farming due to vaccination. In: Gudding, R., Lillehaug, A., Midtlyng, P.J., Brown, F. (Eds.), Fish Vaccinology. Dev. Biol. Stand. Karger, Basel, pp. 201–209.

Mousseau, T.A., Roff, D.A., 1987. Natural selection and the heritability of fitness components. Heredity 59, 181–198.

Mrode, R.A., 1996. Linear Models for the Prediction of Animal Breeding Values. CAB International. Wilts, UK. 187 pp.

Na Nakorn, U., Chantsawang, S., Tarnchalanukit, W., 1994. Response to mass selection for disease resistance in walking catfish, *Clarias macrocephalus*. J. Appl. Aquac. 4, 65–73.

Pakkasmaa, S., Piironen, J., 2001. Water velocity shapes juvenile salmonids. Evol. Ecol. 14, 721–730.

Perry, G.M.L., Martyniuk, C.M., Ferguson, M.M., Danzmann, R.G., submitted for publication. Heritability of upper thermal tolerance and growth-related traits in rainbow trout (*Oncorhynchus mykiss*). Aquaculture.

Pieramati, C., Pepe, M., Silvestrelli, M., Bolla, M., 2003. Heritability estimation of osteochondrosis dissecans in Maremmano horses. Livest. Prod. Sci. 79, 249–255.

Røed, K.H., Fevolden, S.E., Fjalestad, K.T., 2002. Disease resistance and immune characteristics in rainbow trout (*Oncorhynchus mykiss*) selected for lysozyme activity. Aquaculture 209, 91–101.

Roff, D.A., 1997. Evolutionary quantitative genetics. Chapman and Hall, International Thompson Publishing. Toronto, Canada. 493 pp.

Roff, D.A., Mousseau, T.A., 1987. Quantitative genetics and fitness: lessons from *Drosophila*. Heredity 58, 103–118.

Rye, M., Lillevik, K.M., Gjerde, B., 1990. Survival in early life of Atlantic salmon and rainbow trout: estimates of heritabilities and genetic correlations. Aquaculture 89, 209–216.

Schlichting, C.D., Pigliucci, M., 1996. Phenotypic Evolution: A Reaction Norm Perspective. Sinauer Associates, Sunderland, MA. 387 pp.

Statistical Analysis Software (SAS) Institute, 1998. SAS Version 8.1. SAS Institute, Cary, NC.

Stear, M.J., Bishop, S.C., Mallard, B.A., Raadsma, H., 2001. The sustainability, feasibility and desirability of breeding livestock for disease resistance. Res. Vet. Sci. 71, 1–7.

Su, G.-S., Liljedahl, L.-E., Gall, G.A.E., 2002. Genetic correlations between body weight at different ages and with reproductive traits in rainbow trout. Aquaculture 213, 85–94.

Sutton, T.M., Pangle, K.L., Greil, R.W., 2002. Hatchery performance of Nipigon, Assinica and Iron River strains of age-0 brook trout. N. Am. J. Aquac. 64, 188–194.

van Muiswinkel, W.B., Wiegertjes, G.F., Stet, R.J.M., 1999. The influence of environmental and genetic factors on the disease resistance of fish. Aquaculture 172, 103–110.

Wangila, B.C.C., Dick, T.A., 1996. Genetic effects and growth performance in pure and hybrid strains of rainbow trout, *Oncorhynchus mykiss* (Walbaum) (Order: Salmoniformes, Family: Salmonidae). Aquac. Res. 27, 35–41.

Wiegertjes, G.F., Stet, R.J.M., Parmentier, H.K., van Muiswinkel, W.B., 1996. Immunogenetics of disease resistance in fish: a comparative approach. Dev. Comp. Immunol. 20, 365–381.

Withler, R.E., Evelyn, P.T., 1990. Genetic variation in resistance to bacterial kidney disease within and between two strains of coho salmon from British Columbia. Trans. Am. Fish. Soc. 119, 1003–1009.

Yamamoto, S., Sanjyo, I., Sato, R., Kohara, M., Tahara, H., 1991. Estimation of the heritability for disease resistance to infectious haemopoietic necrosis in rainbow trout. Bull. Jpn. Soc. Sci. Fish. 57, 1519–1522.