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Heritability of life-history tactics and genetic correlation with body size in a natural population of brook charr (*Salvelinus fontinalis*)

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Abstract

A common dimorphism in life-history tactic in salmonids is the presence of an anadromous pathway involving a migration to sea followed by a freshwater reproduction, along with an entirely freshwater resident tactic. Although common, the genetic and environmental influence on the adoption of a particular life-history tactic has rarely been studied under natural conditions. Here, we used sibship-reconstruction based on microsatellite data and an 'animal model' approach to estimate the additive genetic basis of the life-history tactic adopted (anadromy vs. residency) in a natural population of brook charr, *Salvelinus fontinalis*. We also assess its genetic correlation with phenotypic correlated traits, body size and body shape. Significant heritability was observed for life-history tactic (varying from 0.52 to 0.56 depending on the pedigree scenario adopted) as well as for body size (from 0.44 to 0.50). There was also a significant genetic correlation between these two traits, whereby anadromous fish were genetically associated with bigger size at age 1 ($r_G = -0.52$ and -0.61). Our findings thus indicate that life-history tactics in this population have the potential to evolve in response to selection acting on the tactic itself or indirectly via selection on body size. This study is one of the very few to have successfully used sibship-reconstruction to estimate quantitative genetic parameters under wild conditions.

Introduction

Organisms that face environmental heterogeneity commonly adjust their phenotype in response to cues that give information about the current or future state of the environment (Roff & Bradford, 2000). Such phenotypic plasticity is shown in an extreme fashion by the existence of discrete morphs within a population, such as dimorphism in defensive structures (horned vs. hornless beetle, Moczek *et al.*, 2002), in feeding speciali zations (omnivorous and carnivorous morphs of toad tadpoles, Frankino & Pfennig, 2001), in life cycle (wing dimorphism in insects, Roff, 1994) or in mating tactics (satellite vs. territorial males in fish, Aubin-Horth & Dodson, 2004). Many of those discrete phenotypes generally involve environmentally cued threshold traits. Threshold traits are thought to be based on underlying characteristics that vary in a continuous way, called the 'liability', with a threshold of sensitivity (Roff, 1996). Individuals lying above the threshold develop into one morph whereas individuals below the threshold develop into the alternate morph (Hazel *et al.*, 1990; Falconer & Mackay, 1996; Roff, 1996). Characteristics such as the concentration of juvenile hormone, lipid storage or growth efficiency have been shown to affect the adoption of alternate morphs, and hypothesized as potential underlying traits (Roff *et al.*, 1997; Thorpe *et al.*, 1998; Forseth *et al.*, 1999; Emlen & Nijhout, 2000). Because it reflects and/or influences many other factors related to the adoption of alternate morphs, body size is the most commonly reported liability trait (Moczek *et al.*, 2002; Aubin-Horth & Dodson, 2004). The threshold trait framework, associated with the conditional strategy theory, relies on the basis of fitness trade-offs, where an individual expresses the phenotype

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that yields the higher fitness payoffs for its particular condition (i.e. depending on the value of its liability trait), even though it may have a lower overall average fitness (Gross, 1996).

The heritable basis of a given phenotypic trait, and its genetic correlation with other traits, must be established to predict its response to selection and thus its evolutionary potential (Falconer & Mackay, 1996; Kruuk, 2004). Threshold traits are likely to have a polygenic basis, and most polygenic traits bear significant levels of genetic variation (Roff, 1996). Indeed, many studies have reported a heritable basis for threshold traits, mainly through heritability of their liability traits (reviewed in Roff, 1996; see also Mousseau *et al.*, 1998; Ostrowski *et al.*, 2000; Garant *et al.*, 2003). Thresholds of sensitivity may also harbour genetic variation themselves, such that threshold values that signal the switch between alternative phenotypes also differ among genotypes (Hazel *et al.*, 1990; Hutchings & Myers, 1994; Emlen, 1996; Hazel *et al.*, 2004). However, most studies that have documented the quantitative genetics of threshold traits were performed in laboratory or controlled experiments (but see Garant *et al.*, 2003; Wilson *et al.*, 2003a). As there is accumulating evidence that heritability and genetic correlations of traits are influenced by their environments (reviewed in Charmantier & Garant, 2005 and in Sgrò & Hoffmann, 2004), direct measures of quantitative genetic parameters in nature are essential and may improve our understanding of how evolution takes place in the wild.

Following hatching in freshwater, many populations of salmonid fishes (trouts, salmons and charrs) may either remain in freshwater during their entire lives (residency tactic) or undertake a feeding migration to sea before returning to freshwater to spawn (anadromy tactic). The conditional strategy framework and threshold trait hypothesis have been employed to understand the evolutionary basis of this common dimorphism in salmonids (Hutchings & Myers, 1994; Gross, 1996; Thorpe *et al.*, 1998; Aubin-Horth & Dodson, 2004). Factors such as body size, growth rate, lipid reserves (Rowe & Thorpe, 1990; Rikardsen *et al.*, 2004) and growth efficiency (Forseth *et al.*, 1999; Morinville & Rasmussen, 2003) have all been suggested as potential underlying traits influencing the adoption of one form or the other. There may be a critical time when a particular threshold value that is genetically influenced and variable among individuals must be exceeded in order for reproduction to occur (Hutchings & Myers, 1994; Thorpe *et al.*, 1998). If this threshold is not exceeded, migration occurs and anadromy is expressed. Some studies have estimated the heritability of adopting a resident life history, by documenting the genetic basis of early sexual maturation, in controlled experiments for aquaculture purposes (Silverstein & Hershberger, 1992; Heath *et al.*, 1994; Wild *et al.*, 1994; Mousseau *et al.*, 1998). To our knowledge, however, no studies have attempted to estimate heritability of anadromy and residency in the wild.

The development of hypervariable genetic markers and analytical tools related to kinship and parentage, have eased the estimation of quantitative genetic parameters in nature (Garant & Kruuk, 2005). Namely, relationships among wild individuals, which are essential to build reliable pedigrees and estimate quantitative genetic parameters, can now be accurately established (Garant & Kruuk, 2005). Moreover, the so-called 'animal model' is being increasingly used in natural conditions for estimating quantitative genetic parameters (reviewed in Kruuk, 2004; see also Garant *et al.*, 2005; Charmantier *et al.*, 2006; Wilson *et al.*, 2006). However, very few studies have yet combined sibship-reconstruction based on genetic data and an 'animal model' approach to estimate quantitative genetic parameters in the wild.

Using this framework, our main objective is to elucidate the mechanisms influencing the expression of a dichotomous life history in a natural population of brook charr, *Salvelinus fontinalis*, which contains both anadromous and resident individuals living in sympatry. Recent studies have demonstrated frequent mating between anadromous and resident individuals in this population, mainly through resident males mating with anadromous females (Thériault *et al.*, 2007). Also, body size in this population has been shown to be correlated with the adoption of anadromy and residency (Thériault & Dodson, 2003). Growth efficiencies also differ between anadromous and resident individuals; before migration, future migrants exhibit lower growth efficiencies than future residents and higher associated metabolic costs (Morinville & Rasmussen, 2003). Also, future migrants inhabit habitats of faster water currents, and thus it is not clear if their higher metabolic costs reflect higher standard metabolic rate or higher swimming costs related to the exploitation of a more energetically costly habitat (Morinville & Rasmussen, 2006). Morphological analyses confirmed previous results on differential habitat use: migrants have a more streamlined morphology than resident fish, which is what is expected in faster current velocity habitat because such an elongated morphology incurs lower swimming costs (Boily & Magnan, 2002; Morinville & Rasmussen, 2007).

Altogether, these results raise the hypothesis that dimorphism in life-history tactics in this brook charr population represents a threshold trait with potential underlying characters related to energetic budget. Here, we aimed to estimate the heritability of this threshold trait (residency/anadromy) by means of pedigree reconstruction assisted by molecular markers and the 'animal model' approach. We also quantified heritability of morphological traits (body size and shape) and tested whether phenotypic correlations previously observed among these traits and the life-history tactics (Thériault & Dodson, 2003; Morinville & Rasmussen, 2006, 2007) translate into significant genetic correlations.

Methods

Study site and sampling

Fish were collected from the Morin Creek (average 5.6 m wide, 0.3 m deep, see Fig. S1), a tributary of the Sainte-Marguerite River, Quebec, Canada. An impassable waterfall (75 m high) is located 4 km upstream from the mouth of the tributary. Sampling was conducted in a 2.5 km section below this waterfall. Previous studies have shown that anadromous brook charr undergo upstream migration into this tributary for spawning, and that reproduction between anadromous and resident fish is common (Thériault *et al.*, 2007).

No obvious external expression of smoltification occurs in migrant brook charr (McCormick *et al.*, 1985 and V. Thériault) making it very difficult to differentiate a migrant from a resident until the moment of migration. We identified migrants as fish captured in trap nets during downstream migration; previous mark–recapture studies on the same system have shown that these fish were true migrants (Thériault & Dodson, 2003). Fish captured in streams following the migration period were defined as residents. Outstream migration of first-time migrants occurs between mid-May to mid-June and involves 1 and 2 year old fish in this system (Thériault & Dodson, 2003). Two cohorts were sampled: cohort 1 was composed of 1+ fish (designating individuals somewhat older than 1 year of age as they are hatched in early May) sampled in 2002 and 2+ fish in 2003, and cohort 2 was composed of 1+ sampled in 2003 and 2+ sampled in 2004. The trap nets were installed 1 km from the mouth of the stream and were operated from mid-May to mid-June for the three years of sampling. They were visited twice daily and the following morphological measures were taken in the field in 2002 and 2003 (see Morinville & Rasmussen, 2007, for detailed methodology): fork length (FL), standard length (SL), body depth (DEP), maximum body width (WID), peduncle depth (PED), caudal fin height (CAUD), pectoral fin length (PECT) and pelvic fin length (PELV). The adipose fin was also clipped and preserved in 95% ethanol for subsequent genetic analyses, and all fish were released. Resident juveniles were captured using a backpack electro-fisher following the migration period beginning mid-June for the three years of sampling. Tissue sampling and body measurements of resident fish followed the same protocols as described for migrant juveniles. Fish were classified as either 1+ or 2+ based on the frequency distribution of body lengths, previously validated with age determination using otoliths (Thériault & Dodson, 2003).

Pedigree reconstruction

Individuals of age 1+ and 2+ were partitioned into groups of putative full siblings using PEDIGREE 2.2 (Herbinger, 2005) based on data from 13 microsatellite loci (see Thériault *et al.*, 2007, for methodological details). Six loci showed departure from Hardy–Weinberg equilibrium (Table 1), which could be explained in part by the presence of null alleles at three loci (Thériault *et al.*, 2007, see discussion about the consequence of null alleles on sib-reconstruction). Genotypic linkage disequilibrium was also found between pairs of loci and we believe that this might lower the power of our dataset to reconstruct pedigrees. The partitioning of full-sibs was carried out for each cohort separately as we assumed that full-sibling relationship between fish of different cohorts is very unlikely. In the complete absence of parental information, PEDIGREE uses a Markov Chain Monte Carlo (MCMC) approach to partition sibling by maximizing an overall likelihood score on the basis of pairwise likelihood ratios of being full siblings or unrelated (Smith *et al.*, 2001; Butler *et al.*, 2004; Herbinger *et al.*, 2006). The algorithm is constrained such that within a group of putative siblings the genotypes at each locus must be able to be derived from a single parental pair (Herbinger, 2005). Multiples runs (from 20 to 60) were performed to find the 'best' sibship configurations, i.e. the one yielding the highest score. This configuration is then retained for significance testing using genotype randomizations followed by full-sib reconstruction. Thus, a total of 100 sets of the same number of unrelated individuals as used for the actual pedigree reconstruction was created, sampled from populations with the same genotypic frequencies as in our original dataset. The overall significance (*P*-value) of the pedigree retained was estimated by the proportion of the 100 randomized trials with a partition score as high or higher than the observed score. Significance of each full-sib family was also assessed. A specific full-sib family was deemed significant and kept in the analysis when its internal cohesion score, the average of the Log of the

Table 1 Number of alleles (n), observed (H_o) and expected (H_e) heterozygosity at each locus.*

Locus	n	H_o	H_e
*SfoB52**	12	0.68	0.74
SfoC113	12	0.79	0.75
SfoC129	7	0.73	0.71
SfoC28	10	0.52	0.54
SfoC88	6	0.60	0.61
*SfoC115**	23	0.55	0.63
*SfoD100**	10	0.76	0.81
*SfoD75**	13	0.75	0.80
*SCO204**	13	0.76	0.80
SCO216	18	0.88	0.89
SCO218	14	0.81	0.80
*Sfo262Lav**	19	0.85	0.82
Sfo266Lav	30	0.87	0.87

*Significant departure from Hardy–Weinberg equilibrium. *SfoB52, SfoC113, SfoC129, SfoC28, SfoC88, SfoC115, SfoD100, SfoD75*, T. L. King, US Geological Survey, unpublished data; *SCO204, SCO216, SCO218* from DeHaan & Ardren (2005); *Sfo262Lav, Sfo266Lav* from Perry *et al.* (2005b).

pairwise likelihood ratios in that group, was higher than the cohesion scores seen in at least 95 full-sib groups of the same size out of the 100 produced by the randomization procedure. All other full-sib groups were discarded from subsequent analyses, as well as all individuals not related to any other (see also Herbinger *et al.*, 2006).

Quantitative genetic analyses

Single cohort analysis did not yield significantly different quantitative genetic estimates (not shown). Consequently, pedigrees obtained for cohorts 1 and 2 were combined in subsequent analyses. Traits of interest included life-history tactic, scored as 0 for anadromous (fish captured as migrants in trap nets) and 1 for resident (fish captured in the stream by electro-fishing), body size (fork length, FL) and six morphological measures: body depth (DEP), maximum body width (WID), peduncle depth (PED), caudal fin height (CAUD), pectoral fin length (PECT) and pelvic fin length (PELV). Principal component analysis (PCA; SAS, software v.8, SAS Institute Inc., Cary, NC, USA) was performed to remove the size-dependent effect on these morphological measures. In the presence of a size effect, the PCA grouped all morphometric measures along one factorial axis, which was largely explained by variation in size among fish (data not shown). To investigate the variation in body shape independently of size variation, we removed the size effect by regressing the value of each variable for each fish on the first factorial axis. The residual values obtained were used to analyse morphological diversity. A general linear model (GLM, JMP™ 5.0.1a; SAS Institute Inc., Cary, NC, USA) for continuous morphological traits, and a logistic regression for the dichotomous trait (life-history tactic) were conducted to determine which factors needed to be included as fixed effects in subsequent quantitative genetic analyses. This was done in order to account for temporal heterogeneity in environmental effects on the phenotype. Because of the small number of 2+ individuals in our pedigree ($n = 41$), analyses could not be calculated separately by age, which was thus fitted as a fixed effect. Other fixed factors were year of sampling (2002, 2003 and 2004) and day of capture. Age had a significant influence only on FL ($F_{1,214} = 115.22$, $P < 0.0001$). Year was included as a fixed effect for every trait except PECT (life-history tactic, $c^2_{2,n=349} = 14.94$, $P = 0.001$; WID, $F_{1,214} = 7.11$, $P = 0.008$; DEP, $F_{1,214} = 8.36$, $P = 0.004$; PED, $F_{1,214} = 31.84$, $P < 0.0001$; CAUD, $F_{1,214} = 30.78$, $P < 0.0001$; PELV, $F_{1,214} = 8.53$, $P = 0.004$). Given its effect on FL ($F_{1,214} = 7.45$, $P = 0.0069$), DEP ($F_{1,214} = 16.88$, $P < 0.0001$), PED ($F_{1,214} = 38.39$, $P < 0.0001$), CAUD ($F_{1,214} = 22.75$, $P < 0.0001$) and PELV ($F_{1,214} = 5.76$, $P = 0.02$), day of capture was fitted as a fixed effect for these traits.

Heritability of each trait was estimated using a mixed model REML estimation procedure using the software package ASReml (1.10; VSN International Ltd., Hemel Hempstead, UK). Pedigree information was used to fit a univariate animal model. The model had the following form:

$$y = Xb + Za + e$$

where **y** is a vector of phenotypic values, **b** and **a** are the vector of fixed and random additive effects, **e** is the vector of residual values, and **X** and **Z** are the corresponding design matrices which relate the effects to **y**. Total phenotypic variance (V_P) of each trait was partitioned into additive genetic variance (V_A) and residual variance (V_R). The narrow-sense heritability (h^2) was estimated as the ratio of the additive genetic variance to the total phenotypic variance: $h^2 = V_A/V_P$. Significance of the additive genetic component of each model was assessed by comparing the full model with a reduced model lacking the additive genetic component using a likelihood ratio test (following a χ^2 distribution, where $\chi^2 = -2$*difference in log likelihood and the change in degrees of freedom between models = 1). As life-history tactic was scored as a binary trait, the heritability estimate and its associated standard error were transformed to the underlying liability scale (see Falconer & Mackay, 1996; Roff, 2001). Heritability of life-history tactic thus refers to the heritability of the liability of the trait, but for convenience, we refer simply to its phenotypic manifestation (i.e. h^2 of life-history tactic, see Roff *et al.*, 1997).

Genetic correlation between life-history tactic and length, as well as between life-history tactic and the six morphological traits related to body shape were calculated using pairwise multivariate animal models. The same fixed and random effects used for heritability estimation were used in theses analyses. Genetic correlations were calculated as

$$r_G = \mathrm{COV}_{AB}/\sqrt{V_A V_B}$$

using the program ASReml. Significance of the genetic covariance was assessed by comparing the likelihood of the model containing the genetic covariance component with the reduced model in which the genetic covariance was fixed at 0, again using likelihood ratio tests.

Power and sensitivity analysis

Power analysis

We used the software package PEDANTIX (Morrissey *et al.*, 2007) to assess the power of the resolved pedigree to detect significant quantitative genetic parameters. This power analysis wants to determine if enough information is found in our dataset to detect quantitative genetic parameters, particularly because data availability is limited for morphological measures (see *Results*).

PEDANTIX uses a given pedigree to simulate phenotypic data for two traits simultaneously (continuous data, PHENSIM application) according to a user-defined variance–covariance matrix. Continuous data can be

converted afterwards to binomial data using the application ADVPHESIM. The phenotypic data obtained are then used for quantitative genetic analyses, following the same procedure as for real data (i.e. animal model implemented in ASReml). We used Pedigree 1 (see below) and simulated phenotypic data for a continuous trait and a binomial trait, according to different values of heritability and genetic correlation (from 0.2 to 0.5) and assessed the power of our pedigree to detect the expected quantitative genetic parameters. Power was expressed by the number of simulations that gave a significant heritability estimate as a fraction of the total number of simulations ($n = 20$).

Varying parameters in PEDIGREE

PEDIGREE can yield different full-sib arrangements depending on the numbers of runs and the parameters used by the user, all of which can be equally probable. PEDIGREE does not provide any strict criterion to choose the reconstruction that represents the most plausible pedigree. Here, we assessed the consequences of varying one parameter, the weight, on the quantitative genetic analyses by comparing two different pedigrees reconstructed using the procedure described above. The weight is an *ad hoc* parameter in the range of 1–10, 1 being neutral and the default value used in most cases (Smith *et al.*, 2001; Butler *et al.*, 2004; Herbinger *et al.*, 2006). A higher weight promotes the coalescence of individuals into larger groups, which is useful as PEDIGREE tends to split very large full-sib families into subgroups (Smith *et al.*, 2001; Butler *et al.*, 2004). Here, we compared the quantitative genetic parameters estimated using pedigrees reconstructed with a weight of 1 (Pedigree 1) and a weight of 5 (Pedigree 2).

Full-sib assumption

We assumed a full-sib structure while using PEDIGREE, but this assumption is likely to be violated because of complex mating patterns in our system, where both sexes have many partners (Thériault *et al.*, 2007). PEDIGREE allows the reconstruction of kin-groups (mixtures of full-sibs, half-sibs and sometimes higher degrees of relationship such as cousins), and to mix kin pedigree with full-sib pedigree in order to reconstruct half-sib structure, thus potentially resolving a more complete pedigree (Herbinger *et al.*, 2006). However, this procedure was too complex to be powerful in our system, and we chose to assess the consequence of using a full-sib constraint on the estimation of quantitative genetic parameters using two alternative methods. First, we added several half-sib relationships to Pedigree 1 by combining results from parentage assignments previously obtained with PASOS (Duchesne *et al.*, 2005; Thériault *et al.*, 2007) with those from PEDIGREE, such that the identity of one parent was added to a full-sib family when one or more juveniles in that particular full-sib family had a known parent. This exercise resulted in 12 full-sib families sharing four different known parents (involving a total of 60 progeny, Pedigree 3). Secondly, we used the software package PEDANTIX (Morrissey *et al.*, 2007) to simulate half-sib structure in Pedigree 1 and to evaluate the consequences on quantitative genetic parameters. We first added some half-sib relationships to full-sib Pedigree 1 by assigning a common parent to full-sib families on a pairwise basis. For example, full-sib families 1 and 2 now shared a common parent, as well as families 3 and 4, and so on. This half-sib pedigree was then used by PEDANTIX to simulate phenotypic data according to different scenarios where heritabilities and genetic correlations ranged from 0.2 to 0.5. The quantitative genetic parameters were then estimated with ASReml using the simulated phenotypes, along with the actual full-sib pedigree. This allowed assessing the consequences of using a full-sib pedigree even if our true pedigree had an important half-sib structure.

Results

Pedigree reconstruction

A total of 974 age 1+ and 2+ juveniles were available for sib-reconstruction for 2002, 2003 and 2004 combined (anadromous form $n = 440$, resident form $n = 534$). The best pedigrees retained yielded scores that were not reached by any of the 100 randomizations performed, indicating that we could reject the null hypothesis that the full-sib partitions could have been seen in sets of unrelated individuals ($P < 0.01$). From the initial 974 juveniles, we sorted out those not grouping with any other based on the assumption of full-sibs as well as those clustering into families that were not significant at a 0.05 level. This left 349 juveniles grouped into 91 full-sib families for the quantitative genetic analyses for Pedigrees 1 and 3. When using a weight of 5 (Pedigree 2), this same process left 351 juveniles sorted into 89 full-sib families. Family size ranged from two to 12 individuals, with a mean (±SE) of 3.84 ± 0.17 and 3.94 ± 0.19 individuals per family for Pedigree 1 + 3 and Pedigree 2 respectively (Fig. 1). Thus, the use of a higher weight did not translate into the coalescence of individuals into larger families.

Heritability and genetic correlations

Significant additive genetic variance and heritability were obtained with the three pedigrees for the life-history tactic, body size (FL), as well as body depth (DEP), the heritability values ranging from 0.39 to 0.56 (Table 2). Power analysis involving a binomial trait showed that a moderate to high value of heritability was needed in order to be detected with our pedigree structure; power dropped from 0.9 to 0.6 when heritability values of 0.5 and 0.4 were simulated respectively (Table 3). The threshold transformation proved to be reliable, as estimated values following the threshold transformation were generally in

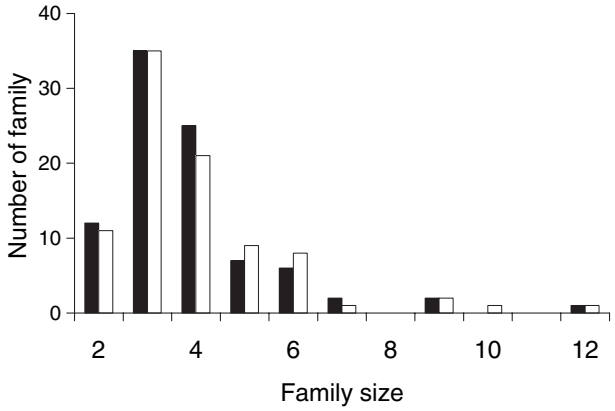

Fig. 1 Distribution of family size for Pedigree 1 and 3 (black bars) and Pedigree 2 (white bars). These families are those that were kept in the final analyses, i.e. the ones that were deemed significant by the randomization procedures (see *Methods*).

good agreement with expected simulated values on the continuous scale (Table 3). This analysis also revealed that the type of pedigree structure that we used was capable of detecting a heritability of 0.2 for body size with a power of 0.75, but that heritabilities below 0.4 were only detected in 60% of the cases for morphological traits (sample size is smaller for morphological traits, Tables 2 and 3). It thus appear that data availability was of concern in this system and that the pedigree used for morphological data was not adequate to detect small to moderate heritabilities. As a result, large standard errors were associated with all quantitative genetic estimates of traits other than body size. Also, estimates for morphological traits varied slightly depending on the pedigree used. Namely, marginally significant heritability was detected for PED with Pedigree 2 but not with the two other pedigrees (Table 2). Moreover, Pedigree 1 and 2 led to marginally significant heritability for PELV, but not Pedigree 3 (Table 2).

Table 2 Sample size (n), trait means with their standard deviation (SD), estimates of residual (V_R), additive (V_A) and phenotypic (V_P) variance components and heritability (h^2) with their standard error (SE) for life-history tactic (anadromy/residency: Tactic), body size (fork length: FL) and six morphological traits (body depth: DEP, maximum body width: WID, peduncle depth: PED, caudal fin height: CAUD, pectoral fin length: PECT and pelvic fin length: PELV, all transformed following PCA, see *Methods*) in brook charr.

Traits	n	Mean (SD)	V_R (SE)	V_A (SE)	V_P (SE)	h^2 (SE)	P-value
Pedigree 1							
Tactic	349	1.47 (0.50)	0.15 (0.03)	0.08 (0.03)	0.24 (0.02)	0.56 (0.18)	0.0003
FL (mm)	349	88.95 (14.55)	42.33 (8.51)	42.73 (12.47)	85.06 (7.18)	0.50 (0.12)	< 0.0001
DEP	215	−0.15 (0.80)	0.26 (0.06)	0.17 (0.08)	0.43 (0.04)	0.40 (0.16)	0.0069
WID	215	0.06 (0.59)	0.26 (0.05)	0.04 (0.05)	0.30 (0.03)	0.13 (0.18)	0.6351
PED	215	−0.04 (0.49)	1.52 (0.30)	0.24 (0.26)	1.77 (0.17)	0.14 (0.15)	0.7574
CAUD	215	0.56 (1.42)	0.19 (0.04)	0.05 (0.04)	0.24 (0.02)	0.22 (0.15)	0.1024
PECT	215	−0.19 (0.57)	0.20 (0.04)	0.01 (0.03)	0.21 (0.02)	0.06 (0.13)	0.7216
PELV	215	−0.07 (0.50)	0.23 (0.05)	0.09 (0.05)	0.32 (0.03)	0.28 (0.15)	0.0386
Pedigree 2							
Tactic	351	1.48 (0.50)	0.11 (0.02)	0.05 (0.02)	0.16 (0.01)	0.52 (0.19)	0.0002
FL (mm)	351	88.99 (14.63)	48.68 (9.65)	38.78 (12.16)	87.48 (7.25)	0.44 (0.12)	< 0.0001
DEP	216	−0.15 (0.79)	0.20 (0.06)	0.22 (0.08)	0.41 (0.04)	0.53 (0.16)	0.0003
WID	216	0.03 (0.58)	0.26 (0.05)	0.03 (0.05)	0.30 (0.03)	0.11 (0.16)	0.5868
PED	216	−0.02 (0.50)	1.29 (0.28)	0.55 (0.31)	1.84 (0.19)	0.30 (0.16)	0.0445
CAUD	216	0.55 (1.45)	0.22 (0.04)	0.04 (0.04)	0.26 (0.03)	0.15 (0.14)	0.2465
PECT	216	−0.06 (0.51)	0.22 (0.04)	0.03 (0.04)	0.25 (0.02)	0.13 (0.14)	0.3126
PELV	216	−0.09 (0.55)	0.22 (0.05)	0.08 (0.05)	0.30 (0.02)	0.27 (0.15)	0.0463
Pedigree 3							
Tactic	349	1.47 (0.50)	0.15 (0.03)	0.08 (0.03)	0.24 (0.01)	0.55 (0.19)	0.0005
FL (mm)	349	88.95 (14.55)	43.21 (9.83)	42.65 (13.00)	85.86 (7.34)	0.50 (0.13)	< 0.0001
DEP	215	−0.146 (0.80)	0.26 (0.07)	0.17 (0.08)	0.43 (0.04)	0.39 (0.16)	0.0105
WID	215	0.055 (0.59)	0.28 (0.05)	0.02 (0.05)	0.30 (0.03)	0.06 (0.16)	0.8024
PED	215	−0.037 (0.49)	1.55 (0.28)	0.22 (0.25)	1.77 (0.17)	0.12 (0.14)	0.3753
CAUD	215	0.560 (1.42)	0.19 (0.04)	0.06 (0.04)	0.24 (0.02)	0.23 (0.15)	0.0797
PECT	215	−0.109 (0.57)	0.22 (0.04)	0.03 (0.04)	0.25 (0.02)	0.12 (0.14)	0.3820
PELV	215	−0.069 (0.50)	0.26 (0.05)	0.07 (0.05)	0.33 (0.03)	0.22 (0.15)	0.1160

Pedigree 1 is the best one obtained with a weight of 1, Pedigree 2 is the best one obtained with a weight of 5, and Pedigree 3 is the same as Pedigree 1, but where half-sib relationships were added (see *Methods*). h^2 of the life-history tactic is given transformed to the liability scale (see *Methods*). *P-values* are those obtained from likelihood ratio tests to assess the significance of the additive genetic component.

Table 3 Power analysis for a range of simulated heritabilities (h^2) for a continuous and a binomial trait.

	$n = 349$ individuals				$n = 215$ individuals	
	Binomial trait		Continuous trait		Continuous trait	
h^2 simulated	h^2 estimated mean (SE)	Power	h^2 estimated mean (SE)	Power	h^2 estimated mean (SE)	Power
0.20	0.28 (0.15)	0.35	0.25 (0.11)	0.75	0.24 (0.14)	0.39
0.30	0.44 (0.14)	0.55	0.32 (0.11)	0.90	0.33 (0.15)	0.60
0.40	0.37 (0.18)	0.60	0.41 (0.11)	0.95	0.44 (0.16)	0.85
0.50	0.47 (0.17)	0.90	0.50 (0.12)	1.00	0.50 (0.16)	0.90

The mean heritability estimate and the mean standard error of 20 simulations are presented. Simulations were ran in order to mimic the actual data. Heritabilities estimated with a sample size of 349 individuals refer to body size and tactic, whereas a sample size of 215 relates to morphological traits. The heritability of tactic is given transformed to the liability scale for comparison purposes (see *Methods*). Power is assessed by dividing the number of simulations that gave a significant additive variance estimate over a total number of 20 simulations.

Table 4 Genetic correlations with their standard errors between life-history tactic (anadromy/residency) and body size (FL) and the six morphological traits for the three pedigrees used (as in Table 2).

Traits	Pedigree 1	*P*-value	Pedigree 2	*P*-value	Pedigree 3	*P*-value
FL	−0.52 (0.22)	0.0333	−0.20 (0.24)	0.4401	−0.61 (0.23)	0.0188
DEP	0.11 (0.32)	0.8149	−0.0007 (0.0002)	1.0000	0.13 (0.33)	0.7876
WID	0.55 (0.74)	0.3762	0.37 (0.51)	0.4708	1.09 (2.45)	0.2506
PED	−0.003 (0.55)	0.6634	0.21 (0.31)	0.5508	−0.06 (0.66)	1.0000
CAUD	−0.50 (0.54)	0.4393	0.07 (0.47)	1.0000	−0.54 (0.54)	0.3877
PECT	−0.82 (0.31)	0.0655	−0.70 (0.25)	0.0477	−0.60 (0.32)	0.1526
PELV	0.16 (0.35)	1.0000	0.35 (0.30)	0.3192	0.24 (0.37)	0.5565

Life-history tactic was coded as 0 for anadromous and 1 for resident. *P*-values are those obtained from likelihood ratio tests to assess the significance of the covariance genetic component.

A negative genetic correlation between life-history tactic and body size was significant for pedigree 1 and 3, whereby anadromous fish were genetically associated with bigger size at age (Table 4). Pedigree 2 yielded a significant genetic correlation between life-history tactic and PECT (Table 4). None of the other genetic correlations between life-history tactic and morphological traits were significant, and all the estimates were associated with large standard errors, showing an important amount of variation between pedigrees (Table 4). Power analysis revealed that genetic correlations were harder to detect than heritabilities. Assuming a heritability of 0.5 for both continuous and binomial traits, a genetic correlation of 0.5 could be detected in 65% of the cases between life-history tactic and body size ($n = 349$, mean estimated $r_G = 0.52$, mean standard error = 0.22) and in 55% of the cases between tactic and morphological data ($n = 215$, mean estimated $r_G = 0.68$, mean standard error = 0.32).

Adding information on half-sib relationships resolved using PASOS to the reconstructed pedigree did not change any of the estimates of heritability or genetic correlation as these were not significantly different between Pedigree 1 and 3 for any of the traits (*z*-scores from 0.01 to 0.78, *P*-values from 0.43 to 0.99). The same conclusion was obtained using PEDANTIX as quantitative genetic parameters estimated using the full-sib pedigree yielded results that agreed well with the expected values assuming a half-sib structure (Table 5).

Table 5 Mean heritability (h^2) and genetic correlation (r_G) estimates as well as mean standard error (SE) obtained over 20 simulations using a full-sib pedigree along with simulated phenotypes data from a half-sib pedigree. Phenotypes data were simulated according to h^2 and r_G similar to those estimated in the present study.

	Simulated	Estimated mean (SE)
h^2 binomial	0.50	0.48 (0.17)
h^2 continuous	0.50	0.51 (0.12)
r_G binomial – continuous	0.50	0.52 (0.23)

Discussion

The main objective of this study was to estimate the heritability of life-history tactics (residency/anadromy) in brook charr under natural conditions by means of pedigree reconstruction assisted by molecular markers. Our results demonstrate that the adoption of either anadromy or residency involves a significant amount of additive genetic variance. To our knowledge, this is the

first study to provide an estimate of heritability for these tactics under natural conditions.

The heritability value obtained for life-history tactic is within the range of heritability estimates generally reported for threshold traits (reviewed in Roff, 1996), where approximately one-half of the phenotypic variation can be attributed to additive genetic variance. It thus suggests that tactics have considerable potential to respond to selection and evolve, but it also implies a relatively important environmental influence on the adoption of a tactic. Other studies have provided estimates of heritability of threshold traits linked to anadromy and residency in salmonids under controlled experiment (early maturity, Wild *et al.*, 1994; precocious maturity, Silverstein & Hershberger, 1992; jacking, Heath *et al.*, 1994; Mousseau *et al.*, 1998; Heath *et al.*, 2002; smolting and maturing, Thrower *et al.*, 2004) and the values obtained were highly variable (from low and nonsignificant to high). These wide differences highlight the fact that it is unwarranted to compare absolute values of heritability as they are a property of the population under study and the conditions where they were measured (Stearns, 1992; Falconer & Mackay, 1996).

We also found significant heritability for body size, our estimates being higher (0.44–0.50) than other reported estimates for body size (at age 1+) in salmonids under natural conditions (e.g. 0.001 ± 0.04 and 0.26 ± 0.12 in brook charr for two populations respectively, Wilson *et al.*, 2003a; 0.04 ± 0.15, in Atlantic salmon, *Salmo salar*, Garant *et al.*, 2003). Body size is an important liability trait for early sexual maturity and anadromy in salmonids, especially in Atlantic salmon where early maturing males are bigger in early life stages than anadromous males (Whalen & Parrish, 1999; Garant *et al.*, 2002; Aubin-Horth & Dodson, 2004). In a previous study from the same tributary, back-calculated length-at-age revealed that smaller brook charr at age 1+ delay migration to the following year, resulting in age 2+ migrants being smaller than age 2+ resident fish (Thériault & Dodson, 2003). At age 1+ however, when removing these smaller future migrants from the sample of resident fish, these authors observed no difference in body size between migrant and resident fish. Thériault & Dodson (2003) thus proposed that body size was not the only trait associated with the adoption of the life-history tactic at that age. Physiological traits related to the energetic budget are more likely to influence tactic choice (Morinville & Rasmussen, 2003, 2006). Heritability for body size (0.44–0.50) was similar to that of the liability itself (the life-history tactic, 0.52–0.56) suggesting that body size could be a major component of the liability, although other components might also be involved.

The covariance analyses suggest a genetic correlation between life-history tactic and body size, although larger sample sizes would have been needed to yield more accurate estimations of genetic correlation. Another potential concern is the reliability of the correlation between a binomial trait and a continuous one using the animal model method, as this has rarely been done (but see Wilson *et al.*, 2003a). However, simulation analysis using PEDANTIX showed that the estimated genetic correlation between a binomial trait and a continuous trait corresponded to the expected one assuming two continuously distributed traits. Our results thus suggest that differences in body size observed previously between anadromous and resident brook charr (phenotypic correlation of −0.15, $P < 0.01$; Thériault & Dodson, 2003) is partly due to the genetic correlation between size and life-history tactic. As anadromy was coded 0 and residency 1, the negative correlation we documented implies that bigger fish would be genetically more prone to be anadromous. This conclusion must be constrained to age 1+ fish and is consistent with the phenotypic correlation: at age 1+, migrants appear to be bigger because the sample of resident fish includes small fish, which would ultimately migrate the following year (Thériault & Dodson, 2003). Also, the genetic correlation between these two traits suggests that selection acting on one of them would likely have an effect on the other. For example, given continued selection against fish migrating at age 1+ (for instance because of a more pronounced fishing pressure on anadromous than resident fish), one would predict a correlated response in body size, 1+ individual being smaller in subsequent generations. A similar result was obtained in a breeding experiment with steelhead trout (*Oncorhynchus mykiss*), where the proportion maturing at age 2 was negatively genetically correlated with mass at age 1 (Thrower *et al.*, 2004). Yet, the intermediate values of the correlation found in the present study (−0.20 to −0.61) gives support to the hypothesis stated above that body size is not the only component of the liability underlying life-history tactic expression. If so, one would expect body size and the liability trait to reflect more or less the same genetic character and the genetic correlation obtained to be closer to unity (Falconer & Mackay, 1996).

Estimating genetic quantitative parameters in the wild is a difficult task and it is not always feasible to estimate the variance components that are likely to influence phenotypic covariance between relatives. In this study, we lacked the information required to estimate maternal or common environmental effects, which, if present, could potentially inflate heritabilities values (Falconer & Mackay, 1996). First, we had no information on the parental generation of our fish, and thus mother identity was unknown. However, maternal effects on body size have been shown to be nonsignificant at 0, 1 and 2 years old in Atlantic salmon of the Sainte-Marguerite River (Garant *et al.*, 2003). More generally, it has also been showed that when maternal effects are detected in salmonids, they are more important at early larval stage and seem to decrease with age (Heath *et al.*, 1999; Perry *et al.*, 2005a). Because our study focused on later

life-history stage, we assumed that maternal effects were negligible. Common environment effects are another concern that cannot be easily addressed in our system. The sampling location of each resident fish was recorded, but because the migrants were all sampled in one downstream trap during their outmigration in spring, their section of origin was unknown. However, animal models with sampling location as a fixed effect for residents have been fitted for body size, and gave similar results to those without sampling location (results not shown). Moreover, qualitative analysis of our results from parentage analyses (see Thériault *et al.*, 2007) showed that members of only five half-sib families out of 44 were sampled in the same 300 m sampling section. All other juveniles sharing one parent were found all along the stream, from several hundred metres to 3 km apart, suggesting considerable dispersal during the first and/or second year of life. Similar qualitative analysis of our data with the full-sib information obtained here with PEDIGREE revealed that only 30% of the members of a same family were found in the same 300 m section. There is thus little evidences suggesting that common environment effects might be inflating our quantitative genetics parameters.

Estimates of quantitative genetic parameters obtained in nature have mainly been obtained from long-term studies that used large pedigrees (reviewed in Kruuk, 2004). Although the number of recent studies that use highly polymorphic genetic markers to assess pedigree information is growing (reviewed in Garant & Kruuk, 2005), the limitations of such tools need to be acknowledged. Here, we used a sib-reconstruction method assuming unrelated full-sib families only, although this assumption is likely to be violated in the study system. Indeed, mating pattern in brook charr involves both sexes mating with many different partners and thus a more realistic pedigree would necessarily involve an important half-sib structure (Thériault *et al.*, 2007). A procedure nesting full-sib families with kin groups has been used elsewhere to provide a more accurate pedigree reconstruction (see Herbinger *et al.*, 2006), but the limited numbers of large families in our study prevented us from using this approach. Moreover, we attempted to use a more complete pedigree (obtained from parentage analysis), but ended up with a pedigree that did not contain enough related individuals (see Thériault *et al.*, 2007). This prevented model convergence when estimating quantitative genetic parameters. Nevertheless, assuming a full-sib pedigree structure may have only a limited impact as most of the information used in estimating quantitative genetic parameters stems from close relatives (Thomas & Hill, 2000). This was supported here by the absence of significant differences in quantitative genetic parameter estimates between the full-sib pedigree (Pedigree 1) and the pedigree including some half-sib relationships (Pedigree 3). Furthermore, simulations showed no significant effect when using phenotypes created from a half-sib pedigree along with our full-sib pedigree to estimate quantitative genetic parameters. This results also argue in favour of additive genetic effects not being confounded with nonadditive ones because of the use of full-sibs only (Falconer & Mackay, 1996).

Sib-reconstruction using a MCMC approach is not free of errors. Namely, the algorithm used by PEDIGREE 2.2 tends to split large full-sib families into subgroups when using allelic frequencies estimated in the sample instead of true population allelic frequencies (Smith *et al.*, 2001; Butler *et al.*, 2004). This type of error will have the same consequence as considering only full-sibs, i.e. the split families are assumed to be unrelated when in fact they are not, which may artificially decrease phenotypic variance between families and thus produces conservative heritability estimates (Wilson *et al.*, 2003b). However, our use of a higher weight while reconstructing Pedigree 2 specifically aimed to mitigate this tendency but did not result in coalescence of large full-sib groups. We thus conclude that splitting large full-sib families into subgroups was not of concern in this study. Moreover, quantitative genetic estimates were generally in good agreement between pedigrees using a weight of 1 or 5. Genotyping errors are also likely to influence sib-reconstruction (Butler *et al.*, 2004) and these errors are not taken into account in subsequent quantitative genetic analyses under the animal model. Null alleles have been detected in this population (Thériault *et al.*, 2007). Such mistyping of heterozygotes as homozygotes are more likely to cause families to be split rather than incorrect families to be formed, which should cause a downward bias in quantitative genetic parameter estimates (Thomas & Hill, 2000). Simulation analyses performed by Butler *et al.* (2004) have shown that reconstruction algorithm such as that implemented in PEDIGREE 2.2 is relatively robust to such problems. It appears that when information from several other loci is available, the resulting offspring genotypes are still full-sib compatible, and are, in the majority of cases (75%), consistent with Mendelian rules (Butler *et al.*, 2004).

Two studies have compared estimates of quantitative genetic parameters obtained using a sib-reconstructed pedigree to ideal values obtained with a 'true-pedigree' resolved from parentage analysis: both concluded that the results were reliable and close to ideal values, although parameters were generally underestimated (Thomas *et al.*, 2002; Wilson *et al.*, 2003b). In particular, Wilson *et al.* (2003b) used an aquaculture population of rainbow trout (*O. mykiss*) to compare heritability and genetic correlations estimates between pedigree obtained by sibship-reconstructions (using the same MCMC approach as in the present study) and a pedigree built using a parentage analysis based on an exclusion approach. Their population contained a high number of half-sib relationships because of factorial crossing in the

parental generation, but these relationships were not taken into account while performing sibship-reconstructions (as in our study). The authors concluded that the underestimation of quantitative genetic parameters from sib-reconstructed pedigrees, relative to a 'true-pedigree', is mainly explained by the complex structure of the true pedigree. The true pedigree consisted of a high number of half-sibling relationships, which caused an inaccurate partitioning of full-sibships and reduced the recognition of relatedness between families (see Wilson *et al.*, 2003b). Furthermore, the authors reported difficulties in obtaining meaningful estimates of quantitative genetic parameters, especially for genetic correlations, when using subsets of their dataset and thus smaller sample sizes (Wilson *et al.*, 2003b).

Although our analyses suggest no major problems of assuming only full-sibs relationships (e.g. no underestimation of estimated parameters as observed by Wilson *et al.*, 2003b), the methods we used to reach such a conclusion may not completely account for the complexity of the half-sib structure. Furthermore, we do not know the extent of inaccurate partitioning of full-sibs owing to a high number of half-sibs in our system and to the presence of null alleles at certain loci. Resolving the half-sib structure in such a system where mating pattern is complex and where full-sib family sizes seem small (at least when using 1+ and 2+ juveniles for sib-reconstruction) would require parental assignment and thus a sampling of spawners as complete as possible. Because sib-reconstruction was the only alternative, increasing juvenile sample size would have improved the power of analyses in our case. Arguably however, such constraints are likely to apply to most studies performed in similar systems involving natural populations. Finally, as adoption of a particular life-history tactic can occur at two different ages in our study system, it would be pertinent in future studies to measure age-specific values of heritability and genetic correlation. Indeed, age-specific variation in the amount of genetic variance has been shown to occur under natural conditions in morphological and life-history traits (see Perry *et al.*, 2004; Charmantier *et al.*, 2006). As such, different age classes are different with respect to their evolutionary potential or response to selection. Moreover, estimates over many years would also shed light on the temporal stability of response to selection, as genetic parameters are expected to change depending on environmental conditions (Hoffmann & Merila, 1999; Charmantier & Garant, 2005).

To conclude, this study represents a contribution towards the acquisition of estimates of quantitative genetic parameters under wild conditions, and more than being one of the few (see also Wilson *et al.*, 2003a) to demonstrate the usefulness of sibship-reconstruction in nature in the absence of parental information, it also provides for the first time heritability estimates of anadromy and residency in salmonids in an entirely natural set-up.

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Supplementary material

The following supplementary material is available for this article:

Figure S1. Location of the study area, sampling traps and electro-fishing sites on Morin Creek, a tributary of the Sainte-Marguerite River, Quebec, Canada.

This material is available as part of the online article from:

http://www.blackwell-synergy.com/doi/abs/10.1111/j.1420-9101.2007.01417.x

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