

## ORIGINAL ARTICLE

# Maintenance of phenotypic variation: repeatability, heritability and size-dependent processes in a wild brook trout population

Benjamin H. Letcher,<sup>1</sup> Jason A. Coombs<sup>1,2</sup> and Keith H. Nislow<sup>3</sup>

1 S.O. Conte Anadromous Fish Research Center, US Geological Survey/Leetown Science Center, Turners Falls, MA, USA

2 Program in Organismic and Evolutionary Biology, University of Massachusetts, Amherst, MA, USA

3 Northern Research Station, USDA Forest Service, University of Massachusetts, Amherst, MA, USA

## Keywords

brook trout, growth compensation, heritability, repeatability, size-dependent growth.

## Correspondence

Benjamin H. Letcher, S.O. Conte Anadromous Fish Research Center, US Geological Survey/Leetown Science Center, Turners Falls, MA 01376, USA.  
Tel.: 413 863 3803; fax: 413 863 9810;  
e-mail: ben\_letcher@usgs.gov

Received: 1 December 2010

Accepted: 26 January 2011

First published online: 8 March 2011

doi:10.1111/j.1752-4571.2011.00184.x

## Abstract

Phenotypic variation in body size can result from within-cohort variation in birth dates, among-individual growth variation and size-selective processes. We explore the relative effects of these processes on the maintenance of wide observed body size variation in stream-dwelling brook trout (*Salvelinus fontinalis*). Based on the analyses of multiple recaptures of individual fish, it appears that size distributions are largely determined by the maintenance of early size variation. We found no evidence for size-dependent compensatory growth (which would reduce size variation) and found no indication that size-dependent survival substantially influenced body size distributions. Depensatory growth (faster growth by larger individuals) reinforced early size variation, but was relatively strong only during the first sampling interval (age-0, fall). Maternal decisions on the timing and location of spawning could have a major influence on early, and as our results suggest, later (>age-0) size distributions. If this is the case, our estimates of heritability of body size (body length = 0.25) will be dominated by processes that generate and maintain early size differences. As a result, evolutionary responses to environmental change that are mediated by body size may be largely expressed via changes in the timing and location of reproduction.

## Introduction

Forecasting effects of environmental change on population persistence, especially effects of climate change, can be improved by incorporating evolutionary processes into population models (Skelly et al. 2007). Forecasts that ignore evolutionary processes may overestimate rates of extinction or population decline because they do not allow for adaptation to environmental change on contemporary time scales. The potential for evolutionary response to environmental change can be evaluated using the breeder's equation (Kruuk et al. 2008; Wilson et al. 2010), which states that evolutionary rate of change ( $R$ ) for a trait depends on the strength of selection ( $S$ ) and the heritability ( $h^2$ ), where  $R = S \times h^2$ . Multivariate versions

of the breeder's equation are also possible (usually preferable, McGuigan 2006). In an effort to move toward incorporation of evolutionary processes into population models and to understand the relative importance of size-dependent processes including growth and survival, we provide estimates of quantitative genetic variables for the key phenotypic traits of body size and growth from a wild population of brook trout (BKT) (*Salvelinus fontinalis*).

Brook trout, a widespread but locally imperiled stream fish in its native range (Hudy et al. 2008), faces multiple threats to local persistence. As climate changes, BKT may be forced into relatively cool, headwater streams to avoid warming mainstem rivers and invasive competitors and predators. While BKT appear to adapt to isolation (Letcher et al. 2007), it is not known whether local

populations can adapt quickly enough to counteract the potentially negative consequences of isolation resulting from climate change.

Because many key demographic processes (growth, survival, movement, reproduction) are size-dependent in BKT and in many other fish species, it is critical to understand how BKT size distributions are structured to provide a mechanistic basis for understanding population response to environmental change. In general, BKT have remarkably wide size distributions within an age class, e.g. ranging from 61 to 140 mm at age-0 in the autumn within a stream (McFadden 1961). Understanding the source of this variation is complex, because many processes can contribute to size variation, and the relative importance of various sources can change over ontogeny. In general, however, the development of size distributions within a generation depends on three factors: (i) differences in initial sizes among individuals resulting from variable birth dates, (ii) differences in body growth within and among individuals, and (iii) losses or gains resulting from size-dependent survival, emigration or immigration. In BKT, birth or emergence dates can vary widely among individuals (Snucins et al. 1992), providing the foundation for broad size distributions. The wide range in emergence dates in the spring results from a combination of variation in fertilization dates in the previous autumn and winter development rates during incubation in stream beds. The emergence-based size differences in the spring can be either magnified or reduced as the cohort grows depending on the direction of size-dependent growth; spreading out if size-dependent growth is positive, narrowing if it is negative. Size distributions can be further shaped in complex ways by the repeatability of individual growth within an individual, e.g. high repeatability of individual growth can further accelerate the spread of a size distribution because fast growers will remain fast growers over time. Finally, size distributions can be shaped by losses (mortality or emigration) or gains (immigration) to the population, provided the losses or gains are size-dependent. Combined with estimates of size-dependent losses, the variance components approach behind estimating heritability can provide useful estimates of within- and among-individual variance in growth or body size. These estimates can be used to determine the extent to which the relative growth and body size of individuals varies over time, providing important insight into mechanisms responsible for the generation of size distributions.

Heritability can be estimated using parent–offspring relationships in controlled settings, but this approach usually involves some level of artificial manipulation (Kruuk and Hadfield 2007). Recently, researchers have taken advantage of long-term field studies to estimate repeat-

ability and heritability in natural settings using multiple-generation pedigrees (Wilson et al. 2010). In this approach, heritability is simply the proportion of total phenotypic variance that can be attributed to additive genetic effects, and repeatability represents the proportion of total phenotypic variance owing to both genetic and nongenetic sources of among-individual phenotypic variation (Falconer and Mackay 1996; Wilson et al. 2010). The pedigree is used to isolate phenotypic variance resulting from additive genetic effects from other sources of variance (other sources of individual variance and residual variance) in a mixed linear model (often called the ‘animal model’; Kruuk 2004). This is an extremely flexible modeling approach that can be used for effective heritability and repeatability estimation, but also to test hypotheses in a linear modeling, model selection framework.

To date, most studies estimating heritability in the wild have focused on species for which parent–offspring relationships can be observed, most notably ungulates and birds (Garant et al. 2005; Pelletier et al. 2007). These studies have yielded tremendous amounts of information about heritability in the wild and about evolutionary processes in general, but they represent a fairly narrow slice of life history space (e.g. determinate growth, low fecundity, size-independent fecundity). Studies with enough detailed information on individual performance combined with pedigree information for species across life history space would be useful to inform the generality of the existing results (Rodriguez-Munoz et al. 2010). A major difficulty extending this approach to other life histories is generating the pedigree when parent–offspring relationships cannot be observed. This is a diminishing roadblock, however, as multilocus genotypes with sufficient power to resolve pedigrees and more rigorous genotype-based pedigree reconstruction tools become available (Pemberton 2008).

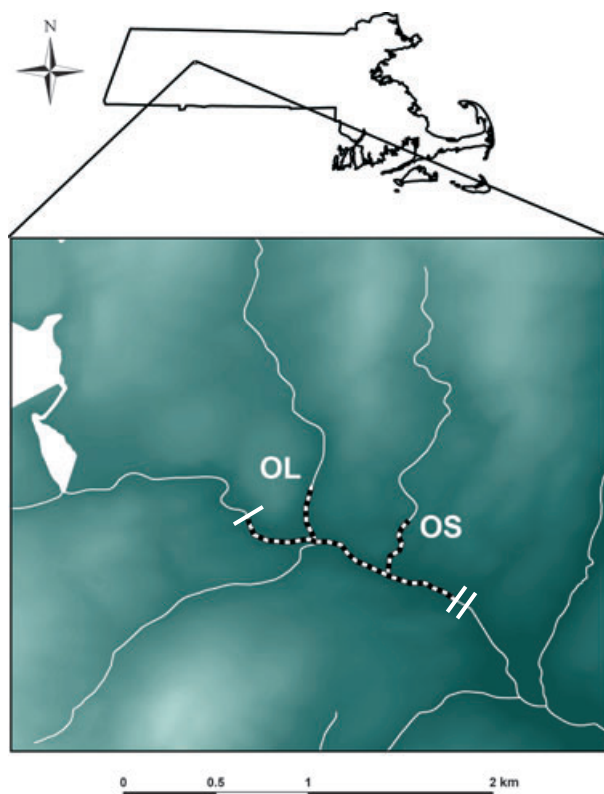
In BKT, the relative importance of variation in emergence dates and growth processes is not well known and is difficult to sort out because studies combining nonlethal estimates of emergence dates and later individual growth rates are challenging. Here, we focus on growth processes and ask three main questions: (i) To what extent do observed size distributions reflect repeatability of individual body size and growth rate? (ii) How are body sizes influenced by size-dependent growth and losses? (iii) Is heritability of body size and growth rate high enough that selection on size or growth could evoke an evolutionary response? Overall, we ask whether an evolutionary response to environmental change is likely to act through body size/growth processes, and to what extent early size differences, later growth rate variation, and size-selective mortality contribute to the generation of size distributions.

## Methods

### Study system

The study was conducted in the West Brook (WB; Whately, MA, USA) and two tributaries (Fig. 1). Watershed area is 11.8 km<sup>2</sup>. Landuse in the watershed is light residential with some farming. The forest is mixed hardwood and provides a strong canopy above the stream. The streambed is primarily cobble with occasional boulders, and stream habitat is largely riffle with scattered pools and glides (details in (Letcher et al. 2002).

The WB study area is a 1-km-long stretch of stream. The downstream end of the study area is bounded by a small waterfall (1-m tall, passable by fish), and the upstream end is unbounded for several km. The two tributaries in the study area are each 300-m long and are both bounded by waterfalls at the upstream ends. The confluence of the OpenLarge tributary with the WB is open, but the confluence of OpenSmall with the WB is interrupted by a perched culvert (0.75-m tall, passable by fish (see Letcher et al. 2007)). The two tributaries are second-order streams with average wetted widths of 2 m



**Figure 1** Map of the study location including study area range (shaded lines) and PIT tag antenna locations (white bars).

(OpenSmall) or 3 m (OpenLarge). The average wetted width of WB is 4.5 m.

Brook trout (BKT, *Salvelinus fontinalis*) and brown trout (*Salmo trutta*) reproduce naturally in the stream. Atlantic salmon (ATS, *Salmo salar*) were stocked as 25-mm-long fry during the early years of the study period (2002–2004). There is no trout stocking in the study stream. The only other fish species consistently present in the stream is blacknose dace (*Rhinichthys atratulus*). Fishing pressure in the WB is very low and nonexistent in the tributaries. Analysis here is limited to BKT.

We sampled fish in the study area seasonally from December 2001 to March 2008 (spring = late March, summer = June, autumn = late September, winter = early December), for a total of 28 sampling occasions (Table 1). We used standard two-pass electrofishing (300 V unpulsed DC current) with block nets at the upstream and downstream ends of each 20-m-long sampling section. Upon capture, we took lengths ( $\pm 1$  mm fork length) of each fish and recorded the sampling location (section). Untagged fish were implanted with 12-mm PIT tags (Digital Angel, St. Paul, MN, USA) if fork length exceeded 60 mm (Gries and Letcher 2002). Fish older than age-0 were aged using size distributions from known-age fish (age-0) captured in the autumn. We also took anal fin clips for genetic analysis. Following work-up, fish were returned to capture sections.

### Analysis

Data for analysis included individual body lengths at capture and growth rates. Growth in length was estimated as change in individual body length over time. For all analyses, we limited the dataset to individuals observed on consecutive sampling occasions (for growth estimates)

**Table 1.** Number of individuals for analysis captured from each cohort across seasons. For each season and cohort combination, numbers of captured individuals were summed over the 7 years of sampling.

Cohort	Season			
	Spring	Summer	Autumn	Winter
<b>Parents</b>				
1999	0	0	2	0
2000	3	9	20	1
2001	36	66	65	14
2002	167	267	389	79
<b>Offspring</b>				
2003	119	169	294	115
2004	148	241	307	139
2005	47	37	71	57

and scaled response variables across all captures to a mean of 0 and a variance of 1. Scaling generates results on relative, not absolute, body size and growth. In the results, growth is labeled as growth from the previous to the current sampling occasion.

### Pedigree

Because we cannot observe parent/offspring pairs in the field, we used individual genotypes to reconstruct the pedigree structure among sampled individuals. All individuals with at least one parent were included in the analyses below. A panel of twelve microsatellite loci [SfoB52, SfoC24, SfoC38, SfoC86, SfoC88, SfoC113, SfoC115, SfoC129, SfoD75, SfoD91a, SfoD100 (King et al. 2003), SsaD237 (King et al. 2005)] was selected based on its ability to accurately reconstruct known full-sibling families and assign parents for synthetic data (see below). Protocols for DNA extraction and amplification were followed as described by King et al. (2005). Loci were electrophoresed on an ABI Prism 3100-*Avant* genetic analyzer (Applied Biosystems Inc., Foster City, CA, USA), and alleles were scored using GENESCAN v3.7 software (Applied Biosystems Inc.).

Genotype error rate was assessed by randomly selecting 100 individuals and performing a second DNA extraction and amplification of all twelve loci. Alleles were compared between the two genotypes for each individual, and a per allele error rate estimate was obtained.

The power of the loci panel to reconstruct full-sibling families accurately and assign parents was assessed through the use of simulated data generated by the program PEDAGOG v1.2 (Coombs et al. 2010a). Genetic and demographic parameters for the simulated population were derived from field data for the BKT population in the study site (Table S1). The simulated population was subjected to a seasonal sampling scheme using field-derived capture probability estimates. Sibship reconstruction and initial parentage assignment analyses were performed on the simulated population using the programs COLONY v1.2 (Wang 2004) (sibship) and PEDAPP v1.1 (Almudevar 2007) (parentage). Final parentage assignments were acquired using the sibship constraint method within the program PEDAGREE v1.04 (Coombs et al. 2010b). The SC method was run using a minimum threshold value of 0.2501 for full-sibling families with two members, and 0.1667 for full-sibling families with three or more members. The results from the two runs were then merged. Accuracy of reconstructed families and assigned parents were calculated using PEDAGREE. A total of ten replicates were simulated. The same methodology outlined above was used to construct the pedigree for the WB dataset. In the following analysis, we only included individuals that were in the pedigree.

### Quantitative genetics

We used the R package MCMCglmm (<http://www.cran.r-project.org>) to estimate quantitative genetics parameters (repeatability, heritability, genetic covariances). This package uses a mixed model approach in a Bayesian estimation framework to estimate individual variance components that are constrained by the relatedness matrix (derived from the pedigree), individual effects that are unconstrained by the relatedness matrix and residual error (as well as other possible random effects). Constrained effects can be attributed to additive genetic variance ( $V_A$ ), unconstrained individual effects to 'permanent environment effects' ( $V_E$ ) and other individual effects to residual error ( $V_e$ ). Repeatability can be estimated as the sum of individual variances divided by total variance [ $(V_A + V_E)/V_P$ ] and can be interpreted as the proportion of total variance attributable to among-individual variation. Heritability is estimated as the ratio of  $V_A$  to the total variance  $V_P$  ( $V_P = V_A + V_E + V_e$ ) and can be interpreted as the proportion of total variance stemming from among-individual variance that derives from the pedigree.

### Repeat measures estimates

Estimates for both repeatability and heritability will be reduced when residual variance is relatively high. Because within-individual variance in body size over time will contribute to residual variance, repeat measures of individuals provide important longitudinal data that can improve understanding of heritability estimates. The mixed model animal model approach can easily accommodate repeat observations of individuals (Wilson et al. 2010). We included observations for each individual in a repeat measures design. This approach takes advantage of the recaptures of individual fish over time and does not limit estimates of heritability and genetic covariances to a single point in time. Including all seasons in the analyses, however, has the potential to reduce the ability to detect repeatability and heritability of body size and growth because growth is quite low during three of four seasons (see results and Xu et al. 2010a). Any growth signal may be very difficult to detect during low-growth intervals because individuals will have minimal opportunity to express growth variation. Also, including intervals of low growth may artificially inflate repeatability or heritability estimates for body size because of limited opportunity for individual change in size. To avoid the potential 'dilution' of repeatability and heritability estimates for growth and the potential inflation for body size estimates, we conducted analyses using growth data from just the fast growth interval (spring) for age 1, 2 and 3 fish. For body size, this includes six sampling occasions (age-1, age-2

and age-3, spring and summer) and for growth includes three sampling intervals (age-1, age-2 and age-3 spring to summer growth).

As a mixed model, the animal model can include both fixed and random effects (Bolker et al. 2009). Because of the highly seasonal BKT growth in our study stream, we included an age  $\times$  season [age = (1, 2, 3), season = (spring, summer)] fixed effect interaction to account for changes in body size over time. For the growth analyses, we included an age fixed effect only (there was only one season). Thus, our repeatability and heritability estimates represent estimates after accounting for seasonal- (size) and age-related (size and growth) variation.

Genetic covariances between body size and growth are very close to 1 when analysis is limited to the fast growth intervals. We ran 'bivariate dependent variable' models across all sampling intervals to estimate sample-specific covariances. In this model, the dependent variables were body length and growth rate for length. To estimate genetic covariances, we divided the additive genetic covariance for a pair of dependent variables (here, size and growth) by the square root of the product of the additive genetic random effects for each variable (Wilson et al. 2010).

Before running models, we tested the effect of prior distributions on model outcome by varying priors from noninformative to informative. We found a moderate effect of priors on model outcome (results not shown), especially on the variances. To minimize the effect of priors on our analyses, we used completely noninformative priors for all parameters in both models. Models were run with 30 000 burn-in iterations followed by 100 000 iterations and a thinning rate of 100. We summarize posterior distributions for model results (repeatability, heritability and genetic covariances) using posterior modes and 95% credible intervals. It is important to note that MCMCglmm only allows positive estimates of variance components, making it difficult to determine whether a variance component estimate or derived parameters like repeatability or heritability are different from 0. To help assess parameter estimates, we provide representative traces and posterior distributions of the Markov chain Monte Carlo (MCMC) samples generated by MCMCglmm.

### Pairwise repeatability estimates

Repeat measures estimates of body size repeatability using the animal model provide an overall estimate of the relative importance of among- to within-individual variance. More detailed information on the time course of repeatability can be generated from pairwise repeatability estimates based on individuals captured in pairs of

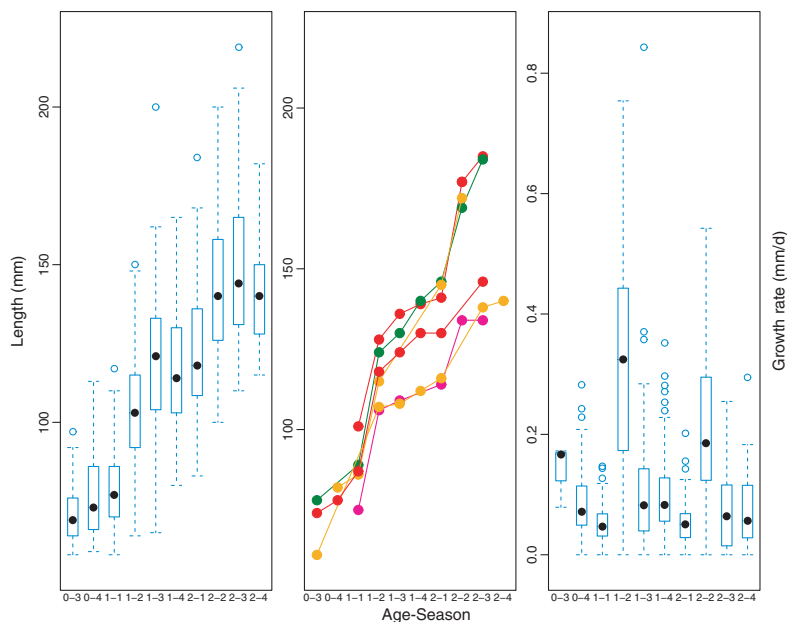
sampling occasions. For pairwise analyses, the correlation coefficient for each pair of sampling occasions provides an estimate of repeatability over the time interval between sampling occasions (Lessells and Boag 1987). We present Pearson correlation coefficient estimates of body length for each pair of sampling occasions in our analysis and for body growth for each pair of sampling intervals.

### Size-dependent survival

To determine the extent to which size-dependent survival could influence size distributions, we estimated survival of fish from different size bins using a multi-state capture-mark-recapture model (Hestbeck et al. 1991; Lebreton and Pradel 2002). Multi-state models offer a flexible framework for estimating survival of individuals belonging to different states, and are particularly useful for estimating survival when the data contain missing observations (Letcher and Horton 2008). We also chose this approach over a method that incorporates size as a continuous variable (such as selection gradients) because it can account for variation in probability of capture and emigration. In our case, the states were size bins (mm, 60–95, 96–115, 116–135, >135). Our analysis included fish from the 2003–2005 cohorts. Some of these fish were not genotyped, so the number of fish for the quantitative genetics analysis does not equal the number of fish for the survival analysis. We used program M-SURGE (Choquet et al. 2004) for parameter estimates and estimated goodness of fit of our data to the multi-state model using program U-CARE (Choquet et al. 2009). We attempted to minimize the confounding of emigration and mortality by coding known permanent emigrants from the study area in the model encounter history file (the input file to the analysis). Known emigrants were identified with PIT tag antennas at the top and the bottom of the study area (Fig. 1). Tag read efficiency of our array has been estimated at 91% (Zydlewski et al. 2006), suggesting that we were able to account for the majority of emigrants from our study area. We used model selection procedures to evaluate the overall effect of body size on survival and compared 95% confidence intervals of state-specific survival estimates (the logit link 'beta' values) to determine whether survival estimates among size states were significantly different. Survival estimates were scaled to a monthly time step.

## Results

Body size distributions were wide, and growth was strongly seasonal (Fig. 2). Typically, the central 50% of the length range for a sampling occasion was about 1/3 of the median size, and 95% size distributions for each



**Figure 2** Body lengths (left), growth trajectories of individuals captured eight times (middle) and growth rates (right, median = dot, 25th to 75th quantile = box, 2.5–97.5% quantile = whisker) across sampling occasions ('age'-'season', where season 1 = spring, 2 = summer, 3 = autumn, 4 = winter).

sampling occasion overlapped from age-0 through age-2 (Fig. 2 left). Growth rates were low ( $<0.1$  mm/day) except during the spring interval and for the youngest fish (Fig. 2 right). During spring, growth rates were high and variable (50% interval 0.2–0.4 mm/day). Individual length trajectories reflected the fast spring growth and slower growth during the rest of the year and also illustrated considerable variation in individual lengths (Fig. 2 middle).

### Pedigree

In total, available data for analysis consisted of 2862 observations of 1290 fish (Table 1). The maximum number of observations per individual was 10, the median was 2, and the mean was 2.2 (Fig. 3 above). The pedigree consisted of 90 parental families, of which 29 were represented by both parents and the remaining 61 were represented by one known parent. The median number of offspring per family was four, the mean was 6.6, and the maximum was 46 (Fig. 3 below).

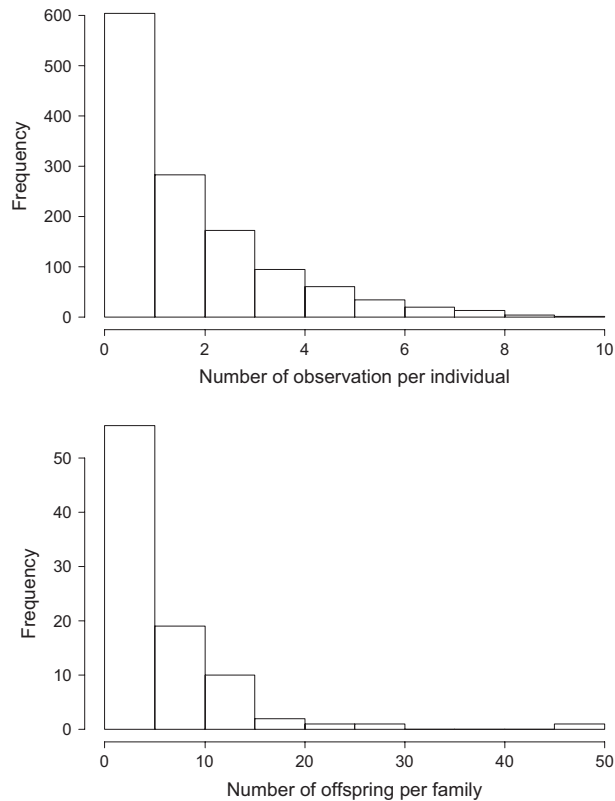
For genotype error assessment, complete genotypes were obtained for 91 of the 100 randomly selected individuals. Of these 91 individuals, four contained allele discrepancies between their two genotypes resulting in seven differing alleles. A single individual accounted for four of the differing alleles suggesting a process error for that individual. The per allele error rate was 0.32% (7/2184).

Sibship reconstruction and parentage assignment analyses performed on the synthetic datasets both indicated a high degree of power of the genetic panel to reconstruct full-sibling families accurately and assign parents. For reconstructed full-sibling families composed of at least two individuals, inferred families had a correct partition rate of 91.2% (0.7%) (SE), and assigned parents had an accuracy of 94.2% (0.6%). Accuracies for both methods improved as reconstructed full-sibling family size increased. For example, reconstructed full-sibling families composed of at least five individuals resulted in accuracies of 97.7% (0.4%) (sibship) and 96.1% (0.5%) (parentage).

As an additional validation of parentage assignment accuracy, known locations of parents during spawning were compared to natal rivers of assigned families for congruence. Of 101 assigned parents available for capture, 84 were detected during the spawning period that produced their assigned family. Of these 84, 76 were located in the natal river of the assigned family, resulting in a congruence rate of 90.5%.

### Quantitative genetics

Overall, estimates of repeatability and heritability indicated that the proportion of total phenotypic variance that could be ascribed to among-individual variation was from 1.5- to 2.5-fold larger for body size compared to growth (Table 2, see Fig. S1 for example MCMC traces



**Figure 3** Frequency distributions of the number of observations per individual (above) and the number of offspring per family (below).

and distributions). Repeatability estimates indicated that 73% of the overall phenotypic variation in body size could be ascribed to among-individual variation and that 29% of the variation in growth was attributable to among-individual variation. Heritability estimates were about 40% of repeatability estimates (Table 2).

Pairwise estimates of repeatability for length and growth in length reflected the overall lower repeatability for growth compared to body size for the pairs of samples for ages 1 and 2 analyzed using the animal model (highlighted boxes in Fig. 4). Repeatability was very high for adjacent sample pairs (e.g. pairs 0-4 and 1-1, or 1-3 and 1-4, Fig. 4, Table 3) for length ( $>0.9$ ) and was generally low for growth ( $<0.5$ ) during the slow growth periods. For nonadjacent sampling pairs, repeatability declined generally linearly as the interval between the pair of

samples increased (away from the diagonal in Fig. 4 and Table 3). For example, for the first sampling occasion, repeatability was 0.94 between the first and second occasions, but was 0.41 between the first and fifth occasions. In contrast to clear repeatability patterns in length, pairwise repeatability patterns in growth during the slow growth intervals were variable, inconsistent and often negative (Fig. 4). For adjacent growth intervals not including the fast growth intervals, the low repeatability probably reflects low potential for variation in individual growth rates. Examining repeatability of growth across years for the fast growth season (spring, interval pairs 1-2 and 2-2) reveals moderate repeatability (0.38, Fig. 4 and Table 3), similar to the repeatability estimate from the animal model for spring only (Table 2).

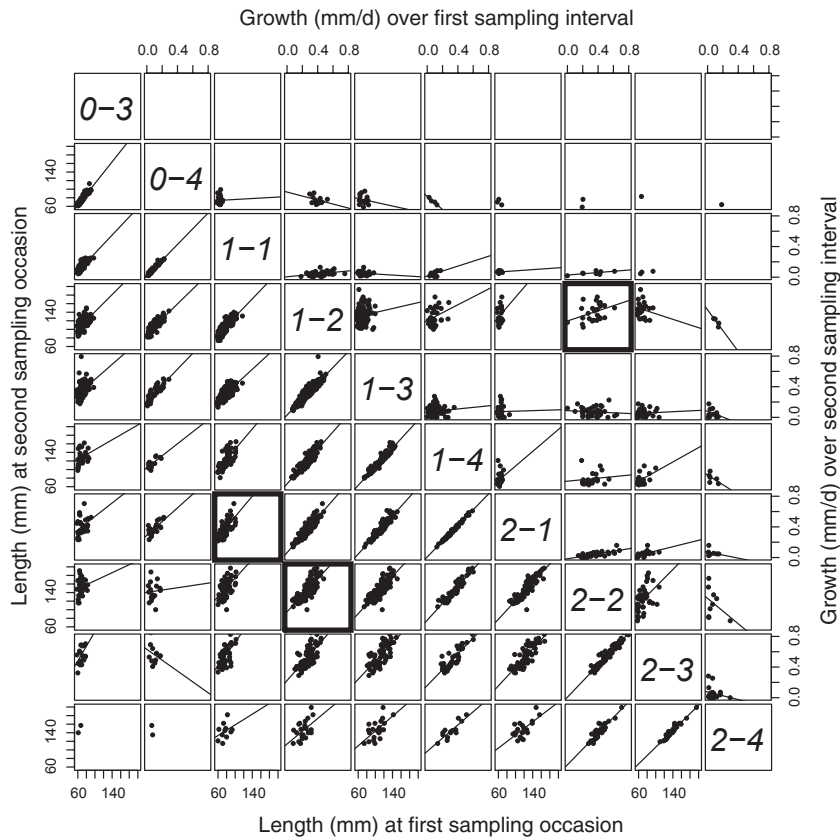
Genetic covariances between body size and growth rate were positive [0.39, 95% credible intervals (0.055: 0.61)], suggesting an additive genetic contribution to the relationship between body size and growth and that any evolutionary response will be in the same direction for length and growth. But the wide confidence intervals suggest other sources of variation may contribute and that the strength of effect may not be consistent over time. In addition to covariances across all intervals, we also estimated body size–growth rate covariances for each sampling interval (Table S2). In all but two intervals, 95% credible intervals overlapped 0, indicating no genetic covariance between body size and growth rate. In the 0-3 to 0-4 intervals and the 2-1 to 2-2 intervals, however, there was evidence for positive covariance. During these intervals, larger fish grew faster leading to growth depensation (Table S2). Linear models for the relationship between body size and growth reveal a similar pattern (Fig. 5); with strong positive size-dependent growth for the 0-3 to 0-4 interval, moderate positive size-dependent growth in interval 2-1 to 2-2 and weak size-dependent growth in all other intervals (Fig. 5).

### Size-dependent survival

We estimated size-dependent survival based on 2937 fish from three study cohorts. Goodness of fit indicated that assumptions of the multi-state model were not violated ( $P$ -value = 0.81,  $df = 123$ ). Model selection indicated important variation in survival among size states and

**Table 2.** Variance estimates ( $V_A$  = additive genetic variance,  $V_E$  = permanent environmental variance,  $V_e$  = residual variance) and resulting repeatabilities and heritabilities for body size and growth rate for length [95% credible interval].

	$V_A$	$V_E$	$V_e$	Repeatability	Heritability
Body size	0.054	0.099	0.063	0.73 [0.63: 0.78]	0.25 [0.13: 0.48]
Growth	0.052	0.082	0.33	0.29 [0.19: 0.40]	0.11 [0.055: 0.22]



**Figure 4** Pairwise repeatability relationships for body length (below diagonal) and growth in length (above diagonal). The numbers on the diagonal represent specific age/season sampling occasions, designated as ‘age’–‘season’ where season represents either the sampling occasion or the season at the end of the growth interval (1 = spring, 2 = summer, 3 = autumn, 4 = winter). Highlighted boxes represent sampling pairs included in the quantitative genetics analysis.

seasons (Table 4, Fig. 6 above). Over the spring interval, survival was similar among the smaller three size bins and was slightly higher for the largest fish. The opposite pattern was observed over the summer; survival was poorer for the larger fish. During autumn, survival was relatively low for the largest three size classes, likely a consequence of reproductive activities. Survival was size-independent during winter.

Aggregated over seasons, survival on a monthly time step varied from 0.85 to 0.88 among size states (Table 5, Fig. 6 below). Based on the confidence intervals of the beta parameters, survival for size states 1 and 2 did not differ from each other, and survival of size states 2, 3, and 4 did not differ (Table 5). Extending the monthly survival estimates to a full year indicates that survival differences would range from 0.13 to 0.22 among states.

**Discussion**

Our analysis indicates that most of the size variation in our study stream derives from early (age-0, autumn) size

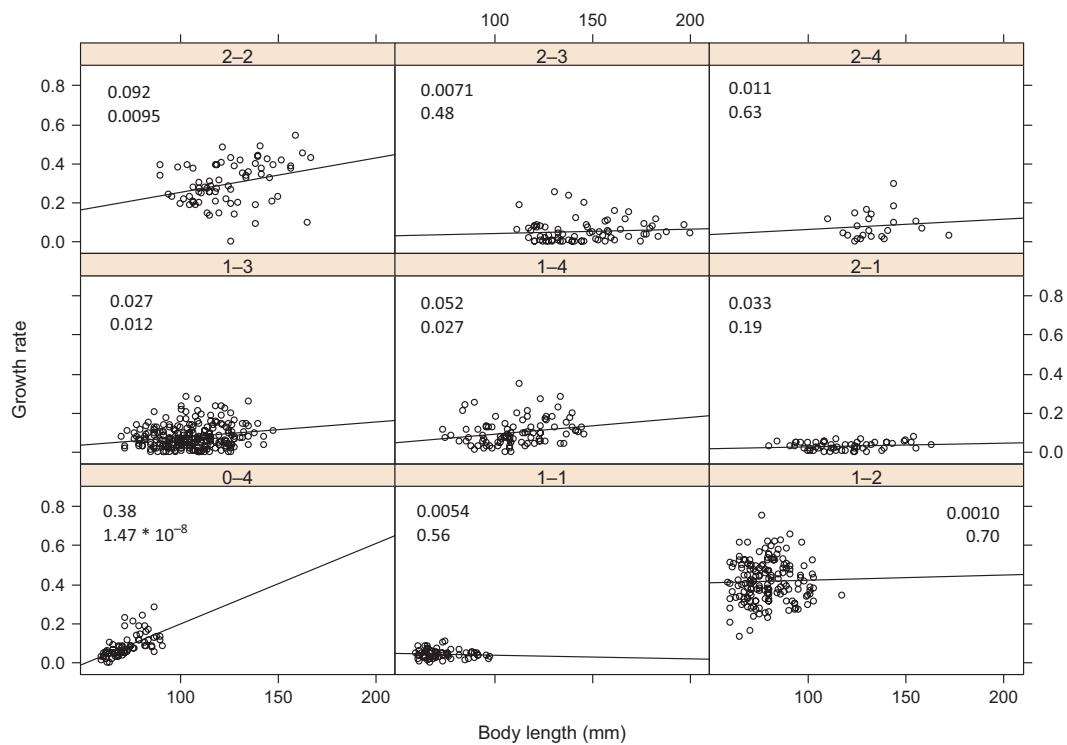
differences that are only moderately influenced by subsequent size-dependent processes. Size ranks, and to a lesser extent growth ranks, were generally maintained over time (leading to high repeatability), but there was no evidence for strong and consistent size-dependent growth and no consistent pattern of size-dependent survival. Size and growth were also highly (body length, 0.25) to moderately (growth in length, 0.11) heritable. This high heritability, combined with high repeatability of body size ranks over ontogeny, indicates the potential for environmental change to elicit an evolutionary response in body size. However, our results suggest this response will be mediated largely via effects on early size distributions, which are strongly influenced by maternal decisions on the timing and location of reproduction.

Based on BKT spawning tactics and habitats, it is not particularly surprising that there is wide variation in body size in early life. What is surprising is that these early sizes seem to be largely maintained over the course of a trout’s life. Variation in early body size (age-0, spring) depends on the variability of spawning time and location.



**Table 3.** Correlation coefficients for the relationship between pairs of sampling occasions for body length (below diagonal) and for pairs of sampling intervals for growth in length (above diagonal). This table follows the same format as Fig. 4. Sampling occasions (for body length) and the end of the sampling interval (for body growth) are indicated on the diagonal by ‘age’ – ‘season’, where season 1 = spring, 2 = summer, 3 = autumn, 4 = winter. NA indicates too few points for an estimate.

<b>0-3</b>									
0.94	<b>0-4</b>	0.022	-0.35	-0.15	-0.97	0.66	NA	NA	NA
0.82	0.97	<b>1-1</b>	0.41	-0.14	0.48	0.11	0.69	0.82	NA
0.63	0.87	0.85	<b>1-2</b>	0.12	0.31	0.24	0.38	-0.17	-0.87
0.41	0.86	0.76	0.92	<b>1-3</b>	0.12	0.014	-0.12	0.47	-0.55
0.29	0.78	0.70	0.90	0.96	<b>1-4</b>	0.26	0.12	0.47	-0.55
0.34	0.73	0.69	0.90	0.94	0.99	<b>2-1</b>	0.75	0.50	-0.26
0.16	0.067	0.54	0.79	0.82	0.94	0.90	<b>2-2</b>	0.48	-0.40
0.44	-0.28	0.63	0.71	0.73	0.86	0.85	0.96	<b>2-3</b>	-0.22
NA	NA	0.35	0.51	0.62	0.86	0.67	0.90	0.97	<b>2-4</b>



**Figure 5** Growth (mm/d) as a function of body size (mm) for age 0 through age 2 sampling intervals. Numbers in each panel are r-squares (above), and P-value (below) for linear regressions represented by the lines. Growth intervals labeled as in Fig. 2.

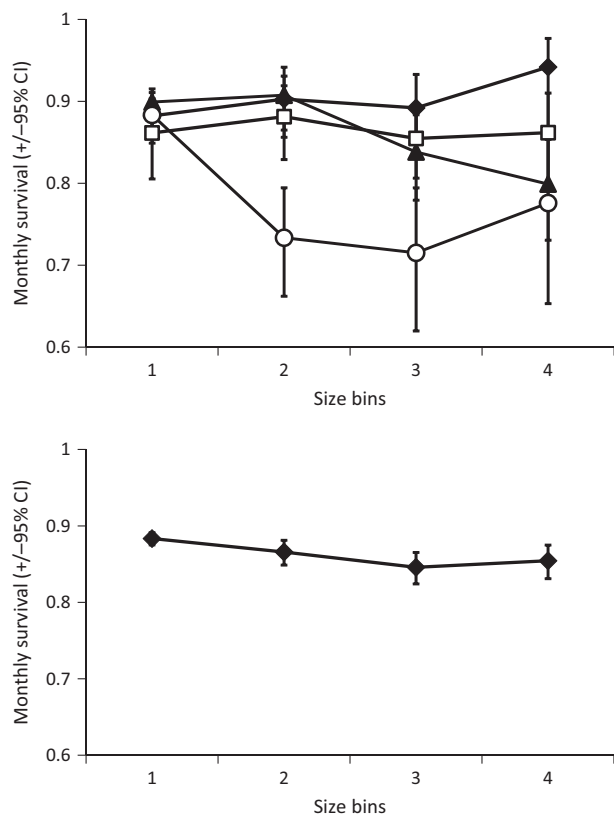
BKT mate in the autumn, embryos (alevins) incubate in stream-bottom gravels over the winter, and fry emerge in the spring. Fertilization date initiates the developmental clock, but development rates are modified by water temperature experienced by the alevin in the redd (egg nest). Because winter groundwater is usually at least a couple of degrees warmer than surface water (Power et al. 1999), the extent of groundwater infiltration into the redd can have a profound effect on developmental rates. Regardless of the exact mechanism driving emergence date, there is

wide variation (> 2 months) in emergence date both among (Curry et al. 1991; Snucins et al. 1992) and within (Curry et al. 1995) redds.

Observations on the timing of entry into the tributaries in the autumn in our study system indicate that variation in spawn date may explain some of the variation in emergence timing (Letcher, BH, Dubreuil, TD, and O’Donnell MJ, unpublished data). PIT tag antennas at the mouths of the tributaries indicate that movements into the tributaries increase sharply in late autumn and continue for

**Table 4.** Model selection data for the multi-state capture-mark-recapture model of size-dependent survival. Models included variation among size bins (size) and seasons (season) or no variation among sizes or seasons (-).

Model	Number of parameters	AIC	$\Delta$ AIC
Size, season	66	12671	–
Size, -	50	12718	47
-, season	51	12723	52
-, -	47	12736	65



**Figure 6** Survival estimates for the 'size, season' (above, spring = diamond, summer = triangle, autumn = circle, winter = square) and the 'size, -' (below) multi-state capture-mark-recapture models (Table 4).

about 4 weeks. If the month-long duration of movements into the tributaries represents spawn date, the range of actual spawning dates could also equal a month. The rapidly dropping stream temperature during this time (typically from 10 to 5°C) means that early-spawned alevins will experience warmer temperatures during early development than late-spawned alevins. The increased degree-days experienced by the early-spawned fish should lead to more rapid development rates as long as all fish

experience the average water temperature, exaggerating the effect of spawn date on early body sizes. As mentioned earlier, experienced temperatures may vary among redd locations owing to groundwater infiltration, and this variation may moderate (or exaggerate) the effect of spawn date on size distributions (Elliott and Hurley 1998). Detailed maps of temperature in spawning streams could be developed using new fiber-optic temperature sensing technologies that can provide extremely fine-scale (1 m, 15 min) temperature records for stream reaches as long as 5 km (e.g. Henderson et al. 2009). Further work relating spawning migration timing, actual spawning time, development rate, emergence dates and fry size distributions based on pedigrees, PIT tagging, PIT antennas and detailed temperature maps should help clarify sources of variation in fry size distributions.

Unless there is very strong growth compensation of newly-emerged fry, the wide range of emergence dates will produce broad size ranges in the first few months of life. In the WB, we observed a size range of 18–32 mm in April, at most a month after emergence (B.H. Letcher and M. Chapman, unpublished data). Other studies have found similarly wide size ranges for young BKT (McFadden 1961; Hutchings 1996), suggesting that a wide range of emergence dates may be common in BKT (Snucins et al. 1992). It is very difficult to track growth rates of individual fry, so it is unknown to what extent size-dependent growth may modify body sizes of young fish. For age-0 fish in the autumn, we observed strong growth depensation that will augment size differences among fish. It is possible that growth depensation prior to our age-0 autumn sampling occasion also exaggerated early size differences related to emergence date, but we have no data to assess this possibility. A likely mechanism for growth depensation is size-based competitive interactions between larger, early emergers and smaller late emergers (Curry et al. 1995; Letcher et al. 2004), although an unpredictable environment for early emergers combined with small body size of late emergers should provide stabilizing selection on emergence date (e.g. Brannon 1987).

We observed very high repeatability of body size following age-0 autumn (0.73), suggesting that early size differences could be largely maintained throughout life. This has several important implications. First, high repeatability indicates that size ranks are largely maintained over time. While we have no direct information, the most likely mechanism for maintaining size ranks in salmonids is the establishment of dominance hierarchies (Hughes 1992). Strong evidence for the importance of size-dependent dominance interactions structuring size distributions is provided by an analysis of Atlantic salmon growth. Early size differences generated by variation in the timing of stocking of 25-mm fry in our study area were maintained

**Table 5.** Monthly survival estimates and beta parameter (logit link) estimates for the 'size, ·' multi-state capture-mark-recapture model (Table 4). Beta value confidence intervals of states with the same letters in the final column overlap.

Size state	Survival				Beta				
	Value	SE	Lower 95% CI	Upper 95% CI	Value	SE	Lower 95% CI	Upper 95% CI	
1	0.88	0.004	0.88	0.89	2.03	0.04	1.95	2.10	A
2	0.87	0.008	0.85	0.88	1.87	0.07	1.73	2.01	AB
3	0.85	0.010	0.82	0.87	1.70	0.08	1.55	1.86	B
4	0.85	0.011	0.83	0.88	1.77	0.09	1.59	1.95	B

over the course of 2 years (Letcher et al. 2004). Laboratory studies from the same study also demonstrated that early-stocked fish could suppress sizes of late-stocked fish, suggesting that size-based competition made a large contribution to the maintenance of early size structure. Similarly, salmon stocked at high density (Ward et al. 2008) or emerging from high-density nests (Einum and Nislow 2005) exhibited a higher level of size variation than when stocked at low densities, further supporting the importance of size-based competition. At the same time, repeatability of size ranks indicates that processes that would narrow size distributions are insufficient to counter early-established ranges of variation. High individual growth variation could swamp size differences if growth was consistently compensatory over multiple intervals among individuals in a population or if a subset of individuals expressed strong compensatory growth (Metcalf and Monaghan 2001). We did not observe evidence for either possibility. Growth among individuals was actually depensatory during the first sampling interval (age-0, autumn), and there was limited evidence for size-dependent growth for any other interval. In the WB, compensatory growth appears to be insufficient to overcome size differences established by age-0 autumn.

Second, repeatability generally sets an upper limit to the estimates of heritability (Lessells and Boag 1987; Dohm 2002), suggesting the potential for high heritability of body size in our system. We estimated heritability (0.25) that was about one-third of the repeatability, indicating that one-third of the among-individual variation in length could be attributed to additive genetic effects. Our estimate of 0.25 for body length is similar to the median value of 0.29 for body length from a review of heritability estimates in salmonids (Carlson and Seamons 2008) and is also similar to a field estimate for BKT (Wilson et al. 2003), but is about one-half of the estimate for a population containing sea-run fish (Thériault et al. 2007). Further, high repeatability of body length suggests that repeat measures may not be necessary for heritability estimates of length. In contrast, like many behavioral traits (Bell et al. 2009), growth in our system had relatively low values of repeatability. When repeatability is low, single observations will not characterize an individual well and

repeat observations are required for reliable estimates of heritability.

Finally, because we observed high repeatability for body length and early (age-0, autumn) size differences were largely maintained over time, a large portion of heritability for length could actually represent heritability for processes that generate the early size differences rather than those affecting sizes for fish older than age-0. If this is the case, an evolutionary response to changing environmental conditions may not act primarily through body size itself, but more through variation in spawning timing and location. In many species of salmonids, there is strong evidence for additive genetic variation for spawning date, especially for anadromous species (Su et al. 1997; Hendry et al. 1999; Einum and Fleming 2000; Quinn et al. 2000; Hendry and Day 2005; Crozier et al. 2008). While little is known about the evolutionary biology of spawning date for BKT, our results suggest a focus on the relationships between autumn spawning date, developmental rate of alevins and emergence date. Further development of relationships between experienced water temperature and otolith microchemistry (Godidsken et al. 2010) of developing fish will be useful for sorting out relative contributions of fertilization date and developmental rate on early body size in BKT.

The wide observed size variation presents a template upon which size selection can act. We found evidence for seasonal variation in size selection, but variable strengths and directions resulted in limited directional selection over the course of the life of a BKT in our study stream. Strong environmental change (e.g. climate change or increased groundwater extraction) or increased anthropogenic pressure (e.g. size-selective fishing), however, could introduce strong selection. Both these sources will reduce survival of larger fish; climate change through poorer survival of large fish during dryer, warmer summers (Xu et al. 2010b), and fishing through the direct removal of larger fish (Naish and Hard 2008). If large fish produce large offspring (high heritability), large fish spawn first and produce early emergers (Doctor and Quinn 2009), and early emergers also end up large [high repeatability, (Letcher et al. 2004)], selection against large fish could result in a shift in average body size directly through

heritable pathways. Our results suggest that an important indirect pathway to an evolutionary response in body size may be variation in emergence timing, in addition to direct selection on body size of older fish. In this scenario, the critical, but largely unknown, link is between adult body size and emergence time. In anadromous species, larger fish tend to migrate up rivers earlier and spawn earlier (Doctor and Quinn 2009), but the relationship between body size and spawn time/emergence date has not been well studied in stream-dwelling populations of BKT. For example, we do not know whether BKT that are large either spawn early or spawn in locations that produce early emergence. Whatever the exact mechanism, reduction in variation of body size distributions acting through changes in emergence timing could have direct effects on population persistence by limiting contributions of large fish. Importantly, the reduction in variation of size distributions could also limit adaptability to future environmental challenges if loss of phenotypic variation reflects loss of genotypic variation.

Our results suggest that natural resource management needs to recognize that actions which affect variation in body size can affect variation in emergence timing, and vice versa. Management actions that affect body size are particularly important for species like BKT that have a very strong relationship between body size and fecundity. One example of how management actions could have unanticipated and delayed effects on body size acting through emergence date is stream flow regulation. In north temperate streams, salmonids emerge during a highly dynamic flow environment, with high among-year variation. As a result, the emergence time that best 'matches' the requirements of emerging fry varies from year to year (Armstrong and Nislow 2006). Flow regulation, depending on the timing and magnitude of spring releases, could select against early-emerging fish (Letcher et al. 2004; Armstrong and Nislow 2006) that are likely to achieve large body size. Another management target with likely strong ecological and evolutionary responses, size-dependent harvest (Conover and Munch 2002; Olsen et al. 2004; Thériault et al. 2008), not only will directly reduce body size range of older fish, but it could also indirectly limit the range of emergence dates if larger fish spawn earlier. If body sizes of older fish are, in fact, highly correlated with emergence dates and there is body size-related adaptive variation for emergence date, the combined effect of loss of larger fish with truncation of emergence dates could magnify anticipated evolutionary responses to size-dependent harvest in BKT. These considerations suggest that maintenance of variation in emergence timing will be critical for enhancing future probabilities of BKT population persistence in streams.

## Acknowledgements

We thank Matt O'Donnell, Todd Dubreuil, Tyler Evans and many field assistants. Michael Morrissey and Krzysztof Sakrejda-Leavitt provided assistance with heritability estimates. The USFS Northern Research Station, the USGS Conte Anadromous Fish Research Center, and The Nature Conservancy's Connecticut River program provided funding for the project.

## Literature cited

- Almudevar, A. 2007. A graphical approach to relatedness inference. *Theoretical population biology* 71:213–229.
- Armstrong, J. D., and K. H. Nislow. 2006. Critical habitat during the transition from maternal provisioning in freshwater fish, with emphasis on Atlantic salmon (*Salmo salar*) and brown trout (*Salmo trutta*). *Journal of Zoology* 269:403–413.
- Bell, A. M., S. J. Hankison, and K. L. Laskowski. 2009. The repeatability of behaviour: a meta-analysis. *Animal Behaviour* 77:771–783.
- Bolker, B. M., M. E. Brooks, C. J. Clark, S. W. Geange, J. R. Poulsen, M. H. H. Stevens, and J. S. White. 2009. Generalized linear mixed models: a practical guide for ecology and evolution. *Trends in ecology & evolution* 24:127–135.
- Brannon, E. L. 1987. Mechanisms stabilizing salmonid fry emergence timing. *Canadian Special Publication of Fisheries and Aquatic Sciences* 96:120–124.
- Carlson, S. M., and T. R. Seamons. 2008. A review of quantitative genetic components of fitness in salmonids: implications for adaptation to future change. *Evolutionary Applications* 1:222–238.
- Choquet, R., A. M. Reboulet, R. Pradel, O. Gimenez, and J. D. Lebreton. 2004. M – SURGE: new software specifically designed for multistate capture – recapture models. *Animal Biodiversity and Conservation* 1:207–215.
- Choquet, R., J.-D. Lebreton, O. Gimenez, A.-M. Reboulet, and R. Pradel. 2009. U-CARE: utilities for performing goodness of fit tests and manipulating capture recapture data. *Ecography* 32:1071–1074.
- Conover, D. O., and S. B. Munch. 2002. Sustaining fisheries yields over evolutionary time scales. *Science* 297:94–96.
- Coombs, J. A., B. H. Letcher, and K. H. Nislow. 2010a. PEDAGOG: software for simulating eco-evolutionary population dynamics. *Molecular Ecology Resources* 10:558–563.
- Coombs, J. A., B. H. Letcher, and K. H. Nislow. 2010b. PedAgree: software to quantify error and assess accuracy and congruence for genetically reconstructed pedigree relationships. *Conservation Genetics Resources* 2:147–150.
- Crozier, L. G., A. P. Hendry, P. W. Lawson, T. P. Quinn, N. J. Mantua, J. Battin, R. G. Shaw et al. 2008. Potential responses to climate change in organisms with complex life histories: evolution and plasticity in Pacific salmon. *Evolutionary Applications* 1:252–270.
- Curry, R. A., P. M. Powles, J. M. Gunn, and V. A. Liimatainen. 1991. Emergence chronology of brook charr, *Salvelinus fontinalis*, alevins in an acidic stream. *Environmental Biology of Fishes* 31:25–31.
- Curry, R. A., D. L. G. Noakes, and G. E. Morgan. 1995. Groundwater and the incubation and emergence of brook trout (*Salvelinus fontinalis*). *Canadian Journal of Fisheries and Aquatic Sciences* 52:1741–1749.

- Doctor, K. K., and T. P. Quinn. 2009. Potential for adaptation-by-time in sockeye salmon (*Oncorhynchus nerka*): the interactions of body size and in-stream reproductive life span with date of arrival and breeding location. *Canadian Journal of Zoology* **87**:708–717.
- Dohm, M. R. 2002. Repeatability estimates do not always set an upper limit to heritability. *Functional Ecology* **16**:273–280.
- Einum, S., and I. A. Fleming. 2000. Selection against late emergence and small offspring in Atlantic salmon (*Salmo salar*). *Evolution* **54**:628–639.
- Einum, S., and K. H. Nislow. 2005. Local-scale density-dependent survival of mobile organisms in continuous habitats: an experimental test using Atlantic salmon. *Oecologia* **143**:203–210.
- Elliott, J. M., and M. A. Hurley. 1998. An individual-based model for predicting the emergence period of sea trout in a Lake District stream. *Journal of fish Biology* **53**:414–433.
- Falconer, D. S., and T. F. C. Mackay. 1996. *Introduction to Quantitative Genetics*, 4th edn. Longman Group, Essex, UK.
- Garant, D., L. E. B. Kruuk, T. A. Wilkin, and R. H. McCleery. 2005. Evolution driven by differential dispersal within a wild bird population. *Nature* **433**:60–64.
- Godsidsken, J. A., M. A. Svenning, J. B. Dempson, M. Martilla, A. Storm-Suke, and M. Power. 2010. Development of a species-specific fractionation equation for Arctic charr [*Salvelinus alpinus* (L.)]: an experimental approach. *Hydrobiologia* **650**:67–77.
- Gries, G., and B. H. Letcher. 2002. Tag retention and survival of Age-0 Atlantic Salmon following surgical implantation with passive integrated transponder tags. *North American Journal of Fisheries Management* **22**:219–222.
- Henderson, R. D., F. Day-Lewis, and C. F. Harvey. 2009. Investigation of aquifer-estuary interaction using wavelet analysis of fiber-optic temperature data. *Geophysical Research Letters* **36**:L06403.
- Hendry, A. P., and T. Day. 2005. Population structure attributable to reproductive time: isolation by time and adaptation by time. *Molecular ecology* **14**:901–916.
- Hendry, A. P., O. K. Berg, and S. S. Qian. 1999. Condition dependence and adaptation-by-time: breeding date, life history, and energy allocation within a population of salmon. *Oikos* **85**:499–514.
- Hestbeck, J. B., J. D. Nichols, and R. A. Malecki. 1991. Estimates of movement and site fidelity using mark-resight data of wintering Canada geese. *Ecology* **72**:523.
- Hudy, M., T. M. Thieling, N. Gillespie, and E. P. Smith. 2008. Distribution, status, and land use characteristics of subwatersheds within the native range of Brook Trout in the Eastern United States. *North American Journal of Fisheries Management* **28**:1069–1085.
- Hughes, N. F. 1992. Ranking of feeding positions by drift-feeding Arctic Grayling (*Thymallus arcticus*) in dominance hierarchies. *Canadian Journal of Fisheries and Aquatic Sciences* **49**:1994–1998.
- Hutchings, J. A. 1996. Adaptive phenotypic plasticity in brook trout, *Salvelinus fontinalis*, life histories. *Ecoscience* **3**:25–32.
- King, T. L., S. E. Julian, R. L. Coleman, and M. K. Burnham-Curtis. 2003. Isolation and characterization of novel tri- and tetra-nucleotide microsatellite DNA markers for brook trout *Salvelinus fontinalis*. Genbank submission numbers: AY168186, AY168187, AY168189, AY168191, AY168192, AY 168193, AY168194, AY168195, AY168197, AY168198, AY168199. Available at <http://www.ncbi.nlm.nih.gov/nucleotide/>.
- King, T. L., M. S. Eackles, and B. H. Letcher. 2005. Microsatellite DNA markers for the study of Atlantic salmon (*Salmo salar*) kinship, population structure, and mixed-fishery analyses. *Molecular Ecology Notes* **5**:130–132.
- Kruuk, L. E. B. 2004. Estimating genetic parameters in natural populations using the “animal model”. *Philosophical transactions of the Royal Society of London. Series B, Biological sciences* **359**:873–890.
- Kruuk, L. E. B., and J. D. Hadfield. 2007. How to separate genetic and environmental causes of similarity between relatives. *Journal of evolutionary biology* **20**:1890–1903.
- Kruuk, L. E. B., J. Slate, and A. J. Wilson. 2008. New answers for old questions: the evolutionary quantitative genetics of wild animal populations. *Annual Review of Ecology Evolution and Systematics* **39**:525–548.
- Lebreton, J. D., and R. Pradel. 2002. Multistate recapture models: modelling incomplete individual histories. *Journal of Applied Statistics* **29**:353–369.
- Lessells, C. M., and P. T. Boag. 1987. Unrepeatable repeatabilities: a common mistake. *The Auk* **104**:116–121.
- Letcher, B. H., and G. E. Horton. 2008. Seasonal variation in size-dependent survival of juvenile Atlantic salmon (*Salmo salar*): performance of multistate capture-mark-recapture models. *Canadian Journal of Fisheries and Aquatic Sciences* **65**:1649–1666.
- Letcher, B. H., G. Gries, and F. Juanes. 2002. Survival of stream-dwelling Atlantic Salmon: effects of life history variation, season, and age. *Transactions of the American Fisheries Society* **131**:838–854.
- Letcher, B. H., T. L. Dubreuil, M. J. O'Donnell, M. Obedzinski, K. Griswold, and K. H. Nislow. 2004. Long-term consequences of variation in timing and manner of fry introduction on juvenile Atlantic salmon (*Salmo salar*) growth, survival, and life-history expression. *Canadian Journal of Fisheries and Aquatic Sciences* **61**:2288–2301.
- Letcher, B. H., K. H. Nislow, J. A. Coombs, M. J. O'Donnell, and T. L. Dubreuil. 2007. Population response to habitat fragmentation in a stream-dwelling brook trout population. *PLoS ONE* **2**:e1139.
- McFadden, J. T. 1961. A population study of the brook trout, *Salvelinus fontinalis*. *Wildlife Monographs* **7**:3–73.
- McGuigan, K. 2006. Studying phenotypic evolution using multivariate quantitative genetics. *Molecular ecology* **15**:883–896.
- Metcalfe, N. B., and P. Monaghan. 2001. Compensation for a bad start: grow now, pay later. *Trends in Ecology and Evolution* **16**:254–260.
- Naish, K. A., and J. J. Hard. 2008. Bridging the gap between the genotype and the phenotype: linking genetic variation, selection and adaptation in fishes. *Fish and Fisheries* **9**:396–422.
- Olsen, E. M., M. Heino, G. R. Lilly, M. J. Morgan, J. Brattey, and B. Ernande, U. Dieckmann. 2004. Maturation trends indicative of rapid evolution preceded the collapse of northern cod. *Nature* **428**:4–7.
- Pelletier, F., T. Clutton-Brock, J. Pemberton, S. Tuljapurkar, and T. Coulson. 2007. The evolutionary demography of ecological change: linking trait variation and population growth. *Science* **315**:1571–1574.
- Pemberton, J. M. 2008. Wild pedigrees: the way forward. *Proceedings of Biological sciences/The Royal Society* **275**:613–621.
- Power, G., R. S. Brown, and J. G. Imhof. 1999. Groundwater and fish – insights from northern North America. *Hydrological processes* **13**:401–422.
- Quinn, T. P., M. J. Unwin, and M. T. Kinnison. 2000. Evolution of temporal isolation in the wild: genetic divergence in timing of migration and breeding by introduced chinook salmon populations. *Evolution* **54**:1372–1385.
- Rodriguez-Munoz, R., A. Bretman, J. Slate, C. A. Walling, and T. Tregenza. 2010. Natural and sexual selection in a wild insect population. *Science* **328**:1269–1272.
- Skelly, D. K., L. N. Joseph, H. P. Possingham, L. K. Freidenburg, T. J. Farrugia, M. T. Kinnison, and A. P. Hendry. 2007. Evolution-

- ary responses to climate change. *Conservation Biology* **21**:1353–1355.
- Snucins, E. J., R. A. Curry, and J. M. Gunn. 1992. Brook trout (*Salvelinus fontinalis*) embryo habitat and timing of alevin emergence in a lake and a stream. *Canadian Journal of Zoology* **70**:423–427.
- Su, G., L. Liljedahl, and G. A. E. Gall. 1997. Genetic and environmental variation of female reproductive traits in rainbow trout (*Oncorhynchus mykiss*). *Aquaculture* **154**:115–124.
- Thériault, V., D. Garant, L. Bernatchez, and J. J. Dodson. 2007. Heritability of life-history tactics and genetic correlation with body size in a natural population of brook charr (*Salvelinus fontinalis*). *Journal of evolutionary biology* **20**:2266–2277.
- Thériault, V., E. S. Dunlop, U. Dieckmann, L. Bernatchez, and J. J. Dodson. 2008. The impact of fishing-induced mortality on the evolution of alternative life-history tactics in brook charr. *Evolutionary Applications* **1**:409–423.
- Wang, J. L. 2004. Sibship reconstruction from genetic data with typing errors. *Genetics* **166**:1963–1979.
- Ward, D. M., K. H. Nislow, and C. L. Folt. 2008. Predators reverse the direction of density dependence for juvenile salmon mortality. *Oecologia* **156**:515–522.
- Wilson, A. J., J. A. Hutchings, and M. M. Ferguson. 2003. Selective and genetic constraints on the evolution of body size in a stream-dwelling salmonid fish. *Journal of evolutionary biology* **16**:584–594.
- Wilson, A. J., D. Réale, M. N. Clements, M. M. Morrissey, E. Postma, C. A. Walling, L. E. B. Kruuk *et al.* 2010. An ecologists guide to the animal model. *Journal of Animal Ecology* **79**:13–26.
- Xu, C. L., B. H. Letcher, and K. H. Nislow. 2010a. Context-specific influence of water temperature on brook trout growth rates in the field. *Freshwater Ecology* **55**:2253–2264.
- Xu, C. L., B. H. Letcher, and K. H. Nislow. 2010b. Size-dependent survival of brook trout *Salvelinus fontinalis* in summer: effects of water temperature and stream flow. *Journal of Fish Biology* **76**:2342–2369.
- Zydlewski, G. B., G. Horton, T. L. Dubreuil, B. H. Letcher, S. Casey, and J. Zydlewski. 2006. Remote monitoring of fish in small streams: a unified approach using PIT tags. *Fisheries* **31**:492–502.

## Supporting Information

Additional Supporting Information may be found in the online version of this article:

**Figure S1.** Traces and densities of body length (X1) and growth rate in length (X2) for repeatabilities (above) and heritabilities (below).

**Table S1.** Single locus summary statistics for young-of-year brook trout captured in the West Brook stream complex.

**Table S2.** Genetic covariances between growth rate in length and body size at the beginning of the growth interval (2.5% and 97.5% credible interval).

Please note: Wiley-Blackwell are not responsible for the content or functionality of any supporting materials supplied by the authors. Any queries (other than missing material) should be directed to the corresponding author for the article.