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Genetic and maternal variation in early growth in the Atlantic silverside *Menidia menidia*

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ABSTRACT: In the early life histories of many fish species, larger individuals have lower mortality rates and, as a consequence, growth is thought to be a primary determinant of recruitment. Although much is known about environmental influences on growth in the early life history, far less is known about genetic and maternal contributions. As the relative contributions of maternal, genetic, and environmental effects determine how populations respond to selective harvest, evaluating their magnitudes is an important step in constructing evolutionarily sustainable harvest strategies. Using Atlantic silversides *Menidia menidia* as a model organism, we conducted a full-factorial breeding study to measure maternal and genetic contributions to size at age through the first 2 wk of life. A total of 418 full- and half-sib families were raised in a common environment, and body lengths were recorded at 1, 5, 10, and 15 d post-hatch. Maximum likelihood was used to estimate the genetic and maternal variance components. Heritability for size at age was between 0.10 and 0.25, and increased with age. At hatch, non-genetic maternal contributions were ~7 times greater than the genetic contributions to size, but decreased with age. These significant genetic and maternal contributions to size at age indicate substantial potential to respond to selection, with subsequent population dynamic consequences.

KEY WORDS: Heritability · Maternal effect · Growth rate · Life history · Evolution · Recruitment · Juvenile growth · Body size

INTRODUCTION

Mortality rates experienced in the early life history of fishes are exceedingly high and it is not uncommon that only 0.01 to 0.1% of fish that hatch survive to Age 1 (Houde 1989). Therefore, the number of individuals that survive the early life history is a primary determinant of year-class strength and a dominant driver of recruitment in fish populations (see, e.g., Hjort 1914, Gulland 1965, Anderson 1988). Although in some cases larger size results in increased predation risk due, for example, to increased detectability (e.g. Rice et al. 1993, Cushing & Horwood 1994), mortality typically declines rapidly as size increases (Miller et al. 1988, Pepin 1991). In keeping with this, Perez & Munch (2010) found that 77% of the selection differentials for larval and juvenile fish are positive, indicating that larger body size generally favors early survival. Moreover, they found that the median selection differential for size in fish was more than 5 times larger than selection on size in terrestrial taxa. Thus, the early life history of fish is a period of intense selection on size, and variation in growth through the early life history is critical to determining recruitment success (reviewed by Houde 1997).

Although extrinsic influences on growth, such as temperature and food availability, are well documented (see, e.g., Hjort 1914, Buckley et al. 1984, Sogard 1992), far less is known about maternal (e.g. Berkeley et al. 2004) and genetic (e.g. Páez et al. 2010) contributions to growth in marine fishes. As

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growth is tied to early survival and parental contributions to growth can be modified by non-random removal of adults, fisheries may indirectly influence recruitment (Jennings et al. 1998, Munch et al. 2005, Kuparinen et al. 2009). Understanding the relative magnitudes of maternal and genetic contributions to growth is therefore relevant to the design of evolutionarily sustainable harvest strategies (Law 2007, Enberg et al. 2009).

Non-genetic 'maternal influences' on size have been described in many fish species (e.g. Berg et al. 2001, Trippel et al. 2005). Larger or older mothers produce larger, better-provisioned eggs (Sargent et al. 1987, Monteleone & Houde 1990, Uusi-Heikkilä et al. 2012), and offspring with larger size at hatch (Blaxter & Hempel 1963) and greater viability (Berkeley et al. 2004). Consequently, larger or older females may contribute disproportionately to recruitment (Birkeland & Dayton 2005, Sogard et al. 2008). In support of this, time-series analyses indicate that changes in population age structure contribute to fluctuations in population dynamics (Hidalgo et al. 2011, Shelton et al. 2012). Several other studies investigating the demographic relevance of maternal effects in fishes have concluded that harvesting large, old females may increase the risk of population collapse (McCormick 1999, Berkeley et al. 2004).

We note that few studies that demonstrate size-dependent maternal effects account for genetic differences among females (but see Kokita 2003). Moreover, in the fish literature, the term 'maternal effect' seems to be virtually synonymous with size- or age-dependent offspring quality. However, non-genetic maternal contributions to offspring that are not driven directly by size are common in terrestrial taxa (reviewed by Mousseau & Fox 1998), raising the possibility that there are size-independent maternal effects in fishes as well (Heath & Blouw 1998).

Although quantitative genetic analyses of adult size are common for aquaculture species (see reviews by Gjedrem 1983, Law 2000), there are relatively few studies of the genetic contributions to early growth in marine fishes. Common garden experiments reveal ubiquitous genetic differentiation among populations (see review by Conover et al. 2009), but do not measure the genetic variation within populations. There are a handful of studies that explicitly demonstrate genetic variation within populations in early growth in marine teleosts (Green & McCormick 2005, Shimada et al. 2007, Ma et al. 2008, Johnson et al. 2010). However, except for one study (Johnson et al. 2010), the sample sizes used were quite small, on the order of tens of families. As quantitative genetic analyses typically require several hundred families to obtain precise estimates (Falconer & Mackay 1996), these sample sizes are enough to demonstrate significance but not enough to partition variation among genetic, maternal, and environmental components. As environmental factors such as temperature and food availability (Heath 1992, Houde & Zastrow 1993) are so important in the early life history, it may well be that the heritability of early growth is quite small (e.g. Heath & Blouw 1998).

Thus, for most marine fish species, the relative magnitude of parental effects in the early life history is unknown. The lack of information makes it almost impossible to determine how populations respond to selective harvest and construct evolutionarily sustainable harvest strategies. To fill in this gap, we used Atlantic silversides *Menidia menidia* as a model system to estimate genetic and maternal contributions to size at age over the first 2 wk of life. As the majority of previous studies on maternal effects in fishes have focused on the influence of female size, we also estimated the correlation between adult size and the mean size of their offspring.

The Atlantic silverside *Menidia menidia* is a marine fish common in North American estuaries from Newfoundland to northeast Florida (Johnson 1974). *M. menidia* is an annual fish; less than 10% survive to a second year (Conover & Ross 1982). Mortality in the juvenile period is size-selective (Juanes & Conover 1995), as is mortality occurring over the winter (Munch et al. 2003). Although several studies have shown that there is genetic variation in growth within (Conover & Munch 2002) and among (Present & Conover 1992, Billerbeck et al. 2001) populations, there are, as yet, no estimates of heritability for growth in the early life history, and it is unknown whether *M. menidia* exhibits maternal effects. However, in a multigeneration selection experiment, Walsh et al. (2006) showed that egg size changed in parallel with evolutionary changes in adult size, which could be indicative of size-dependent maternal provisioning.

MATERIALS AND METHODS

Fish were collected during the peak of the breeding season (end of May to beginning of June) from each of 2 isolated sites: 2 sites on the north shore of Long Island (Poquott, East Setauket, New York and Flax Pond, Old Field, New York: 40° 57′ 49″ N, 73° 8′ 19″ W) and 2 south shore sites (Great South Bay

and Shinnecock Bay, New York: 40° 51′ 10″ N, 72° 29′ 27″ W). Only a limited number of migrants are exchanged each year between the northern and southern sites (Clarke et al. 2010), ensuring that parents from disparate locations are distantly related, if at all. Adults were transported back to the Flax Pond Marine Lab, Old Field, New York (FPML), where they were housed overnight in separate tanks and strip-spawned the following day.

Fish breeding and rearing

Relatively large numbers of families are needed to estimate heritability. In the quantitative genetics literature, typical breeding studies have hundreds to thousands of families (Klein et al. 1973) and smaller studies often fail to identify significant heritability where it is likely to exist (Klein et al. 1973). This is particularly problematic when measuring size at age in the early life history of fish, as genetic differences in growth have had little time to accumulate. However, space constraints prevented us from rearing the required number of families simultaneously, so the study was carried out in 4 batches using adults collected on 8 June 2005, 1 May 2007, 1 June 2007, and 24 May 2008. As described below, the data from these 4 batches were analyzed simultaneously while statistically accounting for batch effects.

Each of the 4 spawning batches consisted of several complete-factorial blocks in which all males within a block were mated with all females. No parents were used in more than one block and, to limit relatedness among parents, males were only mated to females from the alternative collecting site.

To create each family block, eggs were stripped from a female and distributed across several Petri dishes lined with fiberglass screening and a shallow layer of seawater. At the same time, milt from each male was stripped into a small beaker and diluted with UV sterilized seawater. Milt from each sire was then distributed among the Petri dishes for each female, such that within a block all males were mated with all females. After allowing 20 min for the fertilized eggs to harden, eggs from each family were transferred to an aerated 18 l bucket immersed in a previously designated seawater bath. To avoid possible confounding of family and bath effects, families were assigned to baths in a stratified-random manner such that each family block was guaranteed to occur in multiple baths.

The numbers of fish of each sex varied between field collections, leading to differences in the breeding design among batches. Table 1 reports the number of sires and dams per block for each batch as well as the total number of blocks per batch. Note that the total number of families (Table 1) analyzed is always less than the maximum (sires × dams × blocks) due to unsuccessful strip-spawning or limited hatching success. We restricted our analysis to families that produced at least 200 offspring, resulting in a total of 418 families (Table 1).

Throughout the experiment, the seawater baths were maintained at 21 ± 1.2°C. Following hatching, fish were fed to satiation daily using a combination of dry food (Otohime larval feeds, Reed Mariculture) and freshly hatched *Artemia nauplii* (Brine Shrimp Direct). Further details of the rearing protocol are described in Present & Conover (1992).

Length measurements

In each batch, fish were measured on days 1, 5, 10, and 15 post-hatch. The sole exception to this is that in batch 4 the first measurement occurred on Day 2. Over the 4 spawning batches we used several different approaches to obtaining lengths while we endeavored to find the most efficient means of measuring thousands of fish. Fish were measured to the nearest ±0.5 mm using rulers (batch 1), digital photography (batches 2 and 3), or calipers (batch 4). Specifically, a 100 megapixel digital camera (Canon 40D with Canon 60mm Macro lens) was used to photograph the fish from a fixed height of 55 cm at a shutter speed of 1/250 s while the fish were held in a Petri dish with a shallow layer of water. Images were then measured in Image Pro Plus 6.0 (Media Cybernetics). Repeated measurements of 100 individuals indicated that there are no systematic biases in any of the measuring approaches and that they each had comparable levels of precision.

Table 1. Breeding design and sample sizes for each of the 4 Atlantic silverside spawning batches analyzed. N_s and N_d are the number of sires and dams, respectively, used to construct each complete factorial block. N_b is the total number of blocks reared in the batch and F is the number of surviving families that is included in the analysis. The number of individuals measured from each family at each time point is indicated by n

Batch	N_s	N_d	N_b	F	n
1	11	5	1	41	10
2	3	4	10	97	20
3	3	10	5	147	20
4	3	5	11	133	20

Statistical analysis

Heritability is the proportion of the phenotypic variance that is genetic (Falconer & Mackay 1996), which is usually measured through the resemblance among relatives. The statistical approaches used are well developed and there are many good textbooks on quantitative genetics (e.g. Falconer & Mackay 1996, Lynch & Walsh 1998). The standard approach uses a general linear mixed model to partition phenotypic variance into fixed effects, random genetic effects, and 'environmental' noise (Lynch & Walsh 1998). Specifically, the size at age (y_{sdi}) of the ith offspring of sire s and dam d was modeled as:

$$y_{sdi} = \beta_{1,sdi} + \beta_{2,sdi} + \tfrac{1}{2}(g_{si} + g_{di}) + \varepsilon_{sdi} \quad (1)$$

where $\beta_{1,sdi}$ and $\beta_{2,sdi}$ represent the influence, respectively, of spawning batch and seawater bath on size at age and are treated as fixed effects with bath nested within batch. The final term in Eq. (1), ε_{sdi} represents the variation in mean size at age not accounted for by the other terms and is assumed to be normally distributed with mean zero and variance V_ε. In this model, sire s and dam d contribute effects g_{si} and g_{di} to the mean size of their offspring. In the parlance of quantitative genetics, these are known as the 'breeding values' for the sire and dam respectively. Both g_{si} and g_{di} were assumed to follow a normal distribution with mean zero and variances V_s and V_d, respectively. In this analysis, g_{si} represents the genetic contribution (to size) of the sire, while g_{di} represents both genetic and non-genetic maternal contributions. The variance terms were the focal point of this analysis, as the heritability is given by the ratio of V_s to the total variance in size at age. The difference, $V_d - V_s$, provided a measure of the variance due to maternal effects, V_M (Lynch & Walsh 1998, Falconer & Mackay 1996). The proportion of variation in size due to maternal effects is given by V_M/V_p, with V_p being the total phenootypic variance calculated as $1/4(V_s + V_d) + V$ (Lynch & Walsh 1998).

In our experiment, it was not possible to track individual fish, so we extended the standard model to partition variability in the mean size at age among replicate buckets from the same family. Specifically, we modeled the mean size at age for the jth bucket, $\bar{y}_{sdj}$, as:

$$\bar{y}_{sdj} = \beta_{1,sdj} + \beta_{2,sdj} + (g_{sj} + g_{dj})/2 + \bar{\varepsilon}_{sdj} \quad (2)$$

where the fixed and random effect terms are as in Eq. (1). Here, the noise term $\bar{\varepsilon}_{sdj}$ is assumed to follow a normal distribution with mean zero and variance V_s/n where n is the number of measured individuals within a bucket. To estimate parameters for this model, we adopted a maximum likelihood approach. Further details on model specification are given in Appendix 1.

To test for differences in variance among sires and dams, we specified an alternative model in which both sire and dam variances are equal, i.e. $V_s = V_d = V_g$. The remainder of the model is otherwise the same. We used Akaike's information criteria (AIC) (Akaike 1974) for model selection (Appendix 1). To test whether the estimated variance components and heritabilities had a significant temporal trend, we regressed the maximum likelihood estimates against age using standard linear regression.

Although this analysis provided us with the means to partition variability in size at age among genetic and maternal effects, it did so without respect to parent size. As offspring quality is a function of mother's size in other species, we tested whether maternal effects in silversides can be predicted from size by regressing mean offspring size on the size of each parent after controlling for fixed effects of batch and seawater bath. We note that this analysis is equivalent to estimating the co-heritability between offspring size and parent size (Falconer & Mackay 1996). Because parental lengths were not available for 2005, this analysis was restricted to data from batches 2, 3 and 4.

RESULTS

We measured size at ages 1, 5, 10, and 15 d for 418 families (Fig. 1) and a total of 7950 fish overall. Over this time, linear regression explained >91% of

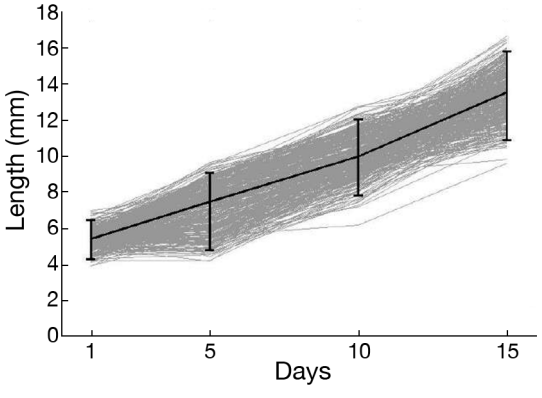

Fig. 1. *Menidia menidia*. Standard length of Atlantic silversides during the first 2 wk of life. The trajectories in grey indicate the mean size at age for each family. The solid line is the overall average trajectory. Error bars indicate a 95% confidence interval on size at each age

the variation in mean size at age within families. Therefore, the growth rate over this period is approximately constant (and therefore independent of body size) with a mean (±SE) of 0.98 ± 0.04 mm d^{-1} ($p < 0.001$).

We used maximum likelihood to estimate the variance components (V_d, V_s, and V_p; Table 2), and found evidence of significant genetic and maternal effects. The dam variance (V_d) was between 0.45 and 2.08 and was always higher than that from sires, which ranged from 0.06 and 0.84 (Fig. 2). Moreover, the model with distinct sire and dam variance components was favored by AIC at all ages (Table 3). Values of ΔAIC greater than 2 are typically considered significant (Burnham & Anderson 2002) and indicate strong evidence of maternal effects for all ages.

All of the variance components increased with age (Fig. 2). In support of this qualitative outcome, regressing the estimated variance components against age produced significant slopes ($p < 0.05$) for all components. Interestingly, the genetic variances increased fastest such that heritability (V_s/V_p) increased with age (Fig. 3) and resulted in a significantly larger heritability at Day 15 than the heritability on Day 1 ($p < 0.01$). The proportion of variation due to maternal effects (V_M/V_p) showed no trend with age ($p = 0.3$).

To test whether maternal effects were driven by the mother's size, we regressed mean size at age of offspring on mother's size, after controlling for fixed effects. We repeated this analysis with father's size as well. However, only one of the slope estimates was significantly different from zero: length at Day 15 significantly decreased ($p < 0.001$) with mother's length at a rate of −0.016 ± 0.003 mm (offspring) per mm (dam).

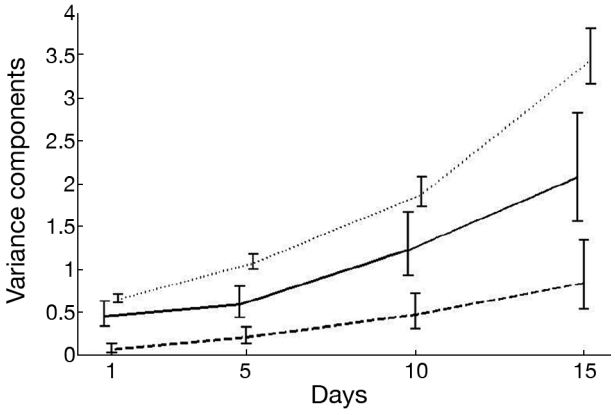

Fig. 2. *Menidia menidia*. Estimated genetic variances for Atlantic silverside dams (V_d) and sires (V_s) and the total phenotypic variance (V_p) for ages 1 to 15 d. V_d, V_s, and V_p are indicated by the solid, dashed and dotted lines, respectively. Error bars indicate 95% confidence intervals. Note that the time scale for V_d and V_p have been slightly offset to make the error bars easier to read

DISCUSSION

In this study, we found non-genetic maternal contributions were ~7 times greater than the genetic contributions to size at hatch, but decreased with age. The heritability for size at age was between 0.10 and 0.25 and increased with age. These significant maternal and genetic contributions to size at age indicate substantial potential to respond to selection, with subsequent population dynamic consequences.

Maternal effects

Few studies have explicitly examined the relative importance of maternal and genetic influences on growth in the early life history of fishes. We found that the maternal contribution to size at hatch is roughly 7-fold greater than the genetic contribution. Moreover, although the relative importance of maternal effects decreased with age, the non-genetic maternal variance was still 50% greater than the genetic variance by age 15 d. Given that early growth is a critical determinant of cohort strength (see, e.g., Hjort 1914, Gulland 1965, Ander-

Table 2. *Menidia menidia*. Mean size and estimated variance components for Atlantic silverside of ages 1 to 15 d. The numbers in parentheses indicate 95% confidence intervals rather than SE. Because distributions for variances are constrained to positive numbers and tend to be skewed, these intervals may be asymmetrical. The lengths indicated are the overall mean length at age. The variance components (V_s, V_d, V_ε, V_p and V_M), as well as the estimates of maternal contribution (V_M/V_p) and heritability (h^2), are as defined in the text

	Day 1	Day 5	Day 10	Day 15
Mean length	5.38 (4.27, 6.44)	7.46 (4.77, 9.04)	9.94 (7.80, 12.01)	13.51 (10.88, 15.80)
V_s	0.06 (0.02, 0.13)	0.20 (0.13, 0.33)	0.47 (0.31, 0.71)	0.84 (0.54, 1.34)
V_d	0.45 (0.28, 0.60)	0.59 (0.44, 0.80)	1.23 (0.93, 1.66)	2.08 (1.56, 2.83)
V_ε	0.48 (0.45, 0.50)	0.77 (0.75, 0.80)	1.21 (1.18, 1.25)	2.28 (2.22, 2.34)
V_p	0.65 (0.60, 0.71)	1.07 (1.00, 1.17)	1.87 (1.73, 2.08)	3.44 (3.17, 3.82)
V_M	0.38 (0.24, 0.58)	0.38 (0.19, 0.61)	0.76 (0.37, 1.24)	1.22 (0.47, 2.07)
V_M/V_p	0.60 (0.37, 0.86)	0.35 (0.17, 0.55)	0.40 (0.19, 0.65)	0.36 (0.13, 0.60)
h^2	0.10 (0.04, 0.19)	0.19 (0.13, 0.28)	0.25 (0.17, 0.37)	0.24 (0.16, 0.35)

Table 3. Model selection via Akaike's information criteria (AIC). *K* is the total number of parameters in each model, which includes the parameters of the fixed effects and the estimated variance components. Ln(*L*) is the log-likelihood of all data in each model (full model and reduced model specified in Appendix 1). The best model for each age of Atlantic silverside is indicated in bold

Age	No maternal effect			Model with maternal effects			
(d)	*K*	Ln(*L*)	AIC	*K*	Ln(*L*)	AIC	ΔAIC
1	30	−5439.5	10939	31	−5407.4	**10876.8**	62.2
5	30	−8492.9	17045.8	31	−8482.5	**17027**	18.8
10	30	−12419	24898	31	−12408	**24878**	20
15	30	−19552	39164	31	−19546	**39154**	10

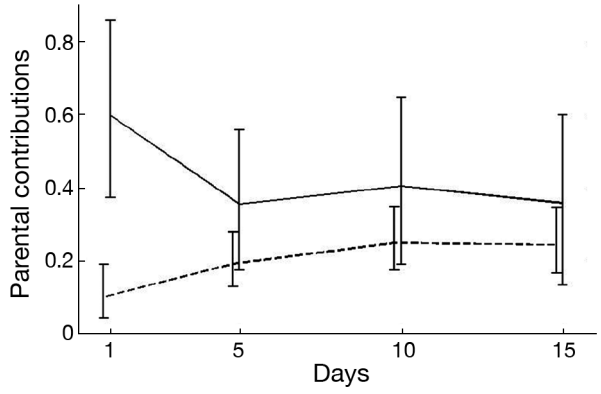

Fig. 3. *Menidia menidia*. Parental contributions of size at age with error bars indicating 95% confidence intervals. The heritability (h^2) is indicated by the dotted line, while the solid line represents the maternal (V_M/V_p) contribution. Note that the time scale of the heritability has been slightly offset to make the error bars easier to read

son 1988), we expect maternal contributions to early survival to be substantially more important than the genetic contributions, at least over the short term.

Although we do not have a specific mechanism for maternal effects in silversides, female contributions to size in the early life history most likely arise through variation in egg quality (Berg et al. 2001, Trippel et al. 2005) or embryo nourishment (Lindholm et al. 2006). As maternal provisioning may also be related to environmental factors such as prey availability (Trexler 1997), maternal effects may be an important source of variation in recruitment (Green 2008).

Recent efforts to include maternal effects in stock recruitment models have shown that including maternal size or age (Shelton et al. 2012) may substantially improve model performance. However, despite clear evidence of maternal effects, we found no correlation between maternal size and offspring size except at age 15 d. Although such size- and age-independent maternal effects are unlikely to be altered directly by fishing, they may nevertheless contribute to variation in recruitment and complex dynamics. Moreover, environmental influences before fertilization (e.g. Marshall & Elliott 1998, Salinas & Munch 2012) and other epigenetic effects (reviewed by Jablonka & Raz 2009) may also be important, leading to complex responses to environmental drivers. Attempts to forecast recruitment from stock-recruit data may therefore benefit from the inclusion of models that include maternally driven environmental forcing.

The maternal contribution to size was greatest at age 1 d and decreased subsequently. This is consistent with the observation in other species that maternal effects are often largest for traits expressed early in life such as egg size and quality (Monteleone & Houde 1990, Marteinsdottir & Steinarsson 1998). Thus, it might be expected that non-genetic parental effects are restricted to the early life stages of fish. We note, however, that in soil mites and other species, these effects create feedback loops that persist through several generations (Livnat et al. 2005, Plaistow et al. 2006). Similarly, the relationship between growth and temperature in sheepshead minnows *Cyprinodon variegats* depends on the thermal history of their grandparents (Salinas & Munch 2012). Such multi-generational feedbacks may give rise to complex population dynamics (Ginzburg 1998) and contribute to higher-order variation beyond what can be captured in a stock-recruitment relationship. Although it is possible that these feedbacks could be explicitly incorporated into the age-structured models commonly used to assess harvested populations, we suspect that it will be more effective to model them implicitly using techniques such as time-delay embedding (see e.g. Sugihara 1994).

Heritability

Although it has been hypothesized that there may be little additive genetic variance in larval traits of fish (e.g. Heath & Blouw 1998), we found that size in the early life history of Atlantic silversides has a substantial genetic component. Thus, rather than having uniquely low heritability, we find that size at age in the early life history of Atlantic silversides has genetic variation comparable to that found in older juveniles and adults. Heritability estimates for size in

older juveniles (ages 30 to 200 d) range from 0.05 to 0.45 for brown trout *Salmo trutta* (Blanc 2005), 0.3 for Atlantic cod *Gadus morhua* (Gjerde et al. 2004), and 0.33 for red drum *Sciaenops ocellatus* (Saillant et al. 2007). Compilations from the aquaculture literature suggest that the heritability values in the range 0.2 to 0.3 are often appropriate for adult size (Law 2007). We note that heritability estimates may be sensitive to food availability (Gebhardthenrich & Van Noordwijk 1991) and many other factors such as temperature fluctuations, currents, and the risk of predation may influence growth rate (Blaxter 1991, Van Buskirk & Yurewicz 1998, Dower et al. 2002). However, Weigensberg & Roff (1996) showed that estimates of lab and field heritabilities tend to be similar, though this review was largely limited to terrestrial taxa.

The increase in heritability with age in Atlantic silversides can be understood by recognizing that, although we measured the heritability of size, it is most likely that genetic variation really occurs in growth and that differences among genotypes accumulate through time. Although there are no comparable demonstrations of increasing genetic variation through time in the early life history of marine fish, there are analogues in freshwater and anadromous species, albeit at older ages. In male guppies *Poecilia reticulata*, heritability of size was negligible (≤0.028) over the first 60 d post hatch but increased to 0.5 at 90 d, leveling off at about 0.8 (Nakajima & Taniguchi 2002). In juvenile brown trout *Salmo trutta* from age 111 to 234 d, Blanc (2005) found that heritability of size increased to a maximum of 0.45 at 3 mo but decreased thereafter.

The observation that heritability of size changes with age suggests that different genetic controls are active in different growth stages. Nakajima & Taniguchi (2002) hypothesize that 3 stages exist: (1) dominance of maternal effects on size at hatch; (2) a period where growth genes are most active during the juvenile stage; and (3) an inhibitory period influencing final body size. Our observation that maternal effects were greatest at hatch and decreased while heritability was increasing broadly supports this argument. However, the change in heritability we found was much more gradual than that observed by Nakajima & Taniguchi (2002), though our study focused on a much shorter range of ages.

Predicting evolution

One of the main motivations for estimating heritability in marine fishes is in the development of evolutionarily sustainable harvest regimes (Conover & Munch 2002, Jørgensen et al. 2007, Law 2007). It is certainly tempting to imagine that we could use this information to predict evolution of size in the early life history (e.g. Munch et al. 2005, Johnson et al. 2010). Indeed, the short-term response to selection is usually well approximated by the product of the heritability and the selection differential (i.e. the change in mean size resulting from mortality or other sources of selection (Lynch & Walsh 1998). But the direct application of quantitative genetics to develop sustainable harvest strategies, which must by definition succeed over many generations, seems rather optimistic.

To evaluate the reasonableness of this idea, we performed the following thought experiment: we obtained a selection differential for a species of similar size and life-history strategy (Perez & Munch 2010), multiplied that by our estimate of heritability, and used this to predict evolutionary change in size at age 15 d. A typical standardized selection differential (i.e. in SD units) for the early life history of fish is 1.12 (Perez & Munch 2010). Given our heritability estimate of 0.24 for size at 15 d (Table 2), we would find that the expected change in length to be 0.06 to 0.19 SD each generation. Multiplying by the phenotypic standard deviation ($V_p^{1/2}$; Table 2) we would obtain an evolutionary response of approximately 0.4 mm after each generation.

To evaluate this prediction, we would ideally compare it with field data. Unfortunately, no single study has tracked early growth in wild silversides over multiple generations. But comparison of our results with early work by Conover & Present (1990) provides the basis for an informal test. Conover & Present (1990) estimated growth for silversides from New York and under conditions nearly identical to ours (21°C, unlimited *Artemia nauplii*) and found growth rates between 0.50 and 0.66 mm d^{-1}, corresponding to an estimated size at Day 15 between 12.88 and 14.62 mm, assuming the mean size at hatch is 5.38 mm. Given that 20 generations have elapsed since the work of Conover & Present (1990), our naive model predicts that size at age 15 d should have increased by 0.4 mm every generation × 20 generations = 8 mm; a 60% increase in length at 15 d! However, the mean length at Day 15 in our study was still 13.51 mm. Clearly, no dramatic shift in early growth of silversides has occurred.

While it might seem obvious that our thought experiment was overly simplistic, we emphasize that this is precisely what many simulation models that predict evolution in response to harvest selection

do (see, e.g., Martinez-Garmendia 1998, Hilborn & Minte-Vera 2008, Andersen & Brander 2009). In light of what can only be thought of as the abysmal failure of our thought experiment, it is worthwhile to consider what went wrong. One obvious possibility is that selection on size in silversides is much less, on average, than the value we used. While certainly possible, more than 70% of observed selection differentials on size in the early life history of fish are positive (Perez & Munch 2010), so it is unlikely that the apparent stasis in early growth results from an absence of selection.

More likely is the possibility that there are trade-offs associated with faster larval growth that manifest either during the larval period or later in life. For instance, faster growth causes reduced motility (Billerbeck et al. 2001) and increased susceptibility to predation in silversides (Lankford et al. 2001, Munch & Conover 2003) and other fishes (e.g. Sundström et al. 2005, Takasuka et al. 2007). These trade-offs are partly caused by a limited energy budget in which food acquisition and tissue synthesis are costly (Arnott et al. 2006). Faster growth can also lead to increased developmental asymmetry (Mather 1953, Van Valen 1962) thereby reducing fitness (e.g. Beardmore 1960).

At first glance, mortality costs to growth might seem to contradict the ubiquitous observation that faster growth increases survival through the larval period. However, it is relatively easy to show that faster growth will increase net survival through a finite interval whenever the marginal increment in mortality due to an instant of growth is less than the marginal benefit of an increase in size accumulated over the time remaining (Appendix 2). Thus, mortality costs to rapid growth may be very difficult to detect from population-level observations of survival, particularly at longer time scales.

As early growth may be correlated with later growth, one interesting possibility is that there may be trade-offs with larval growth that manifest later in life, leading to balancing selection across disparate life-history stages. For instance, although the trade-off between fecundity and contemporaneous growth is well studied (Reznick 1983, Cook et al. 1999, Roff et al. 2006), the possibility of a trade-off between fecundity and larval and early growth has received far less attention. Using a completely different experimental design, Conover & Munch (2002) estimated the coheritability between offspring size at age 15 d and maternal size and obtained nearly identical results (–0.015 compared with –0.016 reported here), suggesting a potential trade-off between the 2 stages. In addition, several studies have shown that rapid early growth reduces survival some time later in life (Gotthard 2000, Metcalfe & Monaghan 2003, Yearsley et al. 2004) and possibly increased rates of senescence (Dmitriew 2011).

In light of these trade-offs and the myriad other factors influencing larval growth, it is clear that predicting evolution of early growth requires far more information. Many authors have noted that selective effects of harvest can lead to important, potentially irreversible, evolutionary changes in life-history characters (Reznick et al. 1990, Conover & Munch 2002, Law 2007). However, the ultimate impact of selective harvest will be determined by the balance of selection, both natural and anthropogenic, which accumulates throughout the life history. Thus, the development of evolutionarily sustainable harvest strategies will require us to know not only the heritability of a handful of traits but the genetic correlations across life stages and how the selective landscape varies with age. In light of this complexity, we suggest that a more practical approach may be to identify harvest strategies that minimize the selection imposed by fisheries.

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Appendix 1. Model specification and Monte Carlo method used to estimate the quantitative genetics parameters. The model comparison to test for maternal effects is also described

Parameter estimation via maximum likelihood

Using the modified gametic model (Lynch & Walsh 1998) in Eq. (2), V_s, V_d and V_ε were estimated for size at age from Days 1 to 15 post-hatch along with fixed effects accounting for batch and seawater bath. The genetic and maternal contribution of size at each age was calculated separately as $h^2 = V_s/V_p$ and V_M/V_p where $V_p = ¼ (V_s + V_d) + V_\varepsilon$ (Lynch & Walsh 1998).

Using the assumptions laid out in the main text, the vector of all of the data, $\bar{\boldsymbol{y}}$, follows a multivariate normal distribution. Specifically the likelihood is given by:

$$L(\bar{\boldsymbol{y}} \mid \boldsymbol{\beta}, \boldsymbol{g}_s, \boldsymbol{g}_d, V_\varepsilon, N_j, s_j^2, \boldsymbol{S}, \boldsymbol{D}, \boldsymbol{X}) = (2\pi)^{-\sum_{j=1}^{F}\frac{N_j}{2}} \mid V_\varepsilon \mid^{-\sum_{j=1}^{F}\frac{N_j}{2}}$$
$$\exp\left\{-\frac{1}{2V_\varepsilon}\left[\sum_{j=1}^{F}(N_j-1)s_j^2 + N_j\left(\bar{\boldsymbol{y}} - \boldsymbol{X}\boldsymbol{\beta} - \frac{1}{2}\boldsymbol{S}\boldsymbol{g}_s - \frac{1}{2}\boldsymbol{D}\boldsymbol{g}_d\right)^T\left(\bar{\boldsymbol{y}} - \boldsymbol{X}\boldsymbol{\beta} - \frac{1}{2}\boldsymbol{S}\boldsymbol{g}_s - \frac{1}{2}\boldsymbol{D}\boldsymbol{g}_d\right)\right]\right\} \quad \text{(A1)}$$

Matrices $\boldsymbol{X}$, $\boldsymbol{S}$ and $\boldsymbol{D}$ are the incidence matrices for the fixed effects, sires, and dams, respectively. The fixed-effect incidence matrix $\boldsymbol{X}$ includes batch effects and seawater baths nested within batches. Vectors $\boldsymbol{\beta}$, $\boldsymbol{g}_s$ and $\boldsymbol{g}_d$ contain parameters for the fixed effects and genetic contributions of sires and dams. In Eq. (A1), N_j and s_j^2 indicate the sample size and sample variance in each bucket, respectively.

In keeping with standard quantitative genetic models, the genetic effects and fixed effects were treated as normal random variables, i.e.:

$$\begin{aligned}(g_{sj} \mid V_s) &\sim N(0, V_s)\\ (g_{dj} \mid V_d) &\sim N(0, V_d)\\ (\beta_j \mid V_\beta) &\sim N(0, V_\beta)\end{aligned} \quad \text{(A2)}$$

To make the model identifiable, the likelihood was multiplied by a penalty term for each variance parameter, specifically the inverse of the variance. We note that this corresponds to using a Jeffrey's prior in a Bayesian analysis.

Definitions for all symbols are summarized in Table A1. We used a Monte Carlo approach to obtain maximum likelihood estimates. Rather than marginalizing over the random effects, we used the 'complete data likelihood' and a Gibbs sampler to obtain estimates as described by Casella & George (1992). We used 5 independent chains with over-dispersed initial values to assess convergence (Gelman & Rubin1992). The 95% confidence intervals for each parameter were constructed using likelihood profiles (Hilborn & Mangel 1997).

Appendix 1 (continued)

Table A1. Symbol definitions

Variables	Meaning
N_j	Total number of individuals measured in each family j
s_j^2	Sample variance in each family j
F	Total number of families
$\boldsymbol{g}_s$	Genetic effects for sire in vector form
$\boldsymbol{g}_d$	Genetic effects for dam in vector form
$\boldsymbol{\beta}$	Fixed location effect in the experimental design
V_s	Genetic variance for sire
V_d	Genetic variance for dam
V_β	Variance for fixed effects. Set to 1000.
V_ε	Environmental variance for each family at each time point

Model comparison via AIC

The overall likelihood (L) for the full model is calculated in Eq. (A1). K is the number of parameters in each model. To test for differences in genetic variance among sires and dams, we specified an alternative model in which both sire and dam variances are equal, i.e. $V_s = V_d = V_g$. The overall model and likelihood function is otherwise the same as in Eqs. (A1) & (A2). Again, we included a penalty term for the variance component by multiplying the likelihood by $|V_g|^{-1}$.

The general formula to calculate the AIC (Akaike 1974) is:

$$\mathrm{AIC} = 2K - 2\mathrm{Ln}(L) \tag{A3}$$

The parameter sets that gave the maximum likelihood in the full and reduced (no maternal effects) models were used to obtain AIC values.

Appendix 2. Demonstration that faster growth can increase net survival through a finite interval even when the instantaneous mortality rate increases with growth

This appendix shows that faster growth may increase net survival through a finite interval even if the instantaneous mortality rate increases with growth. This occurs whenever the marginal increment in mortality due to an instant of growth is less than the marginal benefit of an increase in size accumulated over the time remaining.

It is commonly observed that faster growth through the high-mortality early life stages leads to increased survival. However, there are an increasing number of studies demonstrating that mortality rates increase with growth. How can these 2 observations be reconciled? Here, we show that faster growth will increase net survival through a finite interval even when mortality rates increase with growth, whenever the marginal benefit of size over the remainder of the interval exceeds the marginal cost of growth.

We assume that the instantaneous rate of mortality, $\mu(x,g)$, is a function of size (x) and growth rate (g) and that mortality decreases with size and increases with growth. Survival over the time interval $0,T$ is given by $S = \exp\left[-\int_0^T \mu(x,g)\,\mathrm{d}t\right]$. To isolate the influence of growth, we recognize that size is just the time integral of growth, i.e. $x(t) = x(0) + \int_0^t g(t')\,\mathrm{d}t'$ where t' is just a dummy variable for time. Therefore, we can re-write survival just in terms of growth, $S = \exp\left[-\int_0^T \{\mu(x(0) + \int_0^t g(t')\,\mathrm{d}t', g(t))\}\,\mathrm{d}t\right]$. To evaluate the sensitivity of survival to growth at a specific time, say τ, we take the derivative,

$\frac{\partial \ln[S]}{\partial g(\tau)} = -\int_\tau^T \frac{\partial \mu}{\partial x}\mathrm{d}t - \left.\frac{\partial \mu}{\partial g}\right|_{t=\tau}$ and ask when it is positive. This occurs when $\left.\frac{\partial \mu}{\partial g}\right|_{t=\tau} > -\int_\tau^T \frac{\partial \mu}{\partial x}\mathrm{d}t$.

If we further assume that growth is non-negative, this integral is bounded above by $\left.\frac{\partial \mu}{\partial x}\right|_{t=\tau}(T-\tau)$.

Therefore we can re-write the inequality as $\frac{\partial \mu}{\partial g} > -\frac{\partial \mu}{\partial x}(T-\tau)$. This inequality provides a sufficient, though not necessary, condition for survival over a finite interval to increase with growth despite an instantaneous mortality cost. It tells us that faster growth will increase net survival over a finite interval whenever the mortality cost of growth is less than the product of the marginal benefit of an increment in size and the time remaining. Because of this, any penalty for rapid growth early in the interval is more easily masked by the decrease in size-dependent mortality. Thus, it is certainly possible that rapid growth increases mortality but nevertheless results in increased survival over a finite time interval.

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