Title: Phenotypic and genetic integration of personality and growth under competition in the sheepshead swordtail, *Xiphophorus birchmanni*

Running title: Genetics of competition

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Word count: 8031

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Funding: This work was supported by an EPSRC Studentship to KB, a BBSRC David Phillips Fellowship to AJW and a BBSRC grant (BB/L022656/1). CAW was funded by a NERC post-doctoral Research Fellowship (NE/I020245/1) and a University of Edinburgh Chancellor's Fellowship. GGR was funded by the U.S. National Science Foundation

Acknowledgements: This work was supported by an EPSRC Studentship to KB, a BBSRC David Phillips Fellowship to AJW and a BBSRC grant (BB/L022656/1). CAW was funded by a NERC post-doctoral Research Fellowship (NE/I020245/1) and a University of Edinburgh Chancellor's Fellowship. The federal government of Mexico provided permission to collect fish. GGR was funded by the U.S. National Science Foundation. We are grateful to Dan Nussey, Mark Briffa, John Hunt and Tom Houslay for useful discussions that contributed to the development of the manuscript.

Abstract

Competition for resources including food, physical space, and potential mates is a fundamental ecological process shaping variation in individual phenotype and fitness. The evolution of competitive ability, in particular social dominance, depends on genetic (co)variation among traits causal (e.g., behaviour) or consequent (e.g. growth) to competitive outcomes. If dominance is heritable, it will generate both direct and indirect genetic effects (IGE) on resource dependent traits. The latter are expected to impose evolutionary constraint because winners necessarily gain resources at the expense of losers. We varied competition in a population of sheepshead swordtails, *Xiphophorus birchmanni*, to investigate effects on behaviour, size, growth, and survival. We then applied quantitative genetic analyses to determine (i) whether competition leads to phenotypic and/or genetic integration of behaviour with life history and (ii) the potential for IGE to constrain life history evolution. Size, growth and survival were reduced at high competition. Male dominance was repeatable and dominant individuals show higher growth and survival. Additive genetic contributions to phenotypic covariance were significant, with the **G** matrix largely recapitulating phenotypic relationships. Social dominance has a low but significant heritability and is strongly genetically correlated with size and growth. Assuming causal dependence of growth on dominance, hidden IGE will therefore reduce evolutionary potential.

61 **Introduction**

62 An individual's phenotype is determined by its genotype and the environment it experiences
63 throughout life. Competition with conspecifics for resources (e.g. food, space, mating opportunities)
64 is one important environmental factor known to have large effects on phenotypic traits including
65 growth (Ruzzante and Doyle 1991) and life history traits (e. g, maturation, fecundity, longevity).
66 Importantly, by producing winners and losers, competition generates variation in resource-
67 dependent traits and ultimately in fitness. Since winners increase their (relative) fitness at the
68 expense of losers (Brockelman 1975), those traits contributing to competitive ability are also
69 expected to be under strong selection. If so, then the evolutionary consequences of this selection
70 will depend on the genetic covariance structure between traits causal and consequent to social
71 dominance (Wilson 2014). In the particular case that dominance itself is heritable, this genetic
72 covariance will include contributions from indirect genetic effects (IGE; Bijma and Wade 2008) that
73 can constrain adaptation of resource-dependent traits (Wilson 2014). Here, we use a quantitative
74 genetic approach to characterise the genetic basis of social dominance in a population of the
75 poeciliid fish *Xiphophorus birchmanni* and explore the extent that genetic and environmental effects,
76 notably including the degree of competition itself, shape the multivariate phenotype. Our goals are
77 to assess the extent to which competition leads to phenotypic and/or genetic integration of
78 behavioural and life history traits, and to evaluate the potential for constraining IGE on the latter.

79 In animals, intraspecific competition takes different forms and occurs across many different
80 social contexts – from pure scramble competition leading to density dependence (Hassel 1975), to
81 dyadic contests that can escalate to become an important source of mortality (e.g. Liker and Szekely
82 2005). From an ecological perspective, competition reduces mean (absolute) fitness with
83 consequences for regulation and determination of population demography (Schoener 1983; Sih et al.
84 1985; Chase et al. 2002). However, in evolutionary terms perhaps the most important role of
85 competition is as a mechanism that generates among-individual variation in both phenotypes and
86 fitness. Within populations, individuals can vary in competitive ability, or social *dominance,* defined

here as an individual's repeatable tendency to win or hold resources under competition (Wilson et al. 2011a). Note that this definition is simply phenomenological, and certainly does not imply dominance is determined solely by "intrinsic" factors. Indeed the converse is true; winning resources usually depends on both focal phenotype and the particular social context or environment provided by competitors.

More competitive phenotypes should generally be favoured by selection, and this has implications for the evolution of traits both causal and consequent to competitive outcomes. Although social dominance is not necessarily without costs (e.g., Wong and Kokko 2005; Bell et al. 2012) overall, dominant individuals win resources and thus ultimately gain relative fitness at the expense of subordinates. This in turn allows increased investment in, for instance, growth, earlier maturation or reproductive effort (Bernstein 1976; Huntingford et al. 1990; Fox et al. 1997). Where fitness is tightly linked to competitive outcome, traits determining dominance might be under strong directional selection (Kruuk et al. 2002; Benson and Basolo 2006; Prenter et al. 2008). Simple evolutionary theory predicts that, all else being equal, this should erode genetic variance (Fisher 1958). If so, then at equilibrium phenotypic variation in traits determining dominance (and so dominance itself) will largely be due to environmental effects (Kruuk et al. 2002). However, directional selection on contest outcome can also generate disruptive selection on, and so maintenance of variation in, quantitative traits that mediate competitive outcomes (e.g., Abrams et al 2008).

Although the limited number of studies conducted to date have generally found relatively low heritabilities for measures of social dominance (Wilson et al. 2011b; Sartori and Montavani 2012), this may reflect high environmental variance rather than an absence of genetic effects. For instance, much of our understanding of dominance comes from dyadic animal contest studies where winning is often causally dependent on heritable aspects of morphology such as body size and/or weapons (e.g. horns, Preston et al. 2003). More recently, there has been growing recognition that social dominance can also depend on an individual's (repeatable) behavioural phenotype, or

personality (Reale et al. 2010). Evidence is now accumulating that personality traits linked to competition, such as aggression and boldness (loosely defined as a willingness to take risks) are also heritable (e.g., Drent et al. 2003; Sinn et al. 2006; Ariyomo et al. 2013). Integration of multiple behavioural and morphological traits could result in alternative "strategies" for success in competition having equal fitness and/or being maintained by frequency dependent selection. This may contribute to the maintenance of genetic variance in traits causal to dominance (as we broadly define it) and could occur if, for example, large aggressive individuals succeed in contest competition, but smaller and bolder (or more exploratory) individuals do well in more scramble-like competition. Such a scenario could potentially explain the maintenance of sneaker male morphs in systems with male-male competition for females (e.g., Ryan et al. 1992). However, across taxa the emerging - albeit certainly imperfect - pattern is one of positive covariance between boldness and aggression (e.g., Johnson and Sih 2005; Pintor et al. 2008; Ariyomo and Watt 2012). Furthermore, both these personality traits are commonly positively associated with social dominance, resource-dependent life history traits and fitness measures (e.g., Biro and Stamps 2008; Ariyomo and Watt 2012; Rudin and Briffa 2012).

Regardless of the relative importance of morphological and behavioural traits, it seems likely that social dominance will often be determined by genetically variable components of phenotype and so can be viewed as a heritable trait in its own right. If so, this has important implications for our understanding of life history evolution. This is because genes that increase dominance will allow individuals to succeed in competition, gain more resources, and so invest more in all resource-dependent life history traits. Genetic variance in dominance will therefore be a source not only of heritable variation in downstream traits, but also of positive genetic covariance (defined with respect to fitness consequences) between traits subject to resource-dependent trade-offs (Wilson 2014). Positive genetic correlations are a common empirical finding in natural populations (Kruuk et al. 2008) and seemingly pose a challenge to the view that trade-offs, expected to manifest as negative genetic correlations, ubiquitously impose evolutionary constraint.

However, genetic variance in dominance, or competitive ability, is also expected to generate indirect genetic effects (IGE). IGE occur when the phenotype of one individual is causally dependent on the genotype of another, and are inevitable in the case that dominance is genetically variable. IGEs can have important implications for predicting evolutionary responses, and in particular are predicted to dampen the response of resource dependent traits when they arise from competitive interactions. This is because selection on these trait(s) is expected to result in the correlated evolution of a more competitive social environment that offsets the expected phenotypic change (Hadfield 2010; Wolf et al. 1998; Bijma and Wade 2008; Wilson et al. 2011b). Consequently, while genetic (co)variance is the raw material for adaptive evolution, understanding the extent that it is independent of social competition (and thus constraining IGE) may be a prerequisite for predicting selection responses (Hadfield et al. 2011; Wilson 2014).

Here we tested the genetic basis of dominance, and characterised both genetic and environmental contributions to covariance in and between dominance, personality, size and growth, and survivorship in a laboratory population of the sheepshead swordtail, *Xiphophorus birchmanni*. Swordtails have been widely used in studies of social dominance (e.g., see Earley 2006 for an historical review; Walling et al. 2007; Boulton et al. 2012), while previous work on this particular population has found evidence of stable personality traits including aggressiveness (Wilson et al. 2013) and boldness (Boulton et al. 2014). In adult males, aggressiveness has been shown to be a better predictor of dyadic contest outcome than body size (Wilson et al. 2013). The ability to win food in dyadic contests is repeatable in adults of both sexes, while dominant individuals (i.e. those that consistently win) tend to gain weight at the expense of subordinates (Wilson et al. 2013).

In our experiment we use density manipulations to increase the expected intensity of competition in the sheepshead swordtail. After testing for competition effects on mean growth, personality and survival, we estimate the among-individual and genetic covariance structures between traits related to social dominance. We manipulate competition by subjecting a captive bred generation of fish to contrasting low (L) and high (H) competition treatments in both early and

later life. We hypothesise that high competition (i.e. high density), particularly if experienced in early life, will reduce growth rates and negatively impact fitness components (e.g. survival). Having shown direct effects of competition on phenotypic expression, we use a multivariate modelling approach to estimate the relationships among traits at the individual and additive genetic levels. We predict that personality differences will predict social dominance with bolder individuals tending to be dominant. We also expect that social dominance will positively predict growth and survival. If heritable variation for dominance is present, then similar correlation structure is expected at the (direct additive) genetic level. Importantly, if this is this case the (direct) genetic (co)variance structure among resource-dependent traits (**G**) will be insufficient to predict selection responses. More specifically, **G** will give an upwardly biased expectation of the potential for adaptive evolution because it does not account for IGE that will necessarily arise if dominance is heritable.

Materials and methods

Data analysed here were from a broader study (Boulton et al. 2014; Boulton et al. 2016). Previously described methods are thus kept accordingly brief. Behavioural data from open field trials (OFT) have been previously published (Boulton et al. 2014) but not subjected to genetic analyses.

Husbandry and density treatments

Briefly, one hundred adult fish (60 female and 40 male *Xiphophorus birchmanni*) were caught from the Río Coacuilco, in the municipality of San Felipe Orizatlán, Hidalgo, Mexico, and imported to the UK. Between August 2010 and May 2011, these fish were crossed to produce 384 offspring comprising 61 families nested within a half-sib structure (with 19 male and 32 female parents represented). Families of ≤8 individuals were raised in one half of a brood tank (37 x 37 x 22 cm capacity partitioned into two equal volumes with a mesh divider). Full-sib families of >8 individuals were divided equally across two partitions (in different tanks). Groups of six brood tanks (and eight experimental housing tanks; described below), henceforth referred to as *stacks* shared a single

191 recirculating water supply. Offspring were fed twice daily (fresh brine shrimp nauplii and a mix
192 comprising equal quantities of crushed spirulina and brine shrimp flake). At an average age of 16
193 weeks (range 12-27) and length 27 mm (20-35), fish were tagged below the dorsal fin with coloured
194 elastomer and assigned to mixed family groups (n=8). Each group was subject to one of two initial
195 density treatments; low (L) density groups were housed in a full tank (37 x 37 x 22 cm), while high
196 (H) density groups in a partitioned tank (i.e. half the volume). Six stacks were set up sequentially,
197 (each comprising four L and four H groups) as sufficient fish (64 per stack) reached sufficient size to
198 enable individual identification by use of elastomer tags. Sex ratio was not controlled as external
199 sexing of juveniles is not possible. All groups received the same food ration (commercial flake and
200 frozen tropical fish food) twice daily. After 28 weeks (subsequently referred to as part 1), density
201 treatments were reversed for four randomly chosen groups within each stack. All groups were
202 maintained for a further 22 weeks (part 2 of the study). Thus within each stack, four density regimes
203 were experienced (LL, LH, HL, HH), with two groups per regime. Natural mortality over the course of
204 the experiment resulted in some variation in group size (initially 8) through time, although survival
205 was high (368 of 384) over the first density treatment period (i.e. part 1 of the study).
206
207 *Phenotyping methods*
208 Behavioural data were collected on boldness and dominance. Boldness was determined using open
209 field trials (OFT) described fully in Boulton et al (2014). Individuals were subject to a total of four
210 OFT: two in part 1 (weeks 13 and 21) and two in part 2 (weeks 33 and 41). At each trial a fish was
211 introduced to a 45 x 25 x 25 glass tank filled to a depth of 8 cm with room temperature water (22^{o}C).
212 After 30 s acclimation, a five minute observation period was filmed and a suite of traits putatively
213 indicative of boldness extracted from the video using the Biobserve Viewer tracking software. Our
214 previous analysis shows the among-individual (i.e. repeatable) component of multivariate variation
215 is dominated by a single major axis of variance, broadly matching expectations of a shy-bold

continuum (Boulton et al. 2014). Thus, here we selected a single trait, *activity* (percentage time in trial spent moving at >1.5 $cm.s^{-1}$) for use as a proxy for boldness.

Social dominance was assayed for males only using in-tank observations (ITO). Behaviour of each male in each group was recorded for five minutes, at up to 5 occasions during the experiment: two at the initial density treatment during part 1 of the study (weeks 18 and 25), and three at the final density treatments during part 2 of the study (30, 38, 44 weeks). Remaining males at the end of part 2 were pooled with others from their stack in a large tank (45 x 120 x 30 cm) containing previously un-encountered stock females and observed on a minimum of five further occasions (consecutive days where possible). Within groups, focal males (identifiable from natural markings and elastomer tags) were watched sequentially in a haphazard order by a recorder seated in front of the tanks in full view of the fish. Fish were accustomed to researcher presence and our judgement was that this did not impact behaviour.

While we acknowledge that aggression (actual, threat or signal of attack, Hand 1986, Francis 1988) and dominance are not equivalent, the former is often used to assert the latter (Bernstein 1976). Here, we have previously shown that aggressive behaviours predict feeding dominance among male *X. birchmanni* (Wilson et al. 2013) while male dominance is known to determine access to females in swordtails generally (Magellan and Kaiser 2010). For each five minute observation period, a within-group *dominance score* was therefore assigned to each focal male as the total number of aggressive actions toward other males (attacks, dorsal fin displays, chases), plus the number of courting attempts (displaying to female, shepherding away from other males), minus the number of submissions (retreating or fleeing from another male) and aggressive acts received (see Wilson et al. 2013 for further description of these behaviours).

Finally, standard length (SL) and live mass (WT) were measured at tagging (measure 1) and subsequent four-weekly intervals. Up to 13 measures were made on each fish (with measure eight corresponding to the end of part 1, and measure 13 the end of part 2). We also recorded *longevity*

as the age at death in days (regardless of whether death was natural or by euthanasia), and right-censored to age at the end of part 2 of the experiment (for fish alive at measure 13).

Statistical analyses

Following graphical exploration of the raw data, density treatment and genetic effects on behaviour, size and growth were tested using (univariate) linear mixed effect models, including pedigree-based animal models (Wilson et al. 2010) fitted by restricted maximum likelihood (REML) in ASReml-R. Conditional F-tests were used for inference on fixed effects in the univariate models, with sequential dropping of non-significant terms (but retaining main effects in the presence of significant interactions). Starting fixed effects were included to control for potentially confounding effects, and to test for density treatment effects. Inference on random effects was by likelihood ratio test (LRT). We follow Self and Liang (1987) by assuming the test statistic is distributed as a 50:50 mix χ^2_0 and χ^2_1 (denoted $\chi^2_{0,1}$) for tests of a single variance component. For comparing models differing in more parameters (e.g. random regressions and multivariate models described below) we adopted the more conservative strategy of setting DF equal to the number of additional covariance components in the more complex model.

<u>Determination of fixed effects in univariate models</u>

To test density treatment effects two-level factors were defined corresponding to early life density (*ELD*; L vs H in part 1) and late life density (*LLD*, L vs H in part 2). Thus *ELD*:*LLD* defines a factor specifying the full regime (LL, LH, HL HH). Since *LLD* treatment cannot influence phenotypic observations made during part 1 of the experiment, effects were fitted only to part-specific data (denoted by subscript) where appropriate. ELD_{part1}, LLD_{part2} and $ELD{:}LLD_{part2}$ were therefore fitted for *activity*, *dominance score*, *SL* and *WT*.

All starting models included fixed effects of *stack* (a six-level factor), *sex* (except *dominance score* since male-limited), *sex ratio* (*SR*) and *group size* (*GS*). *Group size* (*GS*) and *sex ratio* (*SR*)

experienced were defined for each individual (*i*) rather than each group. *GS* was defined as the geometric mean number of fish in *i*'s group, averaged across months up to and including the observation, and included to control for effects of mortality (reducing group size from the starting n = 8). *SR* was similarly defined as the geometric mean (across previous months) of the proportion of i's tank mates that are mature males (see Boulton et al 2016). Both variables were (arithmetic) mean-centered across all individuals to aid model interpretation. For behaviours, additional fixed effects included: *trial* (factor, the number of previous assays experiences); *order* (zero-centred covariate, indicating the trialling sequence of individuals tested on a day); and *observation type* (within-group during main experiment versus in larger tank after) for *dominance score*. For size traits (*SL* and *WT*) we included *Measure* (a factor with 13 levels) and *Measure:Sex*, allowing sex specific average growth patterns over the 13 months. A linear effect of absolute *Age* (zero-centred) was also included to account for variation in age among fish entering the experiment.

Random effect specification in univariate models

Random additive genetic and permanent environment effects were fitted using a standard repeat measures animal model (Wilson et al 2010). For *SL* and *WT* this model was extended to include 1st order (linear) random regressions on age (zero centred on the mean age of 294 days) for both additive and permanent environment effects (following e.g., Wilson et al. 2006). This partitions each individual's genetic deviation from the mean trajectory of size over age into a random intercept, and a random slope. Variance in the former represents genetic variance in size (at average age), the latter genetic variance in growth. Environmental deviations from the mean size are treated analogously to partition the non-genetic component of among-individual variation in size (at average age) and growth. Repeatability (R) was estimated as the ratio of among-individual variance (V_I) to phenotypic variance (V_P) conditional on fixed effects using a simple repeat measures mixed model containing *identity* and (for *SL* and *WT* only), *identity* x *age* as random effects. Narrow sense heritabilities, h^2 were estimated from the animal models as the ratio of V_A to V_P. We did not

generate R or h^2 estimates for *Growth* (as inferred from either *SL* or *WT*) because among-individual and additive genetic variances in reaction norm slopes are estimated from the random regression models but residual variances are not.

Analysis of survival data

Right censored age of death data were analysed using a proportional hazards regression model implemented by coxph in the R library survival (Therneau and Grambsch 2000; Therneau 2015). Predictors of *Stack*, *Sex*, GS_i, SR_i and *ELD:LLD* were included. Note that mortality occurred almost exclusively in part 2 so separate effects of ELD and LLD were not modelled. A small number of fish that died with indeterminate sex were excluded. Heritable variation in *survival*, defined as zero (dead before measure 13) or one was also tested for using a univariate animal model and the same fixed predictors with an addition linear effect of age at Measure 1. We assumed a Gaussian error structure in order to obtain an estimate of heritability on the observed (i.e. 0/1) data scale, but note that statistical inferences from this model parameters should be treated caution as a consequence.

Multivariate models to estimate **ID**, **G** and selection through *longevity*

Multivariate mixed models were then used to estimate **ID,** the among-individual phenotypic variance-covariance matrix, and the additive genetic matrix **G**. These were fitted in the standalone implementation of ASReml (v4) assuming Gaussian residuals. The 6x6 **ID** matrix was first estimated among the set of observed traits (*activity*, *dominance score, SL, WT*) and the two growth traits ($Growth_{SL}$ and $Growth_{WT}$ modelled using random regressions). Observed traits were scaled to standard deviation units to facilitate convergence and fixed effects included on each trait as determined from univariate analyses. A random effect of individual identity was included on all traits. This model was compared to one where **ID** was constrained to be a diagonal matrix (i.e. all covariance elements equal to zero) as an overall test of among-trait covariance. The original model was then extended to include *survival* (0, 1) as an additional response variable. *Survival* is observed

once only, such that V_I and V_R are not separable and the latter was therefore fixed to zero. This partitions all variance in survival (conditional on fixed effects) to the extended **ID** structure. Observed *survival* was divided by the mean to convert to relative fitness. The covariance estimates between each trait and relative fitness in **ID** can then be interpreted as the ordinary selection differentials of quantitative genetic theory (Falconer and Mackay 1996) contained within a vector **S**. We compared this model's fit to one where all elements of **S** are fixed to zero as a global test for selection.

The above steps were repeated using male and female data separately to qualitatively check whether pooling sexes for multivariate genetic analysis was sensible, and determined whether selection was similar across sexes. $\mathbf{ID_{male}}$ and $\mathbf{ID_{female}}$ were broadly similar (apart from necessary exclusion of *dominance score* in females; see results) so power was maximised by estimating **G** from a pooled-sex multivariate animal model. We note that if G x *Sex* interactions occur, the resultant estimate of **G** can be viewed as an average of sex-specific matrices. Trait-specific fixed effects were included as before, with random additive genetic and permanent environment effects on all traits. For *SL* and *WT* first order random regressions of *age* were used. The full model fit was compared to one with a diagonal **G** matrix assumed as a global test for genetic covariance among traits and individual COV_A estimates were scaled to genetic correlations (r_G) to facilitate interpretation. Given a lack of V_A in the univariate analysis, we did not expand this analysis to include (relative) *survival*.

Results

Data structure and fixed-effects on traits

The final data set included 384 individuals (222 males, 151 females, 11 fish with undetermined sex at time of death or end of data collection period); 4175 age-specific measures of size (*SL* and *WT*); 1235 observations of *activity* in OFT; and 1385 observations of male *dominance score*. Visual comparison suggests mean growth trajectories are similar across sexes (for *SL* and *WT*; Figure 1), although standard deviations for size at each age are uniformly larger in males (Figure 1A vs 1B, 1C vs 1D). On

345 average, growth continued across the study timeline in all density treatments, although the
346 comparatively constant rate of absolute growth in part 2 masks a decline in relative growth rates
347 after maturation in both sexes (see supplemental figure 1).

348 Effects of density treatment were not obvious from visual inspection of behavioural data
349 (not shown), while univariate models confirmed no significant density treatment effects on *activity*
350 or *dominance score* (Table 1; see also supplemental Table 1 for coefficient estimates). In contrast,
351 high part 1 density reduces age-specific *SL* and *WT* (especially in females; Fig 1A, 1C). Fish
352 experiencing low density in early life were bigger in part 1, both in terms of *SL* (ELD_{part1} L coefficient
353 (SE) = 0.635 (0.113), $F_{1,2583.6} = 31.5$, P<0.001) and *WT* (ELD_{part1} L coefficient (SE) = 0.050 (0.020),
354 $F_{1,2583.6} = 31.5$, P<0.001). Significant effects of LLD_{part2} and $(LLD:ELD)_{Part2}$ on *WT* were also detected
355 (Table 1). Collapsing these terms into a four level factor defining possible treatment combinations
356 on WT_{part2} yields effect size estimates (relative to expected mean for HH fish) of LL -0.038 (0.039) , LH
357 -0.165 (0.039), HL 0.024 (0.033). Thus, the significant effect of LLD on $WT_{\text{part 2}}$ is driven largely by a
358 negative impact of switching from L to H at the end of part 1.

359 Based on significance in univariate models, some additional fixed effects were retained in
360 variance component and multivariate analyses that are not directly relevant to current hypotheses,
361 therefore we describe them only briefly here (but see Table 1 and Supplemental Table 1 for full
362 presentation). Specifically *sex* effects are present on mean size (*SL* and *WT*) and average growth
363 trajectory (manifest as *Measure:Sex* effects) and *GS* effects positively influenced *SL*. *SR* was not a
364 significant predictor of any trait. Among-*stack* differences were present for all traits except
365 *dominance score*. For *activity*, significant *trial* effects were driven by greater mean *activity* at trials
366 2-4 relative to trial 1, while a negative effect of *order* was also present. These were already known
367 from prior analysis of OFT data (Boulton et al 2014). *Trial* also significantly influenced *dominance*
368 *score*, with higher scores seen in the later observations made after mixing fish across units. This is
369 consistent with an expected increase in agonistic interactions among individuals that are unfamiliar
370 with each other (see e.g., Wong and Balshine 2011).

Repeatabilities, heritabilities and (genetic) variance in growth.

Univariate models provided statistical support for among-individual variance (conditional on fixed effects), underpinned by genetic effects for all traits (but not survival as noted above). Testing random effects in univariate models confirmed significant repeatability of *activity* (R = 0.260 (0.034)) and male *dominance score* (R = 0.242 (0.032); Table 2). Behavioural heritabilities were low ($h^2_{activity}$ = 0.093 (0.056), $h^2_{Dominance}$ = 0.066 (0.052)) but V_A estimates significant at P<0.05 (Table2). Size varied among individuals (*SL* and *WT*) and first order random regression models yielded significantly better fits than simple repeated measures models. Thus there is among-individual variance in both size and growth (the latter being I x *age* for size; Table 2). This was mirrored at the genetic level with V_A and G x *age* interaction statistically supported for *SL* and *WT* (Table 2). Noting that ID x *age* and G x *age* imply age-dependence of V_I and V_A respectively, we estimate R_{SL} = 0.908 (0.008) and R_{WT} = 0.839 (0.011) at 294 days (the mean observed age in the data set). At this age h^2_{SL} = 0.247 (0.106) while h^2_{WT} is lower (though not significantly so) at 0.144 (0.076).

Survival analysis

Two hundred fish (52.8%) remained alive at the end of part 2 (measure 13). Observed survival to measure 13 was higher in females (62.0% versus 46.6% in males) with treatment specific rates (sexes combined) of LL = 59.1%, LH = 46.9%, HL = 55.9%, and HH = 49.5%. Testing of the treatment effects in the proportional hazards regression predicts that, conditional on other model effects, survival is lower in LH and HH and higher in HL relative to LL (the reference treatment level (Figure 2)). However, only in LH is the difference from LL statistically significant (Table 3). Thus, experiencing low density in early life and then being switched to high density has a negative effect on survival. In addition there were significant effects of *Stack*, *SR*, and *GS* (with the hazard for a focal individual increased in more male-biased and larger groups; Table 3). The animal model of survival yielded a small non-significant estimated for survival on the observed 0/1 scale of h^2 = 0.016 (0.015).

Multivariate models: **ID**, **G** *and selection through survival*

Comparison of unstructured and diagonal models revealed significant among-trait covariance in **ID**. This was true in pooled-sex (χ^2_{15} = 2057, P<0.001), and sex-specific analyses (males χ^2_{15} = 1430, P<0.001, females χ^2_{10} = 663, P<0.001). Length and weight are strongly positively correlated with each other in the pooled sex estimate of **ID** (Table 4), and also with *growth* traits. In other words random intercepts (size at mean age) and slopes (growth) were positively correlated in the random regression models. For males, *dominance score* is positively correlated with all measures of size and growth but, counter to predictions, is negatively associated with *activity*. The correlation between *activity* and *dominance score* is nominally significant based on Z score ≥ 1.96 (r_I= -0.410 (0.104)).

Noting that *dominance score* is a male limited trait, and excluding relationships with *survival* (see below) the correlation structure in **ID** is qualitatively similar in the two sexes (Supplemental Table 2) and **G** was thus estimated from a pooled analysis. A diagonal genetic matrix was supported over a null model (χ^2_6 = 35.6, P<0.001), and the fully unstructured matrix was significantly better again (χ^2_{15} = 59.4, P<0.001). The first comparison corroborates the presence of genetic variance, the second provides evidence of among-trait genetic covariance structure (Table 4). On a correlation scale, (i.e. upper diagonal of **G** in Table 4), our estimates of between-trait genetic relationships largely mirror those in **ID** (although SEs are larger and not all pairwise estimates of r_G should be deemed significant). Thus, for example, we find a strong negative genetic correlation between *activity* and *dominance score* (r_G = -0.845 (0.361)). The former is also negatively genetically correlated with size and growth, for the latter the correlation structure is positive (Table 4).

Finally, expanding the multivariate model used to estimate **ID** to also include relative survival provided evidence for (among-individual) phenotype-fitness covariance (both sexes combined χ^2_6 = 97.8, P<0.001). Selection differentials, S (contained in the final row of the expanded **ID** matrix; Table 4) and their corresponding correlations indicate positive trait-fitness associations that are nominally significant for all traits except *activity* (r_I = 0.013 (0.074)). Thus large, fast

growing, dominant (if male) individuals showed higher survival, but *activity* does not predict fitness. In males, the phenotype-fitness associations were similar (supplemental table 2), and significant (χ^2_6 = 104.8, P<0.001). However, single sex analysis provided no statistical support for significant selection on female traits (excluding dominance; χ^2_5 = 7.38, P = 0.194).

Discussion

The aims of this study were to ascertain the effects of experimentally manipulated competition on growth, personality and survival in the sheepshead swordtail, and to investigate among-individual and genetic covariance structures between traits related to social dominance. We found evidence of reductions in size and growth at high competition as predicted, but no evidence of density effects on personality. Between traits there was significant among-individual covariance structure, with personality (boldness) predicting social dominance, size, growth and survival, though not all relationships matched our *a priori* predictions. Genetic covariance was also found between traits and we detected low, but statistically significant genetic variance in male dominance that has important consequences for the evolution of traits dependent on competitive outcomes. In what follows we first discuss the density effects on phenotype and patterns of phenotypic covariance before addressing these evolutionary implications in more detail.

The effects of increased competition on phenotype and fitness

As predicted, we found evidence that density (i.e. level of competition for space) influenced phenotypes and fitness. For example, size and growth rates were lower in early life at high density, consistent with the widespread reporting of density dependent growth rates in fishes (see e.g., Rothschild 1986; Lorenzen and Enberg 2002; Hixon et al. 2012). Significant density treatment effects on later life growth were also found, and were driven in particular by reduced growth (measured by live mass (WT)) in fish that experienced the low:high (LH) regime. Thus, it seems that switching from a low to a high competition environment part way through development may impose a greater

challenge to growth than consistently experiencing high density. Conversely, males experiencing the HL regime actually had the greatest mean size at the end of the experiment. This latter pattern is consistent with compensatory growth, a widely reported phenomenon in fishes entailing a phase of accelerated growth following a period of growth depression, usually when favourable conditions are restored (e.g., Metcalfe and Monaghan 2001; Ali et al. 2003). We also found that survival was directly influenced by the competitive environment. Observed survival was highest in fish experiencing low density throughout life (LL) and lowest in the HH treatment. However, the predictions from survival analysis indicated that, after conditioning on other model effects, the most striking outcome is a significant reduction in survival for fish moved from low to high density (relative to those not moved). This may indicate some form of adaptive plastic response to density in early life, such that individuals raised at low density find themselves maladapted if subjected to an environmental switch. In this case it is necessarily a plastic within-generation effect; however, the pattern shows some interesting parallels to results of reciprocal translocation experiments in wild guppies that have been interpreted as evidencing evolution under density dependent selection (Bassar et al 2013).

Reduced allocation to resource dependent traits and a decrease in (absolute) fitness are defining features of competition found ubiquitously across taxa. Thus the reduced growth and survival at higher density are consistent with our density treatment having manipulated the level of competition as intended. We previously reported a weak trend towards later and smaller maturation at high early life density in these fish, highlighting the fact that other aspects of life history are also impacted (Boulton et al. 2016). In contrast however, we found no evidence of density treatment effects on individual behaviour. Although this was not unexpected for male dominance (assayed within groups of individuals experiencing the same treatment regime), several recent studies have reported links between density and “bold type” personality variation (see e.g., Patrick et al. 2013; Müller et al. 2016 for observational and experimental studies respectively). Conversely, Niemelä et al. (2012) reported no impact of experimentally manipulated (larval) rearing density on adult

boldness in the field cricket *Gryllus integer*, a result that mirrors our lack of population level plastic response of boldness to the density treatment applied.

Among-individual correlations between traits and fitness

After controlling for all fixed effects, our mixed model analyses provided strong evidence of among-individual variance in those traits with repeated measures (i.e., *activity*, *dominance score*, *size* and *growth*). Repeatability of *activity*, used here as a proxy for boldness, was known from prior analysis of this data (Boulton et al. 2014). However, consistent among-male differences in *dominance score* provide independent confirmation that male dominance in this species can be viewed as a repeatable trait of the individual (Wilson et al. 2013), albeit one that will also depend on social context (i.e. group, competitor phenotype). We also found evidence of significant correlations between phenotypic traits (at the among-individual level) and between traits and fitness, though not all relationships were as predicted. Most strikingly, we had predicted a positive correlation between individual boldness and dominance, but in fact found a strong and highly significant negative one. The strength of the correlation remains consistent with the idea that this personality trait is part of what determines an individual's competitive ability (Briffa et al. 2015), but clearly our directional prediction, based largely on the emerging pattern in the literature (e.g., Dingemanse and de Goede 2004; Sundstrom et al. 2004; Webster et al. 2007; Dahlbom et al. 2011), was entirely wrong. Speculatively, it is possible that the negative association between boldness and dominance reflects alternate male strategies for obtaining resources (food and / or mating opportunities) that have been reported in some *Xiphophorus* species (Ryan and Causey 1989; Zimmerer and Kallman 1989; Ryan and Keddyhector 1992; Cummings and Gelineau-Kattner 2009). For instance, socially dominant males may be able to hold territories in the natural environment, with subordinates having to use more active, mobile and exploratory (i.e. bold-type) behaviours to find undefended resources.

Other correlations in **ID** were more in line with our *a priori* predictions. Thus, despite being less bold, dominant fish did tend to be larger, and grow faster (as inferred from both standard length

and weight). Although *dominance score* is observed for males only, this finding agrees with previous work on the same population where resource acquisition during dyadic interactions was used to assay dominance in both sexes (e.g., Wilson et al. 2013). Under our experimental rearing conditions, viability selection also tends to favour the dominant, faster growing males as predicted. Size and growth were not under significant viability selection in females, although the qualitative pattern of covariance with survival is not dissimilar. Note that following maturity, female fecundity scales tightly with size so we would expect strong (positive) selection on size through lifetime fitness in the wild. We find no evidence of selection on boldness in either sex, but stress that this may well be a consequence of the artificial conditions. For instance, increased predation risk is widely expected to impose a cost on bold behavioural strategies in wild populations. This was recently found in roach (*Rutilus rutilus*), with bolder fish being more susceptible to avian predation (Hulthén et al. 2017). It is also the case that personality traits can be under sexual selection (Schuett et al. 2010), that will not be apparent in our experiment.

Implications of genetic (co)variance

Our animal model analyses confirmed the presence of significant additive genetic effects contributing to observed phenotypic (co)variance. Therefore, there is evidence for genetic variance in boldness (*activity*) and male *dominance score*, as well as in *size* and *growth* (as measured by standard length and weight). The presence of genetic variance means that there is scope for adaptive evolution (Falconer and Mackay 1996) although the extent that the traits involved can respond independently to selection on them will depend on the genetic covariance / correlation structure in **G** (Walsh and Blows 2009). Overall, there was statistical support for between-trait genetic covariance although we acknowledge that pairwise genetic correlations between traits were characterised by high levels of uncertainty. Nonetheless, a number of correlations were nominally significant at $\alpha = 0.05$ (based on their estimated standard errors). This included, for example, the strong negative estimate of r_G between boldness and male dominance. In this case, and more

527 generally, the sign of the estimated genetic correlations matched that of the phenotypic correlations
528 in **I** as discussed above.
529 Two results from our genetic analysis are worth highlighting. The first is that the among-
530 individual variation in boldness previously reported (Boulton et al. 2014), is underpinned by
531 significant heritable variation. Although it has long been known that genes influence personality in
532 humans (e.g., Horn et al. 1976; Jang et al. 1996; Bouchard and McGue 2003) comparable studies on
533 animals, particularly wild ones, are still quite rare (but see: Drent et al. 2003; Dingemanse et al.
534 2004; van Oers et al. 2004). Our result thus adds to an emerging picture of genetic differences
535 among individuals being important determinants of animal personality generally (Dochtermann et al.
536 2015) and in fishes specifically (Dingemanse et al. 2012).
537 A second important result, and one more germane to our study rationale, is that we found
538 evidence for heritable variation in male *dominance score*. Although the estimate of h^2 is low (6.6%),
539 genetic correlations with other traits examined suggest that this could have important evolutionary
540 consequences. This is because if the resources won by a focal individual in competition depend on
541 its own genotype, it follows that they will also be influenced by the genotypes of competitors, giving
542 rise to indirect genetic effects (IGEs; Moore et al. 1997; Moore et al. 2002) on resource acquisition
543 and resource dependent traits. While IGE can accelerate selection responses in some contexts (Wolf
544 et al. 1998), under competition they are expected to constrain phenotypic responses of resource-
545 dependent traits (e.g., size, growth) to directional selection as a result of the evolution of a more
546 competitive social environment (Hadfield 2010; see Introduction). Equivalently, but conceptualised
547 slightly differently, IGEs reduce the genetic variance available to facilitate a selection response
548 (Wilson 2014). In the limiting case with a finite resource and constant population size, a response to
549 selection in a single trait will depend not on the total magnitude of V_A but on the portion that is
550 independent of competitive ability (and thus free from constraining IGE; Wilson 2014). If *dominance*
551 *score* is a valid measure of competitive ability, then from our estimate of **G** we can determine this as
552 $V_{A|Dom} / V_A$ where $V_{A|Dom}$ is the additive variance conditional on *dominance score*. Following Hansen

553 and Houle 2008, for a single trait y conditional on a single trait x we can calculate $V_{Ay|x} = V_{A(y)} -$
554 $COV_{A(y,x)} \cdot V_{A(x)}^{-1} \cdot COV_{A(x,y)}$. This yields, for example, values of $V_{A|Dom} / V_A$ of 38% and 42% respectively
555 for size and growth as measured by live weight.

556 The presence of heritable variation for dominance coupled to positive genetic covariance
557 with size and growth, thus implies that IGEs arising from competition could reduce observed
558 selection responses by >50% relative to naive predictions. Two caveats need stating however. First
559 this degree of constraint is likely to be an overestimate because it assumes that size and growth are
560 causally dependent on competitive ability but not *vice versa*. We have no way to assess causality
561 from our data although there are a wealth of contest studies in *Xiphophorus* showing that body size
562 is a key predictor of contest outcome (see Earley and Hsu 2008 for a review). Thus the (genetic)
563 covariances between dominance and size and growth are likely to reflect bi-directional causality.
564 Nonetheless, we have previously shown that the presence of dominant *X. birchmanni* males reduces
565 weight gain of subordinate tank mates (Wilson et al. 2013). The specific mechanism underpinning
566 this is unknown. Reduced growth rates in behaviourally subordinate fish could be an indirect
567 consequence of experiencing harassment and bullying from fish with dominant phenotypes (as
568 opposed to a direct consequence of obtaining less resource, e.g. food). It is well known that
569 physiological effects of chronic social stressors such as bullying can impact behaviour, health, life
570 history and survival in animal populations (e.g., Pickering and Pottinger 1989; Boonstra et al. 2001;
571 Barton 2002). Individual fitness may depend therefore not only on the ability to win resources (and
572 thus the phenotypic traits that promote resource winning) but also on the ability to cope with the
573 social stress imposed by socially dominant conspecifics.

574 A second caveat to note is that we did not explicitly model or estimate IGEs on traits
575 presumed consequent to competitive outcomes (i.e. size, growth). Ideally this would have allowed
576 us to verify the expected consequences of heritable dominance for downstream traits (as discussed
577 above). Although in principle it is relatively straightforward to estimate IGEs within a variance
578 partitioning animal model framework (Bijma et al. 2007), we were unable to obtain stable model

convergence from our data. We acknowledge that our breeding design is relatively small here and therefore data availability is likely limiting in this regard (as well as contributing to high uncertainty in the elements of **G**). In fact, a second generation of breeding was initially planned to allow further investigation of IGEs, as well as testing for GxE across the density treatments. However, this was precluded by poor reproductive success of surviving fish both during and following the current experiment. We therefore note that more complex quantitative genetic models are thus unlikely to provide further insight.

Conclusions

In summary, this study sought to investigate the direct effects of social competition on phenotype and fitness, test for among-individual variation in competitive ability (i.e. dominance) and investigate the multivariate genetic architecture linking traits putatively causal and consequent to dominance. We found that higher levels of competition caused reductions in growth and survival but had no effect on average behaviour. *Dominance score* was repeatable in males, and positively correlated with size, growth and survival at the among-individual level as predicted. However, while we found a correlation between personality (boldness) and dominance, the sign of this relationship was negative counter to our predictions. Thus, fish that were bolder actually tended to be less dominant (if male). This is compatible with the premise that personality is an important determinant of social dominance, but the direction of the relationship is something of an anomaly when set against the wider context of empirical studies of boldness. We also found evidence of genetic (co)variance underpinning observed phenotypic variation. Thus there is genetic integration between boldness, dominance, size and growth and these aspects of phenotype will not evolve independently under selection. Of particular significance is the conclusion that dominance is both heritable and genetically correlated with size and growth. Provided growth depends on the outcome of competition, heritable dominance leads to an expectation of indirect genetic effects that will act as constraints on selection responses.

605

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810

Table 1: Fixed effects retained for each trait showing results of conditional Wald F tests from univariate animal models including additive genetic (all traits) and permanent environment (all traits except longevity) effects as random.

Trait	**Effect**	**F**	**DF**	**P**
Activity	Intercept	1332	1, 12.1	<0.001
	Stack	7.80	5, 188.3	<0.001
	Sex	7.51	1, 345.8	0.006
	Trial	131	3, 921.6	<0.001
	Order	10.4	1, 1203.9	0.001
Dominance score	Intercept	12.8	1, 11.6	0.004
	Trial	3.26	15, 1220.4	<0.001
Standard length	Intercept	4196	1, 1.5	0.002
	Age	22.4	1, 494.3	<0.001
	Measure	462	12, 3335.5	<0.001
	Stack	2.43	5, 343.6	0.035
	Sex	0.362	1, 361.4	0.548
	GS	7.22	1, 3824.1	0.007
	Measure:Sex	13.9	12, 3384.3	<0.001
	ELD_{Part1}	31.5	1, 2583.6	<0.001
Weight	Intercept	486	1, 6.7	<0.001
	Age	19.7	1, 279.1	<0.001
	Measure	169	12, 3123.3	<0.001
	Stack	4.39	5, 330.1	0.001
	Sex	0.674	1, 359	0.412
	Measure:Sex	13.4	12, 3477	<0.001
	ELD_{Part1}	47.1	1, 837.1	<0.001
	LLD_{Part2}	11.8	1, 3782.9	0.001
	$(ELD:LLD)_{Part2}$	8.69	2, 2621.5	<0.001

Table 2: Repeatability (R) and heritability (h^2) estimates for all traits and survival. Estimates are from univariate models and standard errors in parentheses. Also shown are likelihood ratio tests of among-individual variance (V_I), additive genetic variance (V_A) and, for size traits only, among-individual (ID x Age) and additive genetic (G x Age) variance in growth.

Trait	**R** (SE)	**h^2** (SE)	**V_I**		**V_A**		**ID x Age**		**G x Age**	
			$\chi^2_{0,1}$	P	$\chi^2_{0,1}$	P	χ^2_2	P	χ^2_2	P
Activity	0.260 (0.034)	0.093 (0.056)	83.3	<0.001	4.97	0.013	-	-	-	-
Dominance score	0.242 (0.032)	0.066 (0.052)	180	<0.001	3.66	0.028	-	-	-	-
Standard length[1]	0.908 (0.008)	0.247 (0.106)	3370	<0.001	19.61	<0.001	3387	<0.001	17.6	<0.001
Weight[1]	0.839 (0.011)	0.144 (0.076)	2030	<0.001	14.14	<0.001	3587	<0.001	11.2	0.004
Survival	-	0.016 (0.015)	-	-	1.93	0.082	-	-	-	-

[1]Since random regression models are used R and h^2 estimates here are for size at mean observed age of fish in the study (= 294 days)

Table 3: Results of survival analysis using proportional hazards regression model

Predictor	Coefficient	SE	Exp (Coefficient)	z	P
Stack (A)	NA				
Stack (B)	1.087	0.274	2.965	3.971	<0.001
Stack (D)	0.270	0.271	1.310	0.995	0.320
Stack (E)	0.808	0.281	2.244	2.879	0.004
Stack (F)	0.120	0.305	1.127	0.392	0.695
Stack (G)	-0.185	0.304	0.831	-0.607	0.544
Sex (Female)	NA				
Sex (male)	0.204	0.183	1.226	1.114	0.265
GS	1.720	0.174	5.584	9.885	<0.001
SR	0.829	0.388	2.290	2.134	0.033
Treatment (LL)	NA				
Treatment (LH)	0.597	0.225	1.817	2.652	0.008
Treatment (HL)	-0.237	0.241	0.789	-0.984	0.325
Treatment (HH)	0.225	0.240	1.253	0.941	0.347

Table 4: Estimates of among-individual (**ID**) and additive genetic (**G)** matrices. Estimates are derived from analyses of both sexes combined (see main text) noting that *Dominance score* is sex-limited (males only). Among-individual or additive genetic variances are shown on the diagonal (light grey shading), with between-trait covariances below the diagonal with corresponding correlations above. Standard errors are shown in parentheses for all estimates and bold font denotes individual correlations deemed nominally significant at $\alpha = 0.05$ (based on $|\text{correlation/SE}| \geq 1.96$). The expanded estimate **ID** is shown with *Survival* included as a further response to estimate the selection differentials on each trait (dark grey shading) and corresponding trait-fitness correlations (black shading).

	TRAIT	*Activity*	*Dominance score*	*Standard length*	*Weight*	*Growth$_{SL}$*	*Growth$_{WT}$*	*Survival*
ID	*Activity*	0.198 (0.029)	**-0.410 (0.104)**	**-0.472 (0.065)**	**-0.513 (0.063)**	**-0.378 (0.069)**	**-0.442 (0.067)**	0.013 (0.074)
	Dominance score	-0.084 (0.023)	0.213 (0.032)	**0.632 (0.063)**	**0.711 (0.055)**	**0.493 (0.069)**	**0.649 (0.059)**	**0.233 (0.082)**
	Standard length	-0.010 (0.017)	0.138 (0.020)	0.225 (0.017)	**0.921 (0.009)**	**0.576 (0.037)**	**0.648 (0.033)**	**0.189 (0.051)**
	Weight	-0.123 (0.019)	0.177 (0.022)	0.236 (0.019)	0.292 (0.022)	**0.743 (0.026)**	**0.867 (0.015)**	**0.358 (0.048)**
	Growth$_{SL}$	-5.08×10^{-4} (1.05×10^{-4})	6.88×10^{-4} (1.20×10^{-4})	8.25×10^{-4} (9.04×10^{-5})	1.21×10^{-3} (1.10×10^{-4})	9.13×10^{-6} (7.07×10^{-7})	**0.926 (0.008)**	**0.391 (0.048)**
	Growth$_{WT}$	-8.54×10^{-4} (1.53×10^{-4})	1.30×10^{-3} (1.77×10^{-4})	1.34×10^{-3} (1.34×10^{-4})	2.04×10^{-3} (1.68×10^{-4})	1.22×10^{-5} (9.76×10^{-7})	1.89×10^{-5} (1.47×10^{-6})	**0.482 (0.044)**
	Survival	0.005 (0.027)	0.087 (0.032)	0.073 (0.021)	0.157 (0.025)	9.59×10^{-4} (1.46×10^{-4})	1.70×10^{-3} (2.18×10^{-4})	0.659 (0.050)
G	*Activity*	0.079 (0.049)	**-0.845 (0.361)**	-0.280 (0.344)	**-0.580 (0.287)**	-0.378 (0.323)	**-0.625 (0.286)**	
	Dominance score	-0.042 (0.032)	0.032 (0.032)	0.424 (0.422)	**0.795 (0.363)**	0.432 (0.468)	0.736 (0.413)	
	Standard length	-0.020 (0.028)	0.0195 (0.023)	0.066 (0.030)	**0.852 (0.093)**	-0.176 (0.330)	-0.080 (0.362)	
	Weight	-0.038 (0.029)	0.0334 (0.024)	0.052 (0.028)	0.056 (0.0290)	0.118 (0.346)	0.367 (0.313)	
	Growth$_{SL}$	1.95×10^{-4} (2.02×10^{-4})	1.42×10^{-4} (1.76×10^{-4})	-8.33×10^{-5} (1.52×10^{-4})	5.11×10^{-5} (1.60×10^{-4})	3.39×10^{-6} (1.55×10^{-6})	**0.890 (0.072)**	
	Growth$_{WT}$	3.64×10^{-4} (2.67×10^{-4})	2.72×10^{-4} (2.24×10^{-4})	-4.28×10^{-5} (1.89×10^{-5})	1.79×10^{-4} (2.11×10^{-4})	3.40×10^{-6} (2.38×10^{-6})	4.30×10^{-6} (2.38×10^{-6})	

Figure 1: Average growth trajectories showing mean standard length (A – females, B - males) and live mass (C - females, D - males) by month from the start of the experiment for fish in each density treatment regime. Bars indicate standard deviations and dashed line indicates the point of treatment switch.

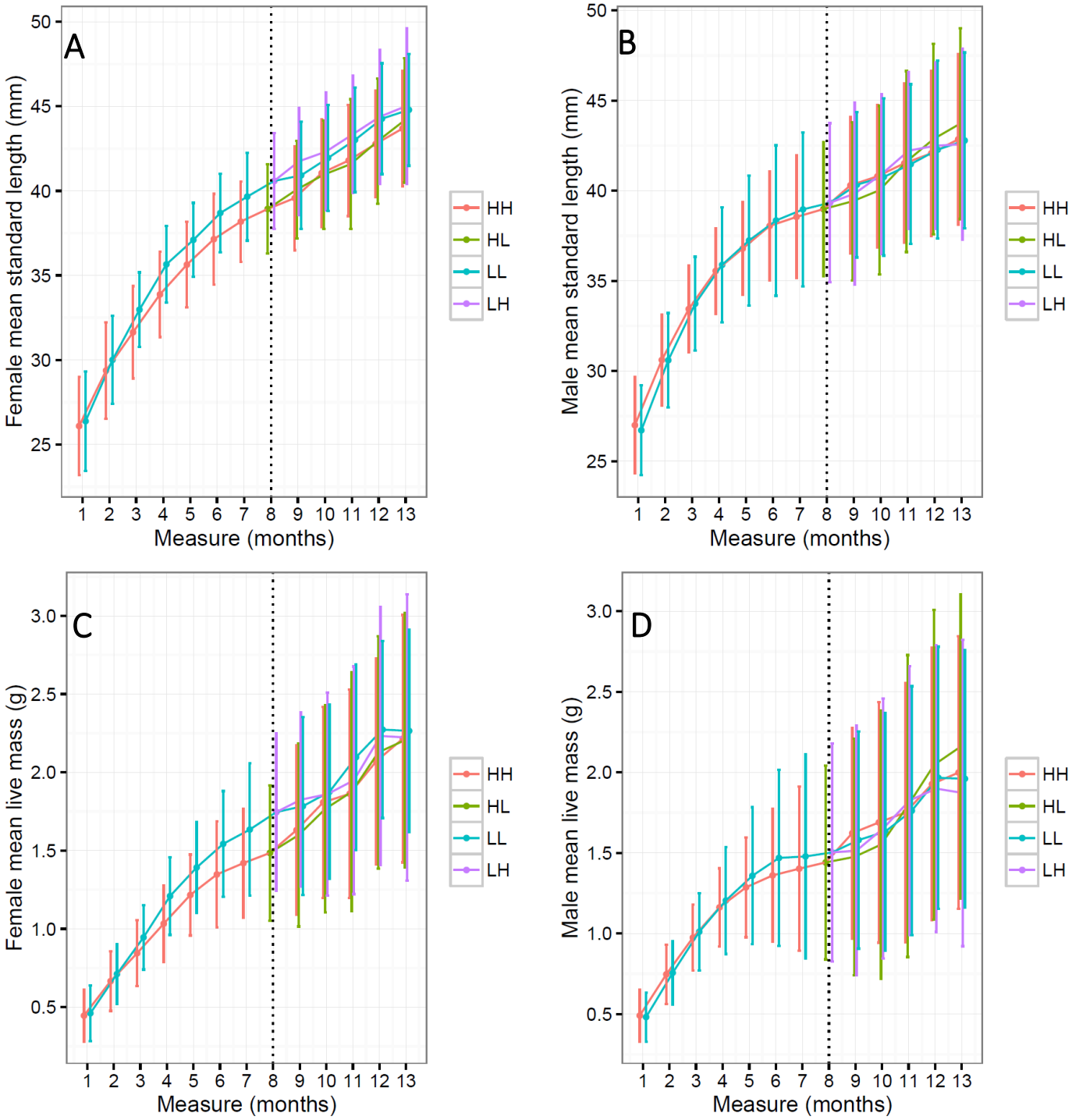
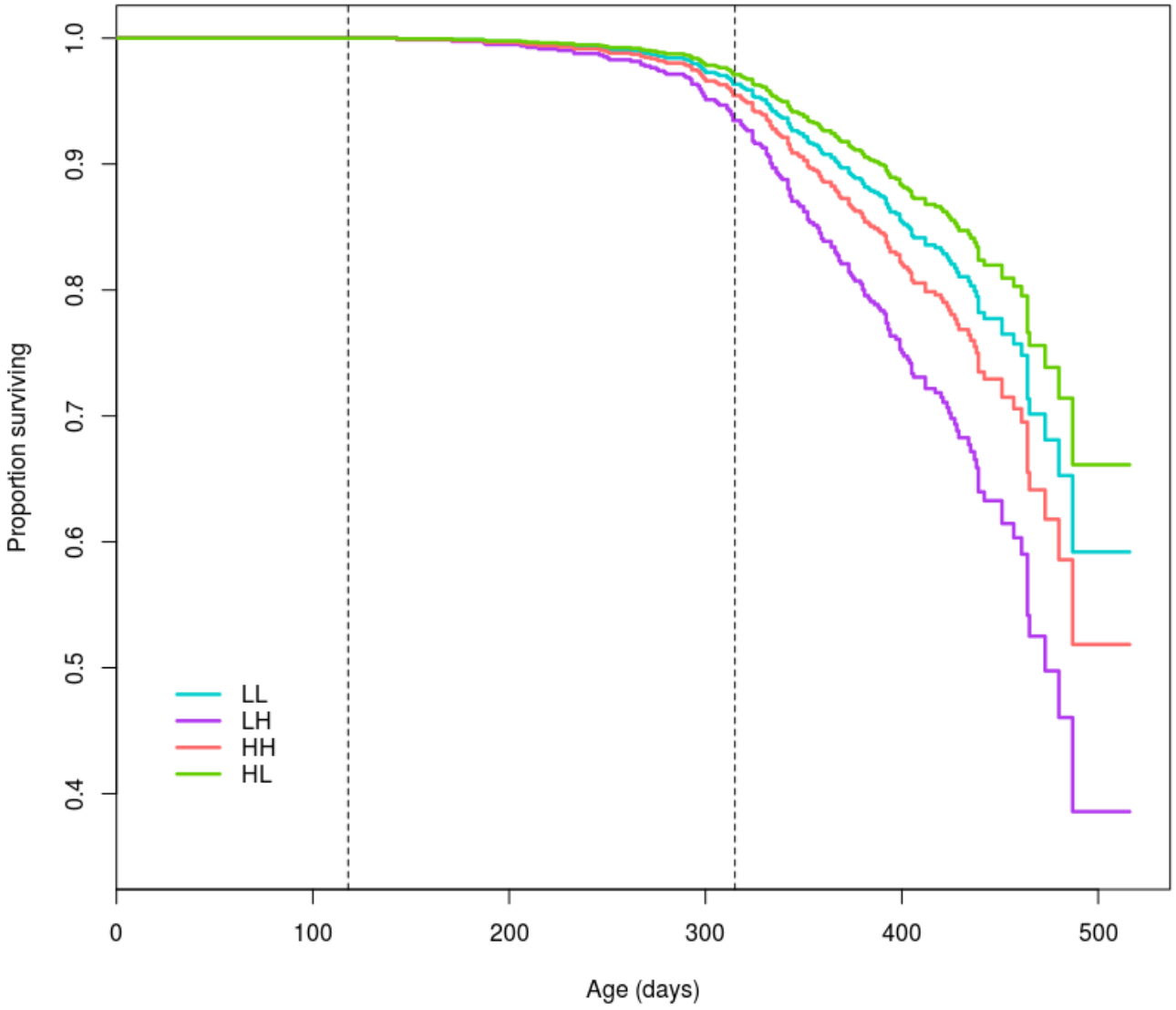


Figure 2. Predicted survival curves by total density treatment. From left to right, the dashed vertical lines denote mean age at start of the experiment, and mean age at measure 8 (end of part1).



Supplemental Materials

Supplemental table 1: Estimated coefficients from univariate animal models of each trait. Standard errors are shown in parentheses (but not for reference levels of the factors that have an effect size of zero). See main text for statistical inference and details of units for traits and predictor variables. Note also that age-dependence of size traits (standard length and weight) is a function of both *Age* and *Measure* (and interactions involving the latter).

Trait	**Effect** (factor level)	**Coefficient** (SE)
Activity	Order	-0.131 (0.041)
	Trial (1)	0 (-)
	Trial (2)	16.0 (1.05)
	Trial (3)	18.5 (1.11)
	Trial (4)	18.0 (1.22)
	Sex (M)	-3.32 (1.21)
	Stack (A)	0 (-)
	Stack (B)	1.29 (2.30)
	Stack (D)	9.07 (2.25)
	Stack (E)	10.2 (2.29)
	Stack (F)	6.10 (2.35)
	Stack (G)	0.411 (2.24)
	Intercept	62.1 (2.55)
Dominance score	Trial (1)	0 (-)
	Trial (2)	-0.453 (0.981)
	Trial (3)	-0.899 (1.00)
	Trial (4)	-1.65 (1.10)
	Trial (5)	-0.080 (1.25)
	Trial (6)	1.42 (1.35)
	Trial (7)	2.81 (1.35)
	Trial (8)	2.60 (1.35)
	Trial (9)	3.12 (1.36)
	Trial (10)	5.95 (1.37)
	Trial (11)	3.09 (1.86)
	Trial (12)	2.49 (1.86)
	Trial (13)	-0.536 (1.86)
	Trial (14)	1.71 (1.88)
	Trial (15)	2.57 (2.19)
	Trial (16)	4.49 (3.27)
	Intercept	2.24 (0.956)
Standard length	ELD_{Part1} (H)	0 (-)
	ELD_{Part1} (L)	0.635 (0.113)
	Measure:Sex (1:Male)	0 (-)
	Measure:Sex (2:Male)	0.211 (0.154)
	Measure:Sex (3:Male)	0.306 (0.175)
	Measure:Sex (4:Male)	-0.037 (0.205)
	Measure:Sex (5:Male)	-0.258 (0.240)

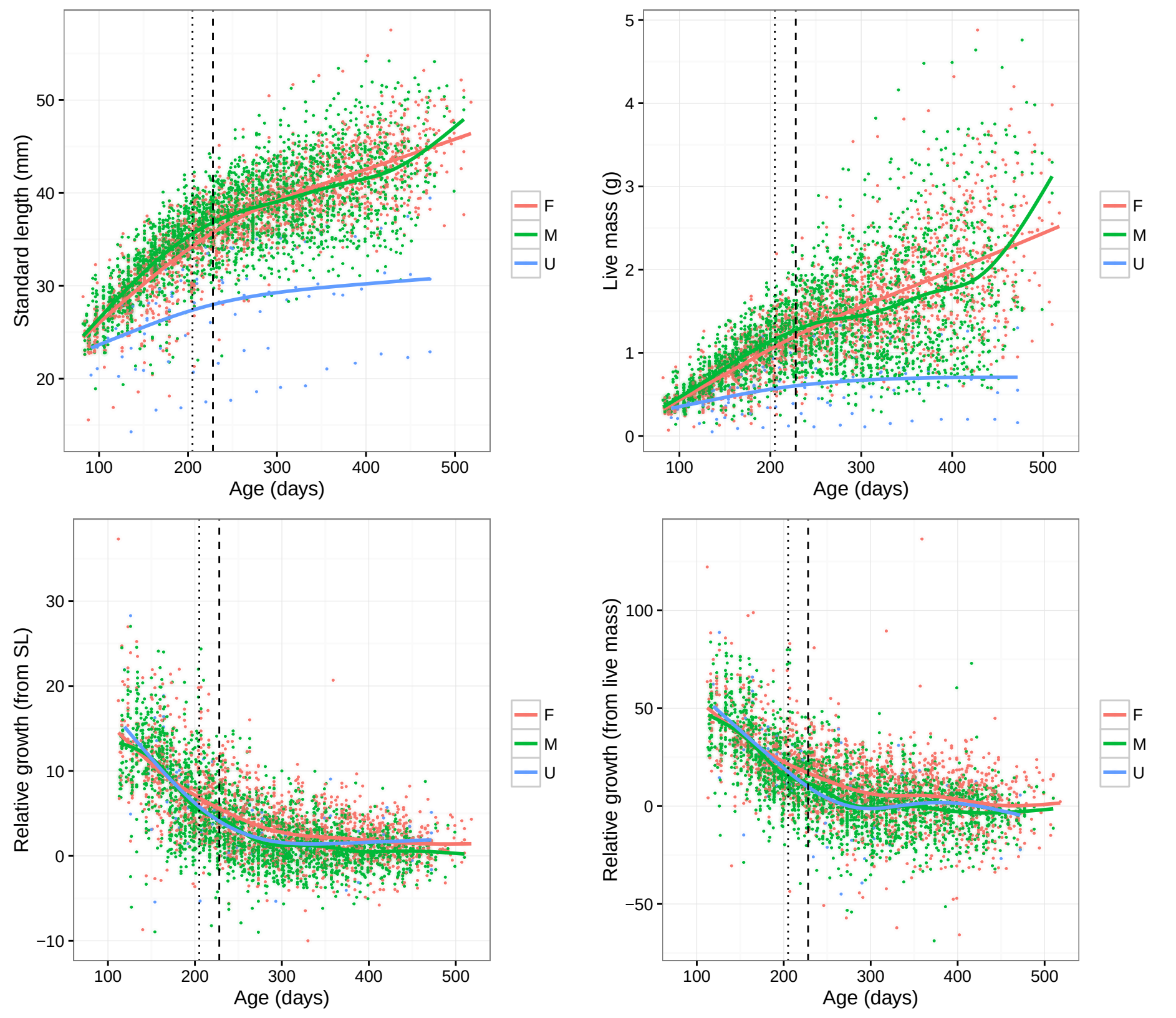
	Measure:Sex (6:Male)	-0.622 (0.281)
	Measure:Sex (7:Male)	-1.04 (0.323)
	Measure:Sex (8:Male)	-1.69 (0.369)
	Measure:Sex (9:Male)	-1.96 (0.412)
	Measure:Sex (10:Male)	-2.48 (0.464)
	Measure:Sex (11:Male)	-3.00 (0.512)
	Measure:Sex (12:Male)	-3.65 (0.563)
	Measure:Sex (13:Male)	-4.28 (0.607)
	GS	0.337 (0.126)
	Sex (M)	-0.275 (0.267)
	Stack (A)	0 (-)
	Stack (B)	0.975 (0.668)
	Stack (D)	1.44 (0.648)
	Stack (E)	0.704 (0.672)
	Stack (F)	2.20 (0.802)
	Stack (G)	1.10 (0.744)
	Measure (1)	0 (-)
	Measure (2)	2.00 (0.375)
	Measure (3)	3.37 (0.612)
	Measure (4)	4.98 (0.866)
	Measure (5)	5.51 (1.13)
	Measure (6)	6.16 (1.40)
	Measure (7)	6.24 (1.67)
	Measure (8)	6.52 (1.94)
	Measure (9)	6.81 (2.21)
	Measure (10)	6.80 (2.51)
	Measure (11)	6.91 (2.78)
	Measure (12)	7.18 (3.06)
	Measure (13)	7.36 (3.31)
	Age	0.046 (0.010)
	Intercept	32.98 (1.41)
Weight	$(LLD:ELD)_{Part2}$ (H:H)	0 (-)
	$(LLD:ELD)_{Part2}$ (H:L)	0 (-)
	$(LLD:ELD)_{Part2}$ (L:H)	-0.165 (0.04)
	$(LLD:ELD)_{Part2}$ (L:L)	-0.061 (0.038)
	LLD_{Part2} (H)	0 (-)
	LLD_{Part2} (L)	0.024 (0.033)
	ELD_{Part1} (H)	0 (-)
	ELD_{Part1} (L)	0.050 (0.020)
	Measure:Sex (1:Male)	0 (-)
	Measure:Sex (2:Male)	0.014 (0.027)
	Measure:Sex (3:Male)	0.030 (0.030)
	Measure:Sex (4:Male)	-0.011 (0.034)
	Measure:Sex (5:Male)	-0.052 (0.039)
	Measure:Sex (6:Male)	-0.097 (0.045)
	Measure:Sex (7:Male)	-0.160 (0.052)

Measure:Sex (8:Male)	-0.242 (0.059)
Measure:Sex (9:Male)	-0.315 (0.065)
Measure:Sex (10:Male)	-0.38 (0.073)
Measure:Sex (11:Male)	-0.490 (0.081)
Measure:Sex (12:Male)	-0.618 (0.089)
Measure:Sex (13:Male)	-0.712 (0.096)
Sex (M)	-0.013 (0.027)
Stack (A)	0 (-)
Stack (B)	0.120 (0.060)
Stack (D)	0.184 (0.058)
Stack (E)	0.106 (0.060)
Stack (F)	0.213 (0.073)
Stack (G)	0.077 (0.068)
Measure (1)	0 (-)
Measure (2)	0.108 (0.053)
Measure (3)	0.188 (0.073)
Measure (4)	0.345 (0.097)
Measure (5)	0.450 (0.123)
Measure (6)	0.516 (0.150)
Measure (7)	0.540 (0.178)
Measure (8)	0.593 (0.205)
Measure (9)	0.717 (0.233)
Measure (10)	0.728 (0.264)
Measure (11)	0.823 (0.291)
Measure (12)	0.990 (0.321)
Measure (13)	0.974 (0.346)
Age	0.004 (0.001)
Intercept	1.05 (0.141)

Supplemental Table 2: Estimates among-individual (**ID**) matrices for males ($\mathbf{ID_M}$) and females ($\mathbf{ID_F}$) separately. Among-individual variances are shown on the shaded diagonal, with between-trait covariances below and correlations above. Standard errors are shown in parentheses and bold font denotes individual off diagonal elements that are nominally significant at $\alpha = 0.05$ (based on $|\text{estimate/SE}| \geq 1.96$). *Dominance scores* available for males only. Directly observed traits (*Activity, Dominance score, Standard length and Weight*) were scaled to standard deviation units prior to modelling. *Survival* was mean standardised for inclusion in the expanded **ID** matrix estimates.

		Activity	*Dominance score*	*Standard length*	*Weight*	*Growth$_{SL}$*	*Growth$_{WT}$*	*Survival*
$\mathbf{ID_M}$	*Activity*	0.205 (0.040)	**-0.461 (0.108)**	**-0.569 (0.078)**	**-0.578 (0.078)**	**-0.407 (0.088)**	**-0.463 (0.086)**	-3.00×10^{-4} (0.098)
	Dominance score	-0.099 (0.027)	0.226 (0.034)	**0.644 (0.063)**	**0.717 (0.056)**	**0.558 (0.069)**	**0.677 (0.059)**	**0.333 (0.083)**
	Standard length	-0.131 (0.024)	0.156 (0.024)	0.257 (0.026)	**0.934 (0.010)**	**0.685 (0.038)**	**0.717 (0.036)**	**0.308 (0.064)**
	Weight	-0.159 (0.029)	0.207 (0.029)	0.288 (0.030)	0.370 (0.037)	**0.818 (0.025)**	**0.901 (0.015)**	**0.492 (0.054)**
	Growth$_{SL}$	-6.25×10^{-4} (1.56×10^{-4})	8.99×10^{-4} (1.57×10^{-4})	1.18×10^{-3} (1.46×10^{-4})	1.68×10^{-3} (1.85×10^{-4})	1.15×10^{-5} (1.15×10^{-6})	**0.942 (0.009)**	**0.493 (0.055)**
	Growth$_{WT}$	-1.02×10^{-4} (2.28×10^{-4})	1.57×10^{-3} (2.33×10^{-4})	1.77×10^{-3} (2.13×10^{-4})	2.66×10^{-3} (2.77×10^{-4})	1.55×10^{-5} (1.59×10^{-6})	2.37×10^{-5} (2.36×10^{-6})	**0.608 (0.047)**
	Survival	-1.05×10^{-4} (0.039)	0.138 (0.039)	0.136 (0.033)	0.260 (0.042)	1.45×10^{-3} (2.35×10^{-4})	2.57×10^{-3} (3.54×10^{-4})	0.757 (0.076)
$\mathbf{ID_F}$	*Activity*	0.192 (0.043)	-	-0.216 (0.112)	**-0.31 (0.111)**	**-0.298 (0.116)**	**-0.361 (0.111)**	-0.027 (0.118)
	Dominance score	-	-	-	-	-	-	-
	Standard length	-0.034 (0.019)	-	0.131 (0.016)	**0.920 (0.015)**	0.163 (0.090)	**0.466 (0.073)**	0.074 (0.087)
	Weight	-0.057 (0.022)	-	0.139 (0.018)	0.174 (0.022)	**0.442 (0.075)**	**0.747 (0.044)**	0.145 (0.088)
	Growth$_{SL}$	-3.07×10^{-4} (1.29×10^{-4})	-	1.39×10^{-4} (7.90×10^{-5})	4.32×10^{-4} (9.68×10^{-5})	5.51×10^{-6} (6.98×10^{-7})	**0.899 (0.018)**	**0.188 (0.093)**
	Growth$_{WT}$	-5.78×10^{-4}	-	6.07×10^{-4} (1.29×10^{-4})	1.12×10^{-3} (1.66×10^{-4})	7.59×10^{-6} (1.02×10^{-6})	1.30×10^{-5} (1.65×10^{-6})	**0.241 (0.090)**
	Survival	-0.009 (0.039)	-	0.020 (0.024)	0.045 (0.0284)	3.29×10^{-4} (1.70×10^{-4})	6.47×10^{-4} (2.61×10^{-4})	0.558 (0.067)

Supplemental Figure 1: Observed size (A - standard length, B - live mass) and relative growth (C,D) by age for female (red), male (green) and fish of unknown sex (blue). Each point represents a single observation and lines show smoothed fits through the data (pooled across treatments) for illustrative purposes only. Note the data contain repeat records and no statistical inference is intended. Relative growth rate at time *t* was defined as $100*\ln(size_t/size_{t\text{-}1month})$ with size measured as either standard length (C) or live mass (D). Dotted and dashed vertical lines indicate average age of maturity in males (205 days) and females (228 days) respectively, with maturity status inferred from external morphology (see Boulton et al. 2016 for details).


Standard length (mm)
Age (days)
Live mass (g)
Age (days)
Relative growth (from SL)
Age (days)
Relative growth (from live mass)
Age (days)
F
M
U