

On the genetic architecture of rapidly adapting and convergent life history traits in guppies

Author list:

James R Whiting¹, Josephine R Paris¹, Paul J Parsons^{1,2}, Sophie Matthews¹, Yuridia Reynoso⁴,
Kimberly A. Hughes³, David Reznick⁴, Bonnie A Fraser¹

Author Affiliations:

- 1) Department of Biosciences, University of Exeter, Stocker Road, Exeter EX4 4QD, UK
- 2) Department of Animal and Plant Sciences, University of Sheffield, Alfred Denny Building, University of Sheffield, Western Bank, Sheffield S10 2TN, UK
- 3) Department of Biological Science, Florida State University, Tallahassee, Florida, 32303, USA
- 4) Department of Biology, University of California Riverside, Riverside, California, 92521, USA

Corresponding Author:

James R Whiting: jwhiting2315@gmail.com

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1 ABSTRACT

2 The genetic basis of traits can shape and constrain how adaptation proceeds in nature; rapid
3 adaptation can be facilitated by polygenic traits, whereas polygenic traits may restrict re-use
4 of the same genes in adaptation (genetic convergence). The rapidly evolving life histories of
5 guppies in response to predation risk provide an opportunity to test this proposition.
6 Guppies adapted to high- (HP) and low-predation (LP) environments in northern Trinidad
7 evolve rapidly and convergently among natural populations. This system has been studied
8 extensively at the phenotypic level, but little is known about the underlying genetic
9 architecture. Here, we use an F2 QTL design to examine the genetic basis of seven (five
10 female, two male) guppy life history phenotypes. We use RAD-sequencing data (16,539
11 SNPs) from 370 male and 267 female F2 individuals. We perform linkage mapping, estimates
12 of genome-wide and per-chromosome heritability (multi-locus associations), and QTL
13 mapping (single-locus associations). Our results are consistent with architectures of many-
14 loci of small effect for male age and size at maturity and female interbrood period. Male
15 trait associations are clustered on specific chromosomes, but female interbrood period
16 exhibits a weak genome-wide signal suggesting a potentially highly polygenic component.
17 Offspring weight and female size at maturity are also associated with a single significant QTL
18 each. These results suggest rapid phenotypic evolution of guppies may be facilitated by
19 polygenic trait architectures, but these may restrict gene-reuse across populations, in
20 agreement with an absence of strong signatures of genetic convergence from recent
21 population genomic analyses of wild HP-LP guppies.
22

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23 INTRODUCTION

24 Recent evidence that phenotypes can evolve rapidly and often with surprising repeatability
25 (convergence) has led to a re-evaluation of our expectations surrounding adaptation in
26 nature. Particularly, understanding the genetic architecture of traits associated with both
27 rapid adaptation and convergence can allow for insights into how adaptive variation may be
28 maintained and be made available to respond to sudden changes in selection. Such research
29 is important not only due to the current global circumstances of rapid environmental
30 change, but also to understand adaptation more generally. Quantitative traits can have
31 architectures made up of many loci of small effect (polygenic), single loci of large effect
32 (monogenic), or an intermediate of these (oligogenic). There are currently theoretical
33 expectations surrounding which of these are most likely to underlie rapidly adapting
34 (Pritchard *et al.*, 2010; Jain and Stephan, 2017b) and/or convergent phenotypes (Yeaman *et*
35 *al.*, 2018) but empirical evidence is only starting to accumulate.

36

37 Polygenic traits may facilitate rapid adaptation by providing a substrate of standing genetic
38 variation to be exploited (Jain and Stephan, 2015, 2017a; Barghi *et al.*, 2019), enabling
39 populations to adapt to shifting fitness optima by many small changes (Jain and Stephan,
40 2017b). Indeed, Fisher's fundamental theorem states that the rate change of mean fitness is
41 equal to the amount of additive genetic variance for fitness (Fisher, 1930). Conversely, rapid
42 adaptation of oligogenic traits is expected to occur through selective sweeps, which can
43 result in less precise shifts across the fitness landscape or 'overshooting' incurring genetic
44 load (Buffalo and Coop, 2019). On the basis of this cost, it is expected that most rapid
45 adaptation of modest changes to trait means should occur using many loci of small effect,
46 with the exception of instances in which sudden environmental change is so extreme as to

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47 be lethal and affect absolute fitness (Bell, 2013; Whitehead *et al.*, 2017). Recent examples of
48 suspected polygenic bases involved in rapid adaptation include shell morphologies of
49 *Littorina* periwinkles (Westram *et al.*, 2018), immunity phenotypes in response to
50 myxomatosis in rabbits (Alves *et al.*, 2019), and killifish adapting to anthropogenic thermal
51 effluent runoff (Dayan *et al.*, 2019).

52

53 Similarly, there are expectations regarding the interactions between genetic architecture
54 and convergent evolution. Whilst convergent phenotypes can arise through non-convergent
55 genetic routes, the likelihood of convergent phenotypes having convergent genetics is
56 expected to vary according to trait architecture. For example, polygenic traits reduce the
57 likelihood of evolution of the same genes by increasing redundancy in the mapping of
58 genotype to phenotype (Yeaman *et al.*, 2018; Barghi *et al.*, 2020; Láruson *et al.*, 2020). In
59 contrast, if genetic architectures are simple and composed of few large effect loci, reduced
60 redundancy can funnel adaptation through repeatable genetic paths. Many of the most
61 notable examples of genetic convergence are single loci of large effect (Stern, 2013),
62 including the *eda* gene associated with marine-freshwater armour plate phenotypes in
63 three-spined stickleback (Colosimo, 2005), and the *optix* gene associated with wing
64 patterning across *Heliconius* species (Reed *et al.*, 2011).

65

66 The guppies of northern Trinidad are a model system for studying phenotypic adaptation,
67 which has provided empirical evidence for both rapid adaptation and convergent evolution.
68 In this system, barrier waterfalls within many rivers have created replicated
69 downstream/high-predation (HP) and upstream/low-predation (LP) habitats. Each river
70 contains HP- and LP-adapted guppy populations that have independently evolved

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71 convergent LP phenotypes in predator-free upstream environments. LP populations are
72 typically longer-lived, exhibiting larger adult sizes, reduced brood size, longer time to reach
73 maturity and longer interbrood period than their HP counterparts (Reznick, 1982; Reznick *et*
74 *al.*, 2001; Torres Dowdall *et al.*, 2012). Experimental translocations of HP guppies to
75 previously uncolonised LP environments further demonstrated that LP life history
76 phenotypes evolve rapidly (Endler, 1980; Reznick and Bryga, 1987; Reznick *et al.*, 1990,
77 1997, 2019; Gordon *et al.*, 2009). Guppies raised under laboratory conditions for multiple
78 generations continue to exhibit differences between HP and LP phenotypes, indicating these
79 traits have a heritable genetic basis (Reznick, 1982; Torres Dowdall *et al.*, 2012). Beyond this
80 however, and despite the wealth of knowledge regarding life history evolution in these
81 populations, little is known about the genetic architecture of these traits.

82

83 Life history traits are typically quantitative and are commonly involved in adaptation to
84 novel or changing environments. Previous studies exploring the genetic basis of life history
85 traits have documented everything from highly polygenic traits, such as clutch size and egg
86 mass in great tits (Santure *et al.*, 2013) and weight of Soay sheep (Bérénos *et al.*, 2015), to
87 traits with single loci explaining a large proportion of phenotypic variance, such as age at
88 maturity in atlantic salmon (*Salmo salar* L) (Ayllon *et al.*, 2015; Barson *et al.*, 2015) and other
89 salmonids (Moghadam *et al.*, 2007; Kodama *et al.*, 2018). Life history traits can often exhibit
90 genetic covariance, with different traits sharing aspects of their genetic architecture, which
91 can have important implications for pleiotropic constraint during adaptation (Hall *et al.*,
92 2006).

93

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94 There are various avenues for exploring the genetic architecture of quantitative traits. For
95 example, single locus quantitative trait locus (QTL) analyses or Genome-Wide Association
96 Studies (GWAS) are now commonplace. However, these approaches can inflate the
97 prominence of single loci and their inherent bias against multi-locus models has come under
98 scrutiny (Rockman, 2012; Slate, 2013). Indeed, multi-locus analyses have resolved some
99 empirical inconsistencies, such as the “missing heritability crisis” (Manolio *et al.*, 2009),
100 subsequently returning much higher estimates of heritability compared with the sum of
101 single-locus outliers for classic quantitative traits such as human height (Yang *et al.*, 2010;
102 Yang, Manolio, *et al.*, 2011). Extensions of these multi-locus approaches have partitioned
103 phenotypic variance into specific chromosomes, with correlations between chromosome
104 size (as a proxy for number of functional loci) and partitioned phenotypic variance taken as
105 evidence for highly polygenic traits (Santure *et al.*, 2013; Bérénos *et al.*, 2015; Kemppainen
106 and Husby, 2018a). The rising prominence of multi-locus models has started to bring
107 empirical evidence back in line with Fisher’s prediction of the importance of many loci of
108 small effect, often dubbed the “infinitesimal model” (Fisher, 1918; Barton *et al.*, 2017). It is
109 therefore important to explore analyses that allow for all possible trait architectures.
110

111 Using an F2 breeding cross design, we examine the genetic basis of seven life history traits
112 in guppies: female age (1) and size (2) at first brood, first brood size (3), interbrood period
113 (4), average dry offspring weight in the first brood (5), and male age (6) and size (7) at
114 maturity. Our aims are to assess the relative extents to which different facets of life history
115 traits have significant genetic elements, and whether guppy life history traits are better
116 explained by polygenic, oligogenic, or monogenic models. By exploring the genetic bases of
117 these traits within a system for which rapid adaptation and convergent evolution is already

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118 well-documented, we can better understand the role of quantitative genetic architecture in
119 these processes.

120

121 **METHODS**

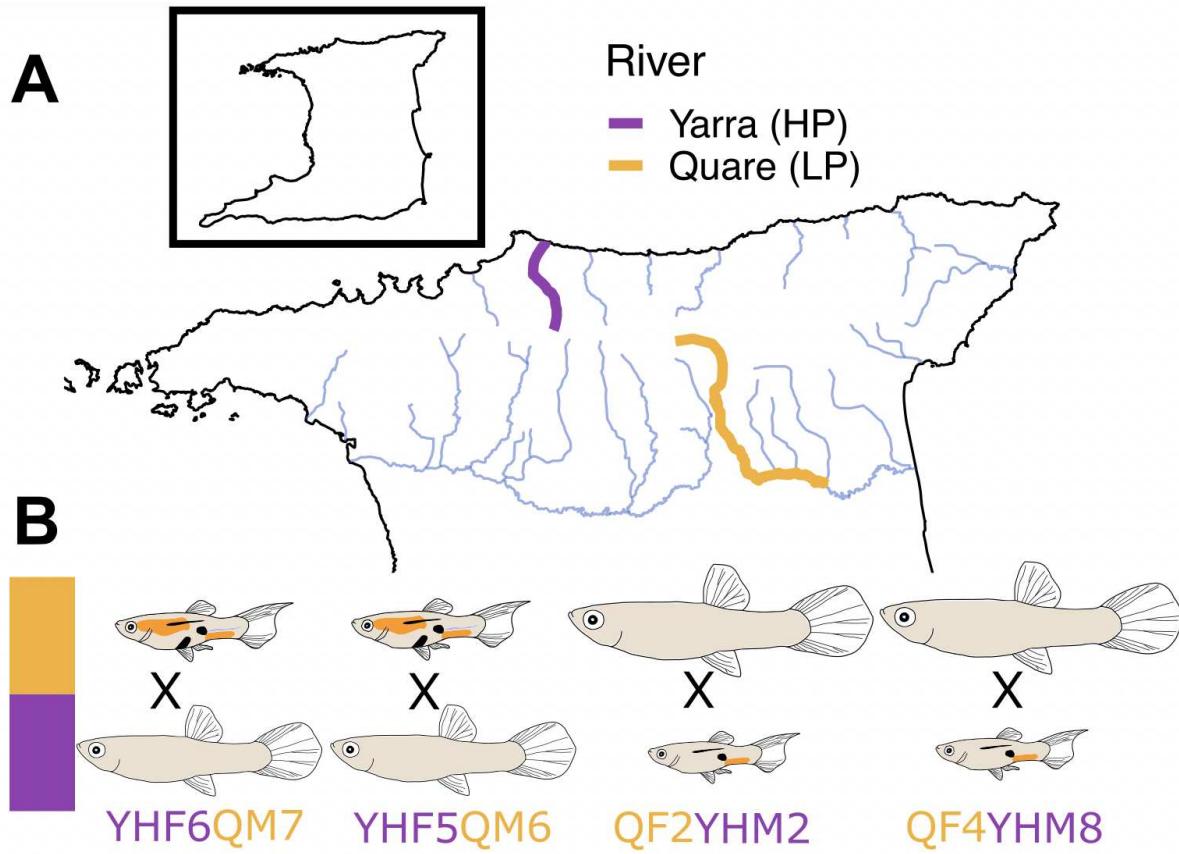
122 *Crosses*

123 Fish were second and third generation lab-reared individuals from an HP site in the Yarra
124 river (680415E, 1193791N) and an LP site in the Quare river (696907E, 1181003N) (Figure 1).
125 These populations have demonstrable HP-LP life history phenotypes (Table S1), and have
126 been studied extensively in prior work (Reznick, 1982; Reznick *et al.*, 1996, 2004, 2005).

127 Four F2 full-sib intercrosses were performed. Two crosses were performed for each cross
128 direction in which wild-caught LP males were crossed with wild-caught HP females and vice
129 versa. F1s were mated within cross and F2s were phenotyped and genotyped. Grandparents
130 were also genotyped.

131

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132

133 **Figure 1:** Sampling rivers in Trinidad and cross design. Sampling rivers are
134 highlighted along with a number of major rivers from northern Trinidad's three
135 drainages (**A**). Four families were produced from eight grandparents (**B**), two for
136 each cross direction. Males (smaller, colourful) and females (larger, uncoloured) in
137 panel **B** also highlight common morphological differences between HP (smaller, less
138 colourful) and LP individuals.

139

140 **Phenotyping and Phenotype GLMs**

141 Life history phenotyping and rearing followed (Reznick, 1982); full rearing details are
142 available in the supplementary materials. Size of females and males was measured under a
143 dissecting scope with Vernier calipers following MS-222 anaesthetisation. Based on the
144 allometric dependency of female brood size, we took residual brood size as the residual
145 difference between observed and linear-predicted brood size based on size. Male age at
146 maturity was judged from the development of the apical hook. Interbrood period was
147 scored as days between first and second parturition. Offspring weight was recorded as the

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148 mean dry weight of individuals from the first litter (data from the second litter was highly
149 correlated). Where necessary, phenotypes were log-transformed to improve fit for
150 normality assumptions.

151

152 Rearing (mean temperature and date of birth (DOB)) and family effects on phenotypes were
153 explored using generalized linear models (GLM) in R (v4 (R Core Team, 2020)). DOB was
154 included as a proxy for subtle unmeasured changes in rearing conditions over time. We used
155 backwards model selection implemented in *step()*, starting from an additive model including
156 family, temperature, and DOB. Relevant model assumptions were checked by comparing
157 residuals to simulated residuals in the R package *DHARMA* (Hartig, 2020). Final model term
158 significance was determined by comparing models with and without each independent
159 variable using *drop1()* in terms of Δ AIC and using F-tests. Where model assumptions could
160 not be met, final model terms were taken and significance ($p < 0.05$) of Spearman's rank
161 correlations were used to confirm model effects. Adjusted partial R-squared was estimated
162 for all final model variables with the R package *rsq* (Zhang, 2020) using the variance-
163 function-based type.

164

165 **Genotyping**

166 Genomic DNA was extracted from fin clips using an ammonium acetate extraction method
167 (Nicholls *et al.*, 2000; Richardson *et al.*, 2001). We genotyped each individual using a RAD-
168 seq library preparation method adapted from Poland and colleagues (Miller *et al.*, 2007;
169 Baird *et al.*, 2008; Poland *et al.*, 2012); full genotyping details are available in the
170 supplementary materials. Of all 661 individuals used in the final analysis, 61 were
171 sequenced two or three times in separate libraries to account for low coverage ("merged")

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172 individuals in Table S2). To ensure optimal coverage, and reduce PCR duplicate effects, all
173 eight grandparents were sequenced four times in four separate PCR reactions and
174 sequencing libraries. Of the total 653 F2s, 637 (370 males, 267 females) were used for
175 phenotype analyses due to missing phenotypes for 16 individuals.

176

177 ***Bioinformatic processing***

178 Raw read data were trimmed and adaptors removed using cutadapt (Martin, 2011). Stacks
179 v2.5 was used for all downstream processing (Rochette *et al.*, 2019). Trimmed read data
180 were used as input in process_radtags, with options to remove reads with uncalled bases (-
181 c), quality filter at Q10 (-q), and rescue barcodes and RAD tags containing sequencing error
182 (-r). Cleaned RAD tags were aligned to the male guppy reference genome using BWA-MEM
183 (Li, 2013), and converted to bam format using samtools. Read group information was added
184 to bam files using Picard Tools v2.6.0 AddOrReplaceReadGroups (Broad Institute, 2019) and
185 alignments based on the same individual were merged using Picard Tools MergeSamFiles.
186 Bam files were used as input for the gstacks module in Stacks2, using only alignments with a
187 minimum mapping quality of 20. The final VCF contained only loci called across all
188 individuals (-p 1), at a max-missing frequency of 80% (-r 80) and minor allele frequency (-
189 maf) of 5%. Samples were retained with \geq 15X average coverage, average coverage across
190 the samples was 33.4X (Table S2) and average missing data was 0.05%. For QTL scans
191 genotypes were imputed based on grandparental phasing (see below).

192

193 ***Linkage mapping***

194 Linkage maps were produced with Lep-MAP3 (Rastas, 2017). Pedigrees were produced for
195 each cross by including dummy parents (one pair per cross) from which all F2s descended.

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196 Genotype likelihoods were called from the VCF input with the *ParentCall2* module, including
197 the *-halfSibs=1* flag. Further filtering was performed with the *Filtering2* module removing
198 markers with a MAF < 0.1 (within families) and with missing data in >10% of individuals
199 (within families). Markers were mapped to linkage groups (LG) with *SeparateChromosomes2*
200 modules based on a logarithm of the odds (LOD) score of 20, using all informative markers,
201 and grandparental phase information. LGs with fewer than 20 markers were discarded,
202 leaving 21 LGs. The two largest LGs were separated by further iterations of
203 *SeparateChromosomes2* run over these specific LGs with an elevated LOD limit of 30. This
204 produced 23 LGs in agreement with the guppy genome. Unmapped markers were joined to
205 the 23 LGs with the *JoinSingles2All* module, with an LOD limit of 5. The module was iterated
206 until no further markers could be mapped. In total, 7,256 markers of 16,539 were mapped
207 to LGs. The module *OrderMarkers2* was then run over each LG independently to order and
208 place markers within LGs. An initial 10 iterations were performed, with order determined by
209 maximum likelihood. For chromosome 12, male recombination was not permitted given
210 previous evidence that males do not recombine over the sex chromosome (Charlesworth,
211 Zhang, *et al.*, 2020). LOD scores from these maps were used to further filter markers on the
212 basis of support for multiple mapping within a LG (multiple LOD peaks) or if maximum LOD
213 was within one standard deviation of the mean. These markers were blacklisted for the final
214 *OrderMarkers2* run, in which the *evaluateOrder* flag was run over the earlier maximum-
215 likelihood based map. Final maps were sex-averaged and trimmed according to graphical
216 evaluation. Grandparental-phased genotypes were exported for QTL analysis. Effects of
217 female-biased heterochiasmy (Bergero *et al.*, 2019) on linkage maps are discussed in the
218 supplementary material.
219

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220 ***Heritability and multi-locus estimates of trait architecture***

221 We used Genome-wide Complex Trait Analysis (GCTA) (Yang, Lee, *et al.*, 2011) to estimate

222 phenotype heritability. This approach estimates the heritability of phenotypes by

223 partitioning phenotypic variance into genetic variance (specifically of the SNPs sequenced

224 rather than heritability in the traditional sense), random genetic effects and residual

225 variance using the restricted maximum likelihood (REML) method within a linear mixed

226 model. Empirical support suggests that heritability estimates using this method are

227 comparable to those estimated from true pedigree studies (Stanton-Geddes *et al.*, 2013;

228 Duntsch *et al.*, 2020). We first separated SNP data into males and females and, for each,

229 estimated a genetic relatedness matrix (GRM) using all SNPs. SNPs from scaffolds were

230 merged onto the beginning/end of chromosomes according to the linkage map (Table S3) to

231 improve the accuracy of per-chromosome estimates. To account for genetic variance among

232 the four families, we included the first three eigenvectors from PCA (the minimum number

233 required to separate four families) as quantitative covariates. Specifically, these analyses are

234 assessing within-family associations between genetic covariance and phenotypic variance,

235 and allowing for among-family intercepts to vary. Rearing covariates were included in a

236 trait-specific manner if these were associated with the phenotype (Table S4). To assess

237 within-family effects, we included an additional GRM calculated with the *--make-bK 0.05*

238 parameter. The addition of this GRM allows us to partition variance into that associated

239 with sequenced SNPs (G1) and within-family structure (G2). At the per-chromosome level,

240 including this additional GRM prevented model convergence in some cases (11/138

241 chromosome-phenotype pairs), but we observed negligible differences in heritability

242 estimated with and without this additional GRM at the whole-genome level.

243

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244 To estimate the heritability associated with specific chromosomes (h^2c), we took two
245 approaches: 1) We partitioned phenotypic variance using a model with a GRM derived from
246 the focal chromosome and quantitative covariates; 2) We used a likelihood-ratio test (LRT)
247 approach following (Santure *et al.*, 2013, 2015; Duntsch *et al.*, 2020) in which we compared
248 a full model *a* (GRM based on all chromosomes except focal chromosome + focal
249 chromosome GRM) to a reduced model *b* (without focal chromosome GRM). Quantitative
250 covariates were included in both models. Models were compared with a LRT according to
251 (1), with p-values taken from the chi-squared distribution with one degree of freedom.

252

253
$$LRT = -2(L_a - L_b) \quad (1)$$

254

255 Correlations can reveal polygenic architectures. Positive correlations suggest traits are
256 associated with many loci of small effect, assuming chromosome size is a proxy for
257 functional loci count. Previous work has suggested p-values derived from both chromosome
258 partitioning approaches highlighted above are comparable (Kemppainen and Husby, 2018a),
259 so we used h^2c estimations from focal chromosome GRMs (approach 1) as issues with
260 model convergence prevented estimations of h^2c on chromosomes that accounted for little
261 phenotypic variance under approach 2 (see Table S5). Regressions were performed using
262 the *HC_Correction()* function presented by Kemppainen and Husby (2018b), which corrects
263 for heteroscedasticity among chromosomes and the constraint of GCTA models that
264 prevents negative estimations of h^2c .

265

266 ***QTL scans***

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267 We produced two datasets for QTL-scanning. The first included fully-informative SNPs for
268 both founding populations, i.e. SNPs that were homozygous in all eight grandparents and
269 fixed for alternative variants between HP and LP-derived grandparents. This dataset
270 included 1,220 SNPs, and allowed for analysis of all individuals together to increase
271 biological power, where individuals could inherit an HP (H) or LP (L) allele. The second
272 dataset comprised four separate datasets, one for each family in which family-informative
273 SNPs were included for each family (homozygous, alternative, SNPs in each grandparent
274 within families). Numbers of family-informative SNPs for each family were similar (3,436;
275 3,400; 3,476; and 3,476 for QF2YHM2, QF4YHM8, YHF5QM6, and YHF6QM7 respectively).
276 These datasets provided weaker biological power, but increased resolution within families,
277 and also allowed us to examine alleles that may have only been captured and are
278 segregating in single crosses. It is important to note, these latter datasets cannot be used to
279 assess loci that are Y-linked. Because all males within a family carry the same Y, the effect of
280 different Y loci among families cannot be separated from general genome-wide relatedness
281 within and among families that captures many autosomal loci.

282

283 We first performed single locus scans using the *scan1()* function in R/qtl2 (Broman *et al.*,
284 2019). We inserted pseudomarkers using *insert_pseudomarkers()* with step=1, calculated
285 genotype probabilities with *calc_genoprob()* and an error_prob=0.002, and converted
286 genotype probabilities to allele probabilities using *genoprob_to_alleleprob()*. We calculated
287 a grid with *calc_grid()*, subsetted genotype probabilities to grid pseudomarkers with
288 *probs_to_grid()*, and used this to calculate a kinship matrix with *calc_kinship()* according to
289 the Leave-One-Chromosome-Out method (LOCO). Rearing covariates and a binary family
290 assignment matrix were included as additive covariates . Significance of LOD peaks was

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291 determined by 1000 permutations for all models with *operm()* at an α of 0.05. For scans of
292 the sex-determining region, inputs were merged for males and females, and the same
293 methodology was used, with the exceptions that sex was modelled as a binary phenotype,
294 and rearing covariates were excluded from the covariate matrix.

295

296 We also explored QTL scans that allow for multiple QTLs using the *stepwiseqtl()* function in
297 R/qtl (Broman *et al.*, 2003) which is not available in R/qtl2. This approach assesses
298 interactions among all pairs of loci, allowing for epistatic effects to be examined. We
299 allowed for models with a maximum of six loci, used the imputation method, and allowed
300 for only additive interactions among loci. Significance of LOD peaks was determined based
301 on 1000 permutations.

302

303 RESULTS

304 **Phenotypes**

305 Both male age (GLM: $F_{3,340} = 14.75, p = 4.82e^{-9}$) and size at maturity (GLM: $F_{3,340} = 16.02, p =$
306 $9.30e^{-10}$) of F2s varied significantly between the four cross families (Figure S1A; Table S4).
307 For age at maturity, cross YHF5QM6 F2s tended to mature later than all other crosses
308 (Tukey $p < 0.05$), and for size at maturity, cross QF4YHM8 F2s matured at a larger size. Male
309 age and size at maturity were not strongly associated with one another (correlation of
310 individuals, Spearman's $\rho = -0.07, p = 0.184$), but a GLM of male size at maturity by an
311 interaction between age at maturity and family revealed a significant effect (GLM: $F_{3,340} =$
312 $2.98, p = 0.031$). However, within this model relationships between male age and size at
313 maturity were both positive and negative depending on family. Rearing conditions affected
314 both male phenotypes. Males matured earlier under increased temperatures (GLM: $F_{1,340} =$

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315 13.75, $p = 2.40e^{-4}$) and if born later in the experiment (GLM: $F_{1,340} = 23.26, p = 2.15e^{-6}$).
316 Larger males at maturity tended to be born later in the experiment (GLM: $F_{1,340} = 41.51, p =$
317 $4.06e^{-10}$), but temperature did not affect size at maturity. Family status explained 10.4% and
318 11.7% of phenotypic variance for age and size at maturity respectively, which will not be
319 captured by our subsequent mapping approaches.

320

321 Female phenotypes were generally less variable among families, only female size at first
322 brood (GLM: $F_{1,265} = 11.88, p = 6.04e^{-4}$) and offspring weight (GLM: $F_{1,249} = 3.03, p = 0.030$)
323 differed significantly between families (Figure S1B; Table S4), with the latter effect only
324 marginally significant. Consistent with a general life history axis, covariance among female
325 phenotypes was generally high (Table S6). All female traits loaded positively onto PC1
326 (37.6%), with female age (loading = 0.68) and size (loading = 0.58) loading particularly
327 strongly. PC2 (27.0%) explained residual variance associated with brood traits, with first
328 brood size (-0.62) and interbrood (-0.59) loading negatively, and offspring weight loading
329 positively (0.44). PC2 therefore summarises variation among females with few, heavier
330 offspring and short interbrood periods, and vice versa. Similar to males, females reached
331 maturity and produced their first brood earlier under increased temperatures (GLM: $F_{1,265} =$
332 $21.65, p = 5.19e^{-5}$). Increased temperature also reduced interbrood period (GLM: $F_{1,265} =$
333 $24.32, p = 1.45e^{-6}$) and females born later in the experiment had longer interbrood periods
334 (GLM: $F_{1,265} = 13.47, p = 2.94e^{-4}$). Other female phenotypes were not associated with rearing
335 conditions (Table S4). In contrast to male phenotypes, family status explained much less
336 phenotypic variance: 6.7% for first brood size, 5% for size at first brood, and 2.2% for
337 offspring weight ('family' was dropped from other models due to low explanatory power).
338

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339 ***Linkage mapping***

340 The final linkage map consisted of 6,765 markers and a length of 1,673.8 cM. There was
341 overall good concordance between this genetic map and the recently updated reference
342 genome (Fraser *et al.*, 2020), with the additional placement of unplaced scaffolds on all
343 linkage groups. There were also minor structural rearrangements and inversions (Figure S2),
344 for which corroborative support could be found from previously published HiC data (Fraser
345 *et al.*, 2020). Typically, unplaced scaffolds were joined to either chromosome end (Table S3;
346 Figure S2).

347

348 ***Heritability and multi-locus estimates of trait architecture***

349 Heritability varied between traits, but in almost all cases (excluding first brood size) the
350 variance explained by within-family structure (V_{G2}) was greater than the variance explained
351 by the specific SNPs themselves (V_{G1}). This is expected given RAD-sequencing is designed to
352 capture SNPs in linkage with causal variants, rather than causal variants themselves.
353 Estimates of heritability were greatest for male size at maturity (43.4%), offspring weight
354 (33.4%), male age at maturity (31.3%), and interbrood period (30.2%). Estimates for the
355 remaining female life history traits were lower, not exceeding 9.7% (female size at first
356 brood), with standard errors that overlapped 0 (Table 1).

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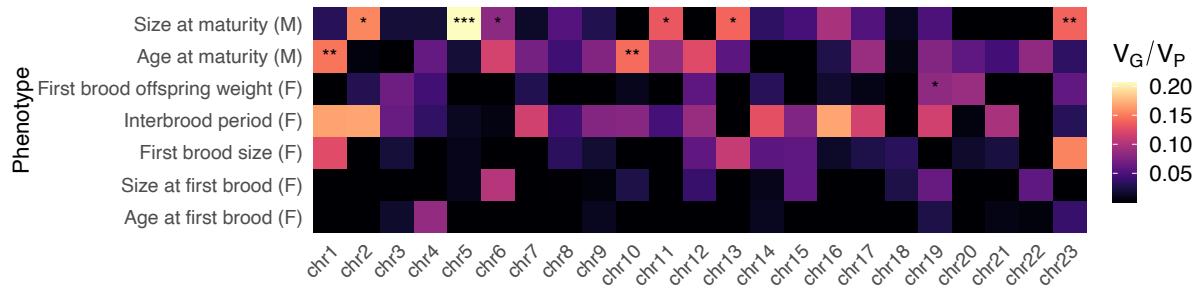
362 **Table 1:** Estimates of genome-wide heritability for each phenotype based on GCTA-GREML.
363 Phenotypic variance (V_p) is partitioned into variance explained by sequenced SNPs (V_{G1}),
364 genetic family structure (V_{G2}) and residual variance (V_e). Final estimates of phenotypic
365 variance partitions are given as proportions. All estimates include standard error.

Phenotype	V_{G1}	V_{G2}	V_e	V_p	V_{G1}/V_p	V_{G1+G2}/V_p
Age at first brood (F)	0 ± 0	0 ± 0	0.02 ± 0	0.02 ± 0	0.03 ± 0.19	0.033 ± 0.1
Size at first brood (F)	0 ± 0	0 ± 0	0.01 ± 0	0.01 ± 0	0 ± 0.2	0.097 ± 0.11
First brood size (F)	0.78 ± 1.96	0.12 ± 2.14	8.86 ± 1.09	9.76 ± 0.89	0.08 ± 0.2	0.092 ± 0.1
Interbrood period (F)	0 ± 0	0 ± 0	0.01 ± 0	0.02 ± 0	0.17 ± 0.27	0.302 ± 0.12
First brood offspring weight (F)	0 ± 0.02	0.03 ± 0.03	0.06 ± 0.01	0.09 ± 0.01	0 ± 0.28	0.334 ± 0.12
Age at maturity (M)	0 ± 0	0.004 ± 0	0.009 ± 0	0.013 ± 0	0 ± 0.2	0.313 ± 0.09
Size at maturity (M)	0 ± 0.07	0.156 ± 0.08	0.203 ± 0.03	0.358 ± 0.03	0 ± 0.18	0.434 ± 0.09

366
367 We repeated the analysis on each chromosome to test whether these estimates could be
368 explained disproportionately by certain chromosomes, or whether per-chromosome
369 associations may exist that cannot be observed within genome-wide estimates. Estimates of
370 $h^2 c$ based on single chromosome GRMs revealed six chromosomes significantly associated
371 with male size at maturity, four chromosomes with male age at maturity, and one
372 chromosome with offspring weight (FDR ≤ 0.05 ; Table S7; Figure 2A). Of these however,
373 according to the LRT approach only three chromosomes for male size at maturity (chr5:
374 20.7%, chr23: 13.9%, chr11: 13.3%), two for male age at maturity (chr1: 14.7%, chr10:
375 14.4%) and one for offspring weight (chr19: 8.4%) were significantly associated (LRT $p \leq$
376 0.05; Table S5). Following multiple-testing correction within phenotypes, only the
377 associations between chr5 and male size at maturity (LRT = 9.346, $fdr = 0.046$), and chr19
378 and offspring weight (LRT = 9.264, $fdr = 0.046$) were significantly associated according to
379 both methods. Agreement between both methods was good according to a correlation of p-
380 values (Spearman's $p = 0.827$, $p < 2.2e-16$). Whilst the correlation here is strong, there is a
381 clear downward biasing of p-values from single chromosome GRMs, evident as a shift away
382 from the $y=x$ relationship (Figure S3).

383

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384
385

386 **Figure 2:** Estimates of phenotypic variance proportions explained by per-
387 chromosome genetic relatedness matrices (h^2c). Tiles are coloured according to the
388 relative proportion of genetic variance (V_G) to phenotypic variance (V_P). FDR-
389 corrected p-values (corrected within phenotypes across all chromosomes) are
390 displayed as asterisks FDR $\leq 0.05 = *$; FDR $\leq 0.01 = **$; FDR $\leq 0.001 = ***$).

391
392

393 We found positive correlations associated with single chromosome GRM estimates of h^2c
394 and chromosome size (following the addition of scaffold sizes according to the linkage map;
395 Figure S4) for interbrood period ($r = 0.373, p = 0.04$, HC-corrected $p = 0.105$) and male size
396 at maturity ($r = 0.253, p = 0.122$, HC-corrected $p = 231$), however these were not significant
397 following HC-correction.

398

399 **QTL mapping**

400 We first mapped sex as a binary trait. The location of the sex-determining locus has been
401 narrowed down to a small region at the distal end of chromosome 12 (Fraser *et al.*, 2020;
402 Charlesworth, Bergero, *et al.*, 2020). Our QTL mapping recovered a single large peak on
403 chromosome 12 (17.79 cM), with confidence intervals extending from 5.35-27.78 cM (Figure
404 S5). In our map, the region following this (27.78-61.79 cM) corresponds to the very distal tip
405 of chromosome 12 (approximately 24.6 Mb onwards, plus additionally placed scaffolds),
406 which is the only fully recombining region of this chromosome and is pseudoautosomal
407 (Charlesworth, Zhang, *et al.*, 2020). This places the sex-determining region somewhere in

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408 the non-recombinating region immediately prior to the pseudoautosomal region (PAR), as
409 proposed by others (Fraser *et al.*, 2020; Charlesworth, Bergero, *et al.*, 2020). This analysis
410 therefore confirms good power to detect loci of large effect and confirms previously
411 published information about the sex chromosome and region containing the sex-
412 determining locus.

413

414 We then mapped female traits using all fully informative markers (N = 1220) across all 267
415 females. Applying a permuted 5% threshold (N=1000), we detected two QTL. The strongest
416 QTL was associated with first brood offspring weight at the very distal tip of chr19
417 (chr19:66.954) (Figure 3B), explained 7.46% of phenotypic variance, and exhibited additive
418 effects in which the HH homozygotes produced smaller offspring than LL homozygotes
419 (Figure 3C). Confidence intervals (drop in LOD of 1.5) extended between 55.335-67.877 cM.
420 The other QTL was associated with size at first brood (chr22:63.593) (Figure 3D), explained
421 5.94% of phenotypic variance, and exhibited additive effects in which the HH homozygotes
422 had their first brood at a larger size than LL homozygotes (Figure 3E); contrary to HP-LP
423 expectations. Confidence intervals for the chromosome 22 QTL extended between 44.235
424 cM - 68.753 cM.

425

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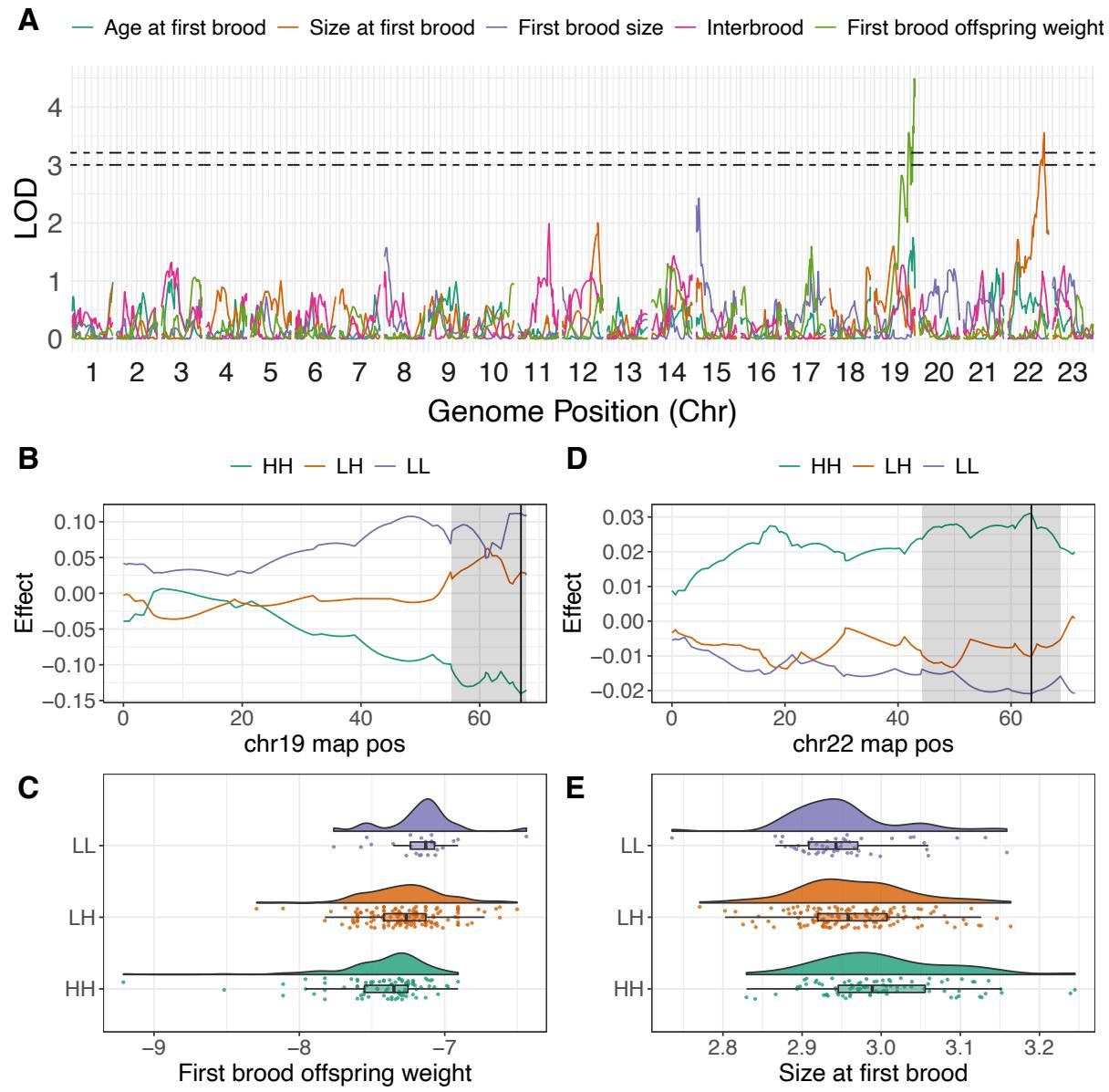


Figure 3: Single locus QTL scans for female life history traits: Age at first brood (days), size at first brood (cm), first brood size (residual), interbrood period (days) and first brood offspring weight (g). All female traits were log-transformed. Panel A shows genome-wide additive model scan results for all traits. 5% significance thresholds are denoted. Significant QTLs for offspring weight (chr19, B-C) and size at first brood (D-E) are visualised in further detail. Panels B-E show QTL effects across the focal linkage groups (B and D), and distributions of phenotypes across genotypes at the peak (C and E). In panels B and D, the QTL peak is shown as a black line, with confidence intervals (LOD drop = 1.5) highlighted by grey shaded areas.

Scans within each of the four families identified an additional three QTL (Figure S6-7). Two of these were observed in cross YHF5QM6, associated with first brood size (chr23:37.33,

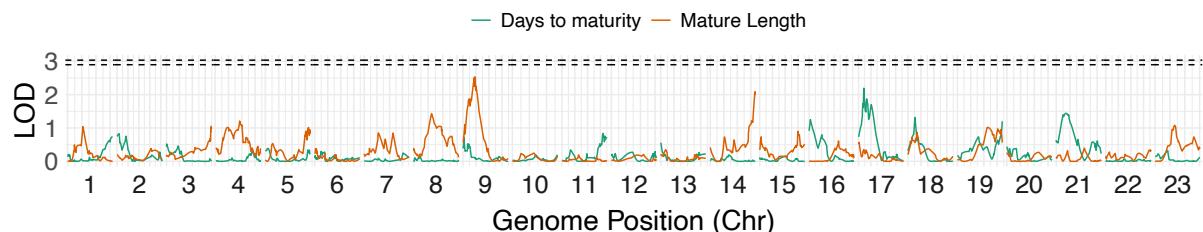
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442 LOD = 3.386, ci low = 4.589 cM, ci high = 53.588 cM) and interbrood period (chr12:60.713,
443 LOD = 3.406, ci low = 54.701 cM, ci high = 72.487 cM). These QTL explained 18.55% and
444 18.65% of phenotypic variance within their families respectively. The QTL linked with
445 interbrood period here is particularly interesting given its proximity to the sex-determining
446 region. We also detected a QTL associated with female interbrood period in cross QF4YHM8
447 (chr14:28.41, LOD = 3.559, ci low = 28.412 cM, ci high = 67.316 cM), explaining 17.9% of
448 phenotypic variance for this trait in this family.

449

450 We applied the same methodology to male traits, however we did not recover any
451 significant QTLs across the whole dataset (Figure 4). Within the YHF6QM7 cross, we
452 observed a significant QTL associated with male mature length on chromosome 23
453 (chr23:31.741, LOD = 3.462, ci low = 0 cM, ci high = 52.193 cM) (Figure S8-9). This
454 confidence interval covered the majority of the chromosome, and the QTL explained 9.48%
455 of phenotypic variance in this cross. At this QTL, HP homozygotes matured at a smaller size
456 than LP homozygotes.

457



458

459
460 **Figure 4:** Single locus QTL scans for male life history traits: Days to maturity (log-
461 transformed), length at maturity (mm). No significant QTLs were detected.
462
463

464 We also explored multi-QTL models using r/qtl's *stepwiseqtl()*, allowing for models that fit
465 up to six loci, with the same additive covariance matrices of family and rearing covariates

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466 used previously. For female models, this approach returned null models for all phenotypes
467 except size at first brood and offspring weight, for which we recovered single QTL models
468 including only the QTL loci previously identified above. For males, null models were
469 returned for both phenotypes.

470

471 ***Candidate genes***

472 Taken together, we find that where traits are heritable, they are associated with whole
473 chromosomes, suggestive of large polygenic regions, rather than single large effect QTL.
474 However, we observed two significant QTL for female traits (offspring weight chr19:66.954
475 and size at first brood chr22:63.593). We also observe several significant QTL within specific
476 families. Of these, the QTL associated with interbrood in cross YHF5QM6 (YHF5QM6-
477 chr12:60.71) is of particular interest given its proximity to the sex-determining region and
478 relatively narrow confidence intervals. We therefore explored these three regions further
479 for candidate genes. Full information on candidate genes can be found in Table S8.

480

481 The QTL at chr19:66.954, associated with offspring weight, covered ~8.2 Mb of chromosome
482 19, and included 267 genes. Due to the large size of this region, and that alternative
483 confidence intervals (Bayesian 5% probability intervals) suggested a single peak at
484 chr19:66.954, we limited our curation of candidate genes to the immediate 0.5 Mb either
485 side of the peak (chr19:18602889-19602889). This region included 40 genes. Several of
486 these genes (*wfikkn2b*, *tob1b*, *sap30bp*, *h3-3b*, *unk*, *mrpl38*, *fdxr*, *narf*, *cybc1*) are expressed
487 in all stages of embryonic development in zebrafish, or interact with growth signalling
488 pathways, suggesting potential functional effects for offspring weight. The closest gene to
489 the peak was *cdr2l*. Of the 40 genes in this region, 18 exhibited female-biased differential

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490 expression in gonads based on the guppy transcriptome (Sharma *et al.*, 2014), and six
491 exhibited male-biased expression (Table S8), suggesting potential reproductive function.

492

493 The chr22 QTL peak (for size at first brood) was on a previously unplaced scaffold
494 000111F_0:651336 and confidence intervals extended over the scaffold (00111F_0:528180-
495 1199617) and a region at the distal end of chromosome 22 (chr22:23415429-24223839).

496 The peak of the QTL did not overlap any genes, however the closest gene was F-box protein
497 33, *fbxo33*. In total, the region included 45 genes. There were no genes with GO terms or

498 KEGG annotations indicative of clear roles in growth. According to the GeneCards database
499 however, several genes had evidence of affecting size and growth phenotypes in mice

500 knockout studies (*fbxo33*, *tonsl*, *cnr1*, *cga*, *htr1b* and *myo6*). Two of the genes (*cga* and
501 *htr1b*) in this region are also associated with various hormonal pathways, including

502 gonadotropin hormone signalling that may affect development. Other genes in this region
503 are associated with myogenesis, including *myo6* and genes within the Akirin family (*akirin2*,

504 *gabrr1*, *pm20d2*, *cnr1*, *syncipi*). In addition, the gene *snx14* has been associated with
505 growth QTLs in grass carp (Huang *et al.*, 2020).

506

507 The interbrood period QTL on chr12 overlapped the YTH domain containing 1 gene, *ythdc1*,
508 on a previously unplaced scaffold 000149F_0 (000149F_0: 131600). This scaffold was placed

509 at the distal end of chromosome 12 near the sex-determining region, and corresponds with
510 scaffold KK215301.1 in the older female genome (Künstner *et al.*, 2016), which has similarly

511 been placed at the distal end of chromosome 12 in other mapping studies (Charlesworth,
512 Bergero, *et al.*, 2020). The confidence interval around this QTL covered additional regions of
513 scaffold 000149F_0 (000149F_0:47124-197258) and chromosome 12 (chr12:24525856-

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514 24705290). This region included 18 genes, but none were associated with clear GO or KEGG
515 terms indicative of roles for female fertility. Four genes exhibited female-biased expression
516 in gonads and seven were male-biased. The gene overlapping the QTL peak, *ythdc1*,
517 however is a promising candidate due to its interactions with N6-methyladenosine (m^6A)
518 (Wang *et al.*, 2015; Xia *et al.*, 2018). Disruption of m^6A by mutation of another modifier
519 *mettl3*, affected oocyte development and reduced the proportion of full-growth follicles in
520 zebrafish (Xia *et al.*, 2018). Further, *Ythdc1*-deficient mice have oocyte maturation blocked
521 at the primary follicle stage and experience alternative splicing defects in oocytes (Kasowitz
522 *et al.*, 2018). Examination of transcripts matching *ythdc1* (largest = CUFF_24477_m.316355)
523 in the guppy transcriptome (2014) revealed significant overexpression in ovaries compared
524 with testes. Sequence analysis alongside other Poeciliids (Ensembl release 101: *P. formosa*,
525 *P. latipinna*, *P. mexicana*, and *Xiphophorus maculeatus*) demonstrated significant purifying
526 selection on this gene ($dN/dS \leq 0.197$; Z-tests for purifying selection: $2.79 \leq Z \leq 7.40$; Table
527 S9). These provide strong evidence for a functional reproductive role for this gene in
528 guppies.

529

530 **DISCUSSION**

531 Using an F2 cross design of outbred populations with divergent life history phenotypes, we
532 have demonstrated both polygenic and oligogenic trait architectures underlying guppy life
533 history evolution. For both male size and age at maturity, we find significant heritability
534 associated with particular chromosomes, suggesting polygenic traits, but little evidence of a
535 genome-wide signal of polygenicity or single loci of large effects. For the five females traits,
536 we recovered significant genome-wide estimates of heritability associated with interbrood
537 period and offspring weight. For offspring weight, all per-chromosome estimates of

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538 heritability were generally weak, but we did identify a single locus of large effect at the
539 distal end of chromosome 19, suggesting an oligogenic architecture. For interbrood period,
540 we detected a weak genome-wide polygenic signal as an association between chromosome
541 size and per-chromosome heritability and also found evidence of a single locus of large
542 effect on the sex chromosome, LG12, in a single family. Together, these results suggest
543 interbrood period has an oligogenic genetic architecture consisting of a combination of
544 genome-wide polygenic loci and an individual locus of larger effect. We also detected a
545 significant single-locus QTL associated with female size at first brood with a small effect size,
546 but negligible estimates of genome-wide heritability for this trait. Finally, we found no
547 evidence for heritable genetic architectures for female first brood size and age at first
548 brood, although we observed a within-family QTL associated with first brood size on chr23.

549

550 The significant genetic components of guppy life history phenotypes found here are in line
551 with previous laboratory rearing estimates of heritability; specifically, the high heritability of
552 male traits, interbrood period, and offspring weight has been documented in laboratory-
553 reared populations under controlled conditions (Reznick, 1982) and in LP-introduction
554 experiments (Reznick *et al.*, 1997). However, there is mixed support for the heritability of
555 female life history traits. Consistent differences between laboratory-reared HP and LP
556 populations for female size and age at maturity and interbrood period have been observed
557 (Reznick, 1982), and similarly, Torres-Dowdall *et al.* (2012) report consistent differences in
558 female life history traits along a predation gradient in wild-caught vs lab-reared guppies
559 from the Guanapo river. However, estimates of heritability of female age and size at
560 maturity from experimental LP-introductions have shown inconsistent, often negligible,
561 estimates of heritability (Reznick *et al.*, 1997). Interestingly, this latter study postulates that

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562 the higher heritability, and more rapid evolution, of male life history traits may involve
563 significant loci associated with the Y chromosome, but we found no evidence here to
564 support this (although see discussion about Y-linked male traits below). Rather, our results
565 of a significant polygenic component of male age and size at maturity also predicts more
566 rapid phenotypic evolution for males, as observed in experimental introductions (Reznick *et*
567 *al.*, 1997).

568

569 Recent work has sought to compare the relative contributions of genetic and plastic effects
570 on guppy life history. HP-LP comparisons are confounded by increased competition for
571 available resources at high densities in LP sites (Reznick *et al.*, 2001), which can result in life
572 history traits that are resource dependent. Across multiple laboratory-reared guppy crosses,
573 Felmy *et al.* (2021), demonstrated that guppy life histories cluster together in terms of those
574 strongly affected by resource plasticity (predominantly size-related traits), those affected by
575 HP-LP ecotype (including interbrood interval and offspring weight), and those affected by
576 both or neither. For female traits, these results align well with ours, particularly the higher
577 within-family heritability for interbrood period and offspring weight (Table 1), and the
578 absence of strong signatures of genetic architectures associated with female size at
579 maturity. The phenotypic covariances for female traits observed here (Table S6) also agree
580 with the proposed “mosaic” of guppy life history traits (Felmy *et al.*, 2021). Felmy *et al.*
581 (2021) also demonstrate resource-based plasticity for male age and size at maturity,
582 however these likely operate alongside underlying genetics, in agreement with observations
583 here, and documented in other studies of male guppy life history (Reznick, 1982; Reznick *et*
584 *al.*, 1997, 2005).

585

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586 Guppy life history traits are also plastic with respect to other features of local environments.
587 Predator cues, for example, influence female size at maturity (Torres-Dowdall *et al.*, 2012)
588 and growth rate (Handelsman *et al.*, 2013), and resource availability affects female
589 reproductive investment (Reznick and Yang, 1993), the latter contributing comparable
590 phenotypic variance to that associated with heritability. We also found significant
591 associations between rearing conditions for both male age and size at maturity and female
592 age at first brood and interbrood period. These rearing effects reflected small fluctuations in
593 temperature (rearing temperature varied between 23.3-27.1 °C) and date of birth (used as a
594 proxy for other unmeasured rearing conditions), suggesting additional plasticity associated
595 with these phenotypes. Whilst we included rearing effects and family classification as
596 covariates in our models, and controlled for resources during rearing, it is quite possible
597 that additional sources of phenotypic plasticity may have obscured our analyses.

598
599 A limitation of our crossing design is that we cannot make across-family comparisons. We
600 controlled for family-specific intercepts in models, either by including kinship information or
601 family status as covariates. However, family status accounted for significant phenotypic
602 variance in five of our seven phenotypes (all except interbrood period and female age at
603 first brood; Table S4). These may be attributed to family-specific alleles segregating across
604 the genome, or Y-specifically, however we cannot separate these within the current dataset.
605 We identified some large effect loci segregating within families (Figure S6-9), however our
606 mapping of male phenotypes in particular will be blind to Y-specific QTL, given all males
607 within a family share the same Y allele. Y-linked loci have been suggested to affect age and
608 size at maturity phenotypes in other poeciliids (Kallman and Borkoski, 1978; Lampert *et al.*,

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609 2010), so there is reason to assume these regions may be important and comprise some of
610 the phenotypic variance associated with family status (which is an upper limit).

611

612 Whilst we detected both polygenic and single-locus architectures for life history
613 phenotypes, our study is limited by sample size. In part this is due to the modest brood sizes
614 of guppies, which restricted our ability to generate larger F2 families, and also because the
615 phenotypes considered are sex-specific. It is well known within quantitative genetics studies
616 that small sample sizes can inflate estimates of heritability and QTL effect sizes, a
617 phenomenon termed the “Beavis Effect” (Beavis, 1994; Rockman, 2012; Slate, 2013). In
618 particular, sample size has been demonstrated to inflate p-values when run with single
619 chromosome GRMs with GCTA (Kemppainen and Husby, 2018a), which explains why we
620 recover more modest p-values when comparing single chromosome GRM results to the LRT
621 approach. Our estimates of per-chromosome (h^2c) and genome-wide heritability should
622 therefore be treated with caution. More generally, this combined approach of using single
623 chromosome GRMs and LRTs may be a useful strategy to alleviate issues with modest
624 sample sizes associated with each approach individually. Specifically, this refers to the
625 inflation of significance tests with single chromosome GRMs and issues with model
626 convergence for chromosomes with minimal effects under the LRT approach.

627

628 The two main QTL observed here, detected across the whole dataset, reflect only marginal
629 PVE (7.46% for offspring weight on chr19, and 5.94% for female size at first brood on chr22)
630 which is likely inflated by our low sample size. Interestingly, each of these chromosome-
631 phenotype pairings was also detected (marginal significance) by our multi-locus approaches.
632 This suggests that these regions may be reasonably large, such that the peak (from which

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633 the PVE is calculated) only represents a portion of the variance explained by the wider
634 region. Neither of these regions have been strongly implicated in HP-LP adaptation before in
635 previous population genomic analyses (Fraser *et al.*, 2015; Whiting *et al.*, 2020), however
636 Whiting *et al.* (2020) recorded a selection scan outlier within the chr22 QTL interval
637 (chr22:23960000-23970000) in HP-LP comparisons from the Aripo and Madamas rivers. The
638 closest gene to this outlier is *sox11a*. While selection scans of HP-LP comparisons are unable
639 to determine which phenotype selection might be acting on, our results here suggest this
640 region may be involved in female growth.

641

642 An absence of prominent large effect loci, as described here for the majority of the traits,
643 would be expected to produce minimal molecular convergent evolution. This is predicted
644 due to redundancy in the mapping of genotype to phenotype, which may allow replicate
645 HP-LP pairs to use different sets of alleles to produce convergent HP-LP phenotypes (Barghi
646 *et al.*, 2020). Limited genomic convergence has been observed in two independent
647 evaluations of natural HP-LP populations (Fraser *et al.*, 2015; Whiting *et al.*, 2020), but
648 appears to be more pervasive in experimental translocations of HP guppies to previously
649 uncolonised LP habitats than naturally colonised LP populations (Fraser *et al.*, 2015). Part of
650 this discrepancy can be explained by the concept of “adaptive architecture”, such that the
651 convergent genetic basis of polygenic traits can be influenced by additional factors such as
652 starting allele frequencies. Starting allele frequencies or amounts of standing variation are
653 likely to be more similar when experimental populations are founded from the same
654 population and/or lack founding bottlenecks compared with naturally-derived HP-LP pairs in
655 different rivers. Empirical evidence for genetic convergence occurring with polygenic traits
656 has been observed for male mating song traits in Hawaiian *Laupala* crickets (Blankers *et al.*,

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657 2019) and myxomatosis resistance in rabbits (Alves *et al.*, 2019), suggesting that genetic
658 architecture alone is not necessarily a constraint on genetic convergence.

659

660 Similarly, polygenic traits are predicted to facilitate rapid adaptation. Several experimental
661 studies involving the translocation of guppies into upstream LP habitats (Reznick and Bryga,
662 1987; Reznick *et al.*, 1997, 2019; Gordon *et al.*, 2009) or manipulating predation within
663 populations (Reznick *et al.*, 1990) have demonstrated that HP-LP adaptive traits evolve
664 rapidly over the course of a few generations. Our findings here, that some of these traits
665 exhibit genetic architectures of many loci of small effect, are in keeping with recent
666 empirical (Barghi *et al.*, 2019) and theoretical work (Bell, 2013; Jain and Stephan, 2017a)
667 suggesting these facilitate rapid adaptation. In this framework, many loci of small effect
668 provide adaptive substrate within populations to rapidly respond to shifting optima. This is
669 particularly true if distance to new fitness optima is short, such that environmental change
670 is modest and fitness effects are relative and/or non-lethal. This may be the case for guppy
671 life history traits under LP regimes, where soft selection is most likely. This model therefore
672 could allow male life history traits to change rapidly through small changes at many loci,
673 whilst additional segregating larger effect loci may act in concert with compensatory
674 changes at small effect loci for female traits. Our results here provide regions of the genome
675 and candidate genes to explore further. For instance, an appreciation that much of the
676 genetic basis of guppy life history traits may be polygenic informs on experimental and
677 sampling designs for future population genetic studies of this system. In particular, temporal
678 sampling and quantifying genome-wide autocovariances of neutral allele frequencies offers
679 a promising avenue for studying the role of polygenic architectures in rapid adaptation

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680 (Buffalo and Coop, 2019). The genomic regions identified here may serve as focal regions in
681 these studies.

682

683 In conclusion, we used an F2 cross to explore the genetic architecture of seven guppy life
684 history traits that are known to evolve rapidly and convergently in natural populations. We
685 find evidence of only two loci of large effect associated with female size at first brood and
686 offspring weight, and evidence of many loci of small effect associated with male age and
687 size at maturity, interbrood period and brood size. In addition, we observed several within-
688 family loci of large effect, suggesting segregating variation within source populations. These
689 results have important implications for improving our understanding of how life history
690 traits evolve in the guppy model, and more broadly, provide empirical evidence for
691 predictions of the genetic architecture of rapidly adapting and convergent phenotypes.

692

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707

708 **DATA AVAILABILITY**

709 All sequencing read data is available from the ENA (DOI: XXX)
710 All scripts and other data associated with analysis will be made available in an archived
711 github repository (Zenodo, DOI: XXX)

712

713 **CONFLICTS OF INTEREST**

714 The authors declare no conflicts of interest.

715

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