Genetic growth potential, rather than phenotypic size, predicts migration phenotype in Atlantic salmon.

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Abstract

Knowledge of the relative importance of genetic vs. environmental determinants of major developmental transitions is pertinent to understanding phenotypic evolution. In salmonid fishes, a major developmental transition enables a risky seaward migration that provides access to feed resources. In Atlantic salmon, initiation of the migrant phenotype, and thus age of migrants, is presumably controlled via thresholds of a quantitative liability, approximated by body size expressed long before the migration. However, how well size approximates liability, both genetically and environmentally, remains uncertain. We studied 32 Atlantic salmon families in two temperatures and feeding regimes (fully fed, temporarily restricted) to completion of migration status at age 1 year. We detected a lower migrant probability in the cold (0.42) than the warm environment (0.76), but no effects of male maturation status or feed restriction. In contrast, body length in late summer predicted migrant probability and its control reduced migrant probability heritability by 50-70%. Furthermore, migrant probability and length showed high heritabilities and between-environment genetic correlations, and were phenotypically highly correlated with stronger genetic than environmental contributions. Altogether, quantitative estimates for the genetic and environmental effects predicting the migrant phenotype indicate, for a given temperature, a larger importance of genetic than environmental size effects.

1. Introduction

Genetic vs. environmental determinants of developmental transitions between life stages are a major topic in ecological and evolutionary studies [1-3]. Generally, both the environment and genes are assumed to underlie the expression of plastic developmental phenotypes [3], but the relative contribution of each often remains unknown [4, 5]. However, knowledge of environmental vs. genetic contributions underlying life-stage transitions is pivotal to making accurate ecological or evolutionary predictions under environmental change, such as global warming [5, 6]. A major life-stage transition in many species is associated with feeding or reproduction migrations, whereby the migrant phenotype, as opposed to the resident phenotype [defined as individuals not migrating during a particular season; 7], may express in only part of the population, or vary with age [8, 9]. Even though such partial migration, or variation of migrant age, is assumed to have considerable ecological and evolutionary consequences [8-10], evidence for major underlying genetic effects is scarce or controversial [11-13]. Nonetheless, selection and crossing experiments in birds and fishes indicate a heritable genetic basis [8, 14], but for fishes, underlying mechanisms remain elusive [reviewed in 14]. To disentangle genetic from environmental components of binary traits, including migration phenotypes, the threshold model may be appropriate [10, 15]. The threshold model assumes that the expression of categorical phenotypes underlies a (usually unobserved) continuous liability [16-18]. Only if the liability, influenced by both environmental and genetic effects, exceeds one (or several) threshold(s) during a sensitive period, the developmental transition towards the alternative phenotype(s) is initiated.

In many fish species also the age of the developmental transition towards the migratory phenotype shows considerable variation. In salmonid species, such as salmon, trout, charr, and whitefish, the age when the freshwater-hatched fish migrate to the sea (or larger water bodies, such as lakes) varies considerably, encompassing one to eight years in Atlantic salmon [reviewed by 19]. Importantly, within a population older migrants are usually larger than younger migrants, whereby body size increases salinity tolerance and predator avoidance ability [4, 19, 20]. As a result of the latter, size at seaward migration, both within and across migrant ages, often covaries with migration survival [4, 21-23].

Whether an Atlantic salmon of a given age undergoes the required developmental transition to become a migrant (“smolt”) in the following spring has long been suggested to be determined by body length in the previous summer or autumn [reviewed in 19]. Therefore, and because the liability remains unknown, body length is often used as an *a priori* liability proxy, and many previous indirect estimates for genetic migration phenotype variation stem from investigations on such proxies [reviewed in 24]. However, the liability proxy-trait variation as relevant to initiating the developmental transition is expressed well in advance of the actual migration [25], as is suspected for many other developmental transitions [26]. Such time lag poses logistical challenges not only for organisms after they have adopted a developmental

transition, but also for researchers studying a dynamic liability proxy. It is thus unsurprising that - despite considerable knowledge on the physiological changes and underlying mechanisms *during* the transition [27] - exact mechanism *initiating* the transition from the resident towards the migrant phenotype remain unknown.

Even though body size may be a useful proxy determining whether the migrant phenotype is initiated, it may provide an inappropriate liability proxy in many cases. Specifically, initiation of the migrant phenotype triggers additional growth differences between prospective migrants and residents (which may migrate at older age in Atlantic salmon) that increase their size differences, thereby blurring cause and effect. During the transition, prospective migrants express accelerated growth relative to residents, which results in size bimodality and culminates in migrants being larger than residents at the time of migration, both among and within families [25, 28-32]. Similar confounding between causes for and effects of the transition towards the migrant phenotype apply to inferences about body condition. Probably owing to these methodological challenges, past inferences about effects on migration phenotypes were often based on size records from any time between presumed growth acceleration and the time of migration [reviewed in 24], and thus statistical associations between size and migrant probability are inconsistent across studies .

Here, we overcome a number of the abovementioned limitations and use Atlantic salmon as a model to investigate genetic and environmental determinants for the probability to exhibit the migrant phenotype in the second spring (hereafter called migrant probability; MIG), focussing on body size at the end of the first summer as a liability proxy. Specifically, we combined longitudinal common-garden experimentation in two water temperatures and two feeding regimes with quantitative genetic methodology. We followed development of 663 individuals from 32 pedigreed Atlantic salmon half-sib families until their second spring, when we assessed migration phenotypes (**figure 1**). We used uni- and bivariate models to test for environmental effects of water temperature, feeding regime, body condition, and male maturation on MIG. Further, we tested within and between two environments with a 2° C seasonal temperature difference, whether body length covaried with MIG and partitioned this covariance into environmental and genetic components. Our results support a long-suspected strong genetic joint-governance of body size in late summer with migration phenotype in spring, which we demonstrate to be extremely stable between two temperature environments. These results widen our understanding of genetic and environmental importance in developmental transitions, as well as their thermal stability, thereby providing quantitative primers for future modelling relying on environmental vs. genetic relationships among life-history traits across environmental temperatures [e.g., 33].

2. Material and methods

(a) Experimental population and settings, and data collection points and routines

In October 2017, we created 32 half-sib families (generation 2) by crossing 32 parents (generation 1) in eight 2x2 factorials of unrelated individuals (**figure 1** ***a***). The parents originated from a broodstock (generation 0) that was hatchery raised, maintained by the Natural Resources Institute Finland, Taivalkoski, Finland, but whose individuals successfully completed a sea migration [Oulu River; described in 34]. We created and reared the experimental cohort commonly in a newly established laboratory with two similar water recirculation systems at the University of Helsinki, Finland, controlled for water temperature, oxygen, dissolved nitrate components, and light. We incubated eggs and larvae in vertical incubators with two replicates per family and temperature environment. At first feeding, we pooled same-temperature family replicates and randomised an equal number of individuals from each family (3-7) to each of eight similar 250 L tank replicates per temperature environment (i.e., all families in all tanks). The fish were part of other studies, of which one required lethal sampling [35], reducing fish numbers continuously. After sampling and natural mortality, 663 individuals yielded data for migration status in May 2019 (393 in cold and 270 in the warm environment, respectively). Water temperature in egg incubators and tanks was controlled to follow a seasonal cycle, approximating a 2 °C difference referred to hereafter as “warm” and “cold” temperature treatments (warm, range: 6.3-17.7 °C; cold, range: 4.1-16.0 °C; **figure 1** ***b***). Tank illumination, using fluorescence lights (4000 K, 35 W; 500 Lux at the surface) and controlled by a digital astronomical time switch (without dimming), followed the natural cycle at 61.054° (latitude) and 25.042° (longitude) (**figure 1** ***c***).

Fish were fed a commercial salmon diet *ad libitum* with particle sizes meeting fish-size compositions at all times (start dates: **figure 1** ***b***). In August 2018 (warm) and September 2018 (cold) we anesthetized individuals (using methanesulfonate), inserted passive integrated transponder tags (into the body cavity; 12 mm) to enable re-identification, and fin clipped individuals to allow for genotyping. After fasting fish for 24 h, we measured fork length (± 1 mm) between August and September 2018 - around the time of daylengths suspected to characterise the sensitive period when migration status initiation is determined [**figure 1 c**, 15-12 h; 30]. These measurements were followed by a feed-restriction-treatment period (either *ad libitum* feeding for seven days per week, or *ad libitum* feeding for two days per week with no feeding for two or three days between feedings) that was crossed with the temperature treatment for a five-week period per tank (September - October 2018; **figure 1** ***b, c***). We again fasted and re-measured fish after the feed-restriction treatment in October. We determined maturation status in December 2018 (during the natural spawning period), when we categorised males as mature when observing milt during gently pressing the abdomen (females rarely mature at this age). We determined migration status in May 2019 [during the population-specifc period of highest migration success; 34] (**figure 1** ***b***), when we

categorised individuals as first-year migrants when showing darkened fin edges, lack of colour patches ("parr marks") and enhanced body silvering (**figure 1** ***d***). We categorized individuals as residents when not displaying signs of the migrant phenotype at this time. It should be noted that many Atlantic salmon remaining resident at 1 year of age may become migrants in later years [4], and therefore the resident category also includes potential later year (older) migrants. However, it is also possible that residents, especially males, can reproduce without migrating [4, 36]. Animal experimentation followed European Union Directive 2010/63/EU under license ESAVI-2778-2018 by the Animal Experiment Board in Finland (ELLA).

(b) Pedigree construction

We determined genotypes of parents and experimental individuals using a multiplex-PCR for 177 single nucleotide polymorphisms (SNPs) of an established panel [37], and by sequencing using an Ion Torrent (984 broodstock individuals from which we drew parental individuals) or Illumina platform (MiSeq or Next-Seq) (parental individuals, experimental individuals). Using unlinked, polymorphic SNPs, we reconstructed grandparents of the experimental individuals (with 131 usable SNPs) under maximum likelihood [38], which we combined with knowledge about the crossing scheme used to create the parental generation, and assigned the experimental individuals to their 32 parents (with 141 usable SNPs) with a likelihood approach [39], resulting in a three-generation pedigree (**figure 1** ***a***). On this pedigree, we based the inverse relationship matrix (A^{-1}) utilized to infer additive genetic variance via animal-model analyses [40] described below.

(c) Statistical analyses

To test model terms and estimate means and (co)variance components for MIG and LEN, we used uni- and bivariate generalized animal models with probit-link function for MIG, corresponding to animal threshold models [41]. The models were fitted with the R package MCMCglmm [42] using Bayesian Markov chain Monte Carlo simulations, which appear an appropriate method for animal models on categorical data [41, 43]. Initially, we fitted a univariate linear model:

$$y = \mu + Feed + Temp + Feed{:}Temp + Mat + Feed{:}Mat + Dam + Animal + Tank + Residual,$$

where y is the liability for migrant probability, μ a model constant, Feed the fixed feed restriction environment effect (full, restricted), Temp the fixed temperature restriction environment effect (cold, warm), Mat the fixed male maturation effect (0, 1), Feed:Mat the fixed interaction effect of the feed restriction with the maturation effect (restricted to the warm environment), Dam the random dam effects (n = 32), Animal the random additive genetic effects (736 entries in A^{-1}), Tank the random tank effects (n = 16), Residual the random residual effects (n = 663). We also extended a reduced version of the univariate model (see results) by including body length in late summer as a continuous covariate ("length", a

predictor; log-transformed, mean centred, and variance scaled), which we interacted with the temperature treatment. We also extended the reduced univariate models to test whether MIG differed among genotypes of a locus (*vgll3*) that has been shown to have a strong effect on male maturation probability in this experimental population [35], but we did not detect *vgll3* effects, either when controlling or not controlling for phenotypic length in late summer (electronic supplementary material, **table S1**). We estimated all variances conditional on temperature environments with 2x2 covariance matrices between environments for dam (D) and animal (G) effects and diagonal covariance matrices for tank (C) and residual (R) effects. We fitted bivariate models by extending the reduced model (without the length covariate) with body length in late summer as a second response ("LEN", "length" as a response). For the bivariate model, we additionally allowed for between-trait covariances.

We ran univariate and bivariate models for 1,500,000 and 6,000,000 iterations, respectively, and sampled every 100 iterations. For each model, we ran four chains and determined i) whether the MCMC sampling had converged as indicated by a scale reduction factor around 1 per chain [44], ii) the required number of samples to discard ("burnin") until consistently reaching a scale reduction factor < 1.1 across chains [44], iii) required thinning to have autocorrelations at lag 2 < 0.1 per chain, and confirmed whether MCMC resulted in sufficient mixing using visual examination of trace plots per chain. These criteria resulted in subsequently combined posteriors across chains with sample sizes between 8,000 and 22,000. We conducted a prior sensitivity analysis as reported in the electronic supplementary material (**figures S2-S4**). We tested feed-restriction-treatment effects on growth rate by a general animal model and predicted the sexual maturation rate using a generalised animal model (electronic supplementary material, **table S2, figure S1** and **table S3**, respectively)

(d) Derived parameters

We calculated heritability as the proportion of additive genetic variance (V_A) to the total phenotypic variance (V_P). Under the threshold-model interpretation of generalized model estimates that include common environmental variance (V_C), residual variance fixed to 1 (V_R), and a variance of 1 for the probit link function [45], this results for MIG in:

$$h^2 = \frac{V_A}{V_P} = \frac{V_A}{V_A + V_C + V_R + 1}.$$

We estimated correlations at the phenotypic (R_P), residual environmental (R_E), and genetic levels (R_G) as in [46], but additionally accounted for common environmental effects on R_P. To translate liability estimates to the proportional scale, we used either the "predict.MCMCglmm" function of the MCMCglmm R-package or (for heritabilities) the "QGmvparams" function of the QGglmm R-package [45]. We present mean estimates with credible intervals (95% highest posterior density estimates of model posteriors).

3. Results

(a) Migrant probability is not affected by a temporary feed restriction, male maturation, or maternal effects

Using a univariate generalized animal model and not controlling for phenotypic body length, we detected that MIG was higher in the 2 °C warmer environment (**table 1**). However, although the feed restriction reduced the specific growth rate by about 50% (electronic supplementary material, **table S2, figure S1),** MIG was not affected by this restriction (**table 1**). MIG was also not affected by male sexual maturation, which occurred only in males in the warm environment at a rate of 0.19 (95% CI: 0.08-0.33; electronic supplementary material, **table S3**), nor by any interaction term (**table 1**). We also did not detect any maternal (dam) or common environmental (tank) effect variance on MIG (**table 1**). We therefore removed dam, but not tank effects, from models because the latter constitute the experimental replicates for the temperature environments.

(b) Migrant probability, but not its heritability, differs between temperature environments

After removing model effects not different from zero, we estimated an average MIG of 0.41 (0.27-0.55) in the cold and 0.76 (0.63-0.87) in the warm environment (probit scale contrast: 1.68, 0.95-2.36). We also estimated relatively high heritabilities (h^2) for MIG liability (h^2_{Cold} = 0.53, 0.33-0.72; h^2_{Warm} = 0.43, 0.22-0.64), that were not different between temperature environments (thereby rejecting evidence for one component of genotype-by-environment interactions; $h^2_{Cold} - h^2_{Warm}$ = 0.10, -0.17-0.36). We further estimated a high between-environment genetic correlation ($R_{G_{Cold,Warm}}$) with the upper 95% credible interval being very close to unity ($R_{G_{Cold,Warm}}$ = 0.84, 0.62-0.99), indicating that genotype re-ranking for MIG was negligible between the temperature environments (thereby rejected another aspect of genotype-by-environment interactions). Thus, despite strong temperature effects on average MIG, environmental temperature appeared to have negligible effects on the relationship between genotype and phenotype.

(c) Migrant probability and body size in late summer and show high correlations

To test whether phenotypic body length in late summer contributes to MIG liability, we expanded the reduced univariate model by adding phenotypic body length as a temperature-specific continuous predictor ("length"). We expected that if length contributes to (or even constitutes) the liability, it would positively covary with MIG. Furthermore, we expected that if genetic effects contribute positively to this covariance, the explicit modelling of this covariance as slope would reduce MIG heritability. Length was indeed a strong phenotypic predictor of MIG (**figure 2**). Controlled for length, temperature effects on MIG became dependent on the size at which the contrast was made because the slope between MIG and length was estimated as steeper in the warm than the cold environment (liability scale contrast: -1.18, -

231 1.85--0.49; **figure 2)**. Estimated at the overall geometric mean length, MIG was higher in the cold than the
232 warm environment (liability scale contrast: -1.18, -1.8--0.47).

233 Controlling for length also reduced heritability similarly in both environments (i.e., the heritability
234 difference between environments was similar regardless of whether length was controlled for; **figure 3**).
235 Heritability became non-significant in the warm but not the cold environment (h^2_{Cold} = 0.26 0.11-0.42;
236 h^2_{Warm} = 0.12, 0.00-0.28; **figure 3 *a*, *b***), and controlling for length also reduced the between-environment
237 genetic correlation (**figure 3 *c***). Thus, phenotypic length in late summer explained a considerable share of
238 MIG heritability (a phenotype expressed eight months later) in both temperature environments (52 and
239 71%, respectively).

240 Having established that MIG covaried with phenotypic length, we quantified the phenotypic covariance
241 and the relative contributions of environmental and genetic effects by fitting a bivariate model for MIG
242 and body length in late summer as a second modelled response (LEN). Bivariate model estimates for
243 average MIG in each temperature met those by the univariate model not controlling for phenotypic length
244 (MIG_{Cold} = 0.42, 0.28-0.57; MIG_{Warm} = 0.76, 0.63-0.88). Estimates for average LEN in each temperature
245 indicated strong temperature effects, whereby average LEN was 1.3 (1.1-1.5) phenotypic standard
246 deviations (equating to 25%, 20-30%) larger in the warm than the cold environment. Back-transformed
247 average LEN was 8.2 cm (7.8-8.7) in the warm and 6.6 cm (6.3-6.9) in the cold environment. For LEN, we
248 estimated, unlikely for MIG, common environmental (tank) effects in the cold, but not the warm,
249 environment that accounted for 9 % of the phenotypic variance (c^2; electronic supplementary material,
250 **figure S2)**.

251 Bivariate model estimates for MIG heritability were somewhat higher than by the univariate model (MIG;
252 h^2_{Cold} = 0.60, 0.38-0.78, h^2_{Warm} = 0.48, 0.26-0.69; **figure 3**; electronic supplementary material, **figure S2**)
253 and we estimated also high LEN heritabilities, which was surprisingly similar to the MIG heritabilities (LEN;
254 h^2_{Cold}= 0.57, 0.38-0.74; h^2_{Warm} = 0.52, 0.30-0.73; **figure 3 *a*, *b***). Using alternative prior specifications for the
255 variances of MIG, heritabilities for both traits were estimated somewhat lower but their difference
256 remained similar (**figure S3**). The between-temperature genetic correlations ($R_{G_{Cold,Warm}}$) were also high
257 and very similar for both MIG and LEN and their credible intervals were very close to unity (all $R_{G_{Cold,Warm}}$ =
258 0.92; **figure 3 *a*, *b***). These remarkably similar genetic correlations estimates differed only marginally with
259 alternative prior specifications for the genetic covariances (electronic supplementary material, **figure S4**).
260 Translated to the probability scale, we found MIG heritabilities to be lower and less similar between
261 environments than on the liability scale (MIG; h^2_{Cold} = 0.37, 0.23-0.49; h^2_{Warm} = 0.26, 0.13-0.38), but the
262 95% credible interval of the contrast on the probability scale also encompassed zero (MIG; $h^2_{Cold} - h^2_{Warm}$

= 0.12, -0.06-0.28). Thus, heritability and between-temperature genetic correlation estimates were similar for MIG (on the liability scale) and the liability proxy trait LEN.

As expected from univariate modelling, we detected high positive phenotypic correlations (R_P) between MIG and LEN in both environments ($R_{P_{Cold}}$ = 0.77, 0.69-0.85; $R_{P_{Warm}}$ = 0.73, 0.62-0.83; **figure 3 *a*, *b***). The phenotypic correlations within temperature environments underlaid both genetic and environmental correlations, whereby the former exceeded the latter; the between-trait genetic correlation (R_G) was with 0.92 consistently high in both environments and thereby equal to the between-temperature genetic correlation estimates for each trait. Between-trait environmental correlation estimates (R_E), for both temperature environments were somewhat lower ($R_{E_{Cold}}$ = 0.60, 0.39-0.79; $R_{E_{Warm}}$ = 0.57, 0.31-0.79; **figure 3 *a*, *b***), and even lower when using alternative prior specifications for the residual covariances (electronic supplementary material, **figure S4**). Relative contributions (weights) to the phenotypic correlation were either stronger or equal for genetic than environmental effects, whereby weights equate to the relative importance of the genetic and non-genetic components, respectively [46]. Specifically, the weights for genetic effects were 0.58 in the cold and 0.50 in the warm environment. As detected for MIG in the univariate model, no difference between temperatures was detected for variance parameters of either MIG or LEN (**figure 3**). Thus, correlation estimates between MIG and LEN were higher at the genetic than environmental level and both traits showed similar heritability and high correlation estimates across environmental temperatures, even though average expression of each trait differed considerably between temperature environments.

4. Discussion

The basis for variation in the developmental switches that result in categorical phenotypes remains a major research topic in several fields, including biology and medicine. The control of such categorical phenotypic variation may occur via (one or many) thresholds for the quantitative expression of a, usually unknown, liability that underlies environmental and genetic effects [16-18]. In this study, we investigated the developmental switch that determines whether Atlantic salmon express the migration phenotype associated with either a sea migration at one year of age (migrant) or with remaining in freshwater for longer (resident), and tested for association with its presumed liability proxy, body size in late summer. We estimated relatively high heritabilities for both migrant probability liability and its liability proxy trait, and high environmental, genetic, and phenotypic correlations between the two traits in two seasonal temperature environments. Furthermore, we estimated high between-environment genetic correlations for each trait. We, however, failed to detect male maturation, maternal, and feed restriction effects on migration phenotypes. Altogether, the results widen our understanding of genetic and environmental importance in the developmental transition towards age-specific migration phenotypes, as well as their

296 strong thermal stability, and have implications for ecological and evolutionary subjects and future studies,
297 which we elaborate on below.

298 **(a) Absence of male maturation and maternal effects on migration probability**

299 Our results strengthen previous findings, under both wild and culture conditions, that sexual maturation
300 of males in autumn is not inhibiting the migrant phenotype expression the following spring [36, 47-49].
301 Thus, the view that maturation and smoltification are conflicting processes [50], and which is also under
302 debate for the congeneric brown trout [15], may not hold generally in Atlantic salmon. We also rejected
303 the hypothesis that maternal effects affect migration phenotypes [such as suggested in 15], agreeing with
304 previous results in rainbow trout [51].

305 **(b) The relationship between migrant probability and body length**

306 Our results on Atlantic salmon strongly support a long-suspected, but often questioned, joint-governance
307 of body size in late summer and migration phenotype expression the following spring [9, 24]. We
308 demonstrate that, for a given temperature environment, this joint governance is stronger for genetic than
309 environmental effects, and that the genetic effects were extremely stable between two temperature
310 environments (**figure 3**). The results, thereby, provide quantitative primers for future modelling relying
311 on environmental vs. genetic relationships among life-history traits across environmental temperatures
312 [e.g., 4, 26, 33]. Given that the tested temperature difference of 2°C aligns with global warming scenarios
313 [52], modelling may encompass evolution under current global warming. Specifically, the combined high
314 between-trait and between-environment genetic correlation estimates suggest strong co-evolution of
315 migration phenotypes and body size across temperature environments, which may, depending on
316 ecological settings [9], be an advantage or a constraint.

317 We estimated high and similar heritability estimates for migrant probability and body size and high genetic
318 correlations, but only moderate environmental (residual) correlations (**figure 3**). Thus, the association
319 between traits appeared stronger for the genetic than the realised phenotypic values, which suggests that
320 genetic growth potential is also a proxy for migrant probability and not only size during a sensitive period.
321 An explanation could be that unknown genetically determined key components, such as growth hormone
322 levels or its receptor densities, may commonly control body size and migrant probability but need not
323 correlate tightly with phenotypic size during the sensitive period. Identifying such components may
324 explain how a scaling of the *genetic* growth potential occurs relative to actual body size, which varies
325 *environmentally* (such as by our temperature environments). Answers to this question may require more
326 detailed genetic mapping and in-depth investigations on the actual biological mechanism involved.

327 Given the time lag between the presumed sensitive period (in late summer) and expression of the
328 migration phenotype (the following spring), there may be time to catch up on missed growth

329 opportunities after the sensitive period. Thus, if we assume that a liability threshold ensures viable
330 migrant sizes, phenotypic size in late summer may be a poorer predictor of migrant size in spring than
331 genetic growth potential. In support of this, individuals initially falling within the lower group of a bimodal
332 distribution in late summer or autumn, assumed to represent prospective residents, may still grow rapidly
333 during winter and become migrants the following spring, both in captivity and in the wild [47, 53, 54]. As
334 an alternative, the sensitive period may last longer than suspected. In that case, individuals with a high
335 genetic potential for, but low realised, growth until late summer may still realise their genetic potential
336 thereafter, thereby reaching a *phenotypic* size threshold later and initiate the transition later. Given that
337 a growth spurt occurs in prospective migrants between late summer and winter as a consequence of the
338 developmental transition [25, 55], it may be difficult to differentiate between "normal" and "spurt"
339 growth to test between competing hypotheses. Nonetheless, the lack of effects on migration phenotypes
340 by the feed restriction, applied after the presumed period, argues against the presence of an extended
341 sensitive period (see below). Regardless of the sensitive period duration, our results suggest that
342 individuals with relatively small body size in late summer may still become migrants if they possess a high
343 genetic growth potential.

344 The presence of a higher genetic than environmental correlation between migrant probability and body
345 size (**figure 3**) has several implications. For example, investigations on the physiological mechanisms of
346 the developmental transition rely on body-size based migration-phenotype prediction for lethally
347 sampling prospective migrants and residents prior to expression of the migration phenotype. Our results
348 suggest that body-size based phenotype prediction may lead to conservative results because small
349 migrants may get incorrectly assigned resident status. Such wrong assignments may blur differences of
350 studied variables between true prospective migrants and residents. Another implication pertains to
351 identifying the genetic basis for migration phenotypes via locus associations such as genome-wide
352 association, or gene transcription studies, which may easily be confounded with identifying the highly
353 polygenic basis for growth because of the high genetic between-trait correlation. To identify genes
354 underlying also, or solely, the migration phenotype, a bivariate approach including size during the
355 sensitive period may be necessary. Investigations on associations based on transcription levels during the
356 sensitive period may also be promising, but pose logistical challenges due to the abovementioned
357 phenotype prediction required for lethal sampling.

358 There are several inferential limitations to our estimates. Genetic parameter estimates often pertain to
359 specific populations and conditions, but knowledge about their magnitudes may still provide useful
360 information [56, 57]. Our estimates within and between temperature environments, may then be useful
361 for future short-term predictions in response to selection under changing environmental conditions [58].
362 However, it is important to remember that heritabilities on the liability scale (and also heritabilities for

liability proxy traits) do not relate linearly to the probability scale across many factors, including the - environmentally governed - overall probability [18]. This effect was here exemplified by disparate heritability differences between temperature environments on the liability (more similar) vs. the proportional (less similar) scale. As recently discussed by de Villemereuil, Schielzeth [45], predicting the responses to selection for migration phenotypes may follow standard assumptions if based on liability-scale heritability, but less so on proportional-scale heritability.

Pertaining to correlation estimates, Cheverud [59] proposed that many genetic, but not phenotypic, correlations estimates may be inflated, especially under sample size limitations and when heritabilities are low [59, 60]. In comparison to studies assessed by Cheverud [59], our effective sample sizes - the product of number of families and geometric mean heritability - are at the lower end where Cheverud [59] suspected upwards bias. However, Bayesian heritability estimates for length matched those by REML that should yield reliable estimates [43], and which supports the presence of moderate to high heritabilities. For migrant probability estimates, we rely on comparisons with previous estimates. To our knowledge, the only study in Atlantic salmon estimated a small heritability on the proportional scale (0.16 ± 0.05) at a lower migrant rate (0.18) than in the present study [55]. In that study migrants were defined as exceeding a particular size threshold in spring [55], altogether making a comparison difficult. In other salmonids, similarly high estimates for migrant probability liability as presented here exist, such as 0.61 in cultured rainbow trout [51] and 0.52-0.56 in wild brook charr [61]. Thus, migrant probability liability in salmonids may exhibit high heritabilities, which lends some support to the correlation estimates. Furthermore, previous genetic correlations estimates in rainbow trout between body length at age 12 and 15 months and migrant probability one year later, under much larger sample sizes, also were relatively high and exceeded the phenotypic correlation [51]. Thus, the detected stronger genetic than environmental relationship may not, at least entirely, be a statistical artefact.

(c) Temperature effects on migration phenotypes: liability vs. threshold variation

Threshold model interpretations affect biological inferences, including inferences about the here-detected temperature difference. Using the threshold model (or its variants) it is generally not possible to discern variation for thresholds from liability [41, 62]. This statistical uncertainty extents to general definitions. As a hypothetical example, it may be unclear whether circulating hormones and their receptors should be regarded as contributing commonly to liability variation, or the latter to threshold variation [as suggested in 24]. Here, we did not adopt either view when interpreting our results, but discuss all possibilities.

A possible interpretation for the detected temperature difference for average migrant probability (not controlled for length) could be that temperature affects body length positively. This more rapid growth in

the warm environment would then increase migrant probability because more individuals exceed a size threshold during a sensitive period. Assuming equal thresholds between temperatures, this idea leads to the expectation that controlling for size results in equal migrant rates. However, length-controlled migrant probability was higher in the cold environment (**figure 2**). This result could then be interpreted as the presence of a lower size threshold in the cold environment, supported by the high genetic correlations between environments as predicted in [62]. Nonetheless, because the exact sensitive period remains unknown, the length measured at a presumed sensitive period may be different than the length expressed at the actual sensitive period. This bias between approximated and actual liability proxy length may be larger in the warm than the cold environment simply because growth proceeds more rapidly in the warm environment. Thus, subtle methodological bias can create statistically different migrant rates at a standardised length that mimic the presence of different environmental (or in other cases: population) thresholds.

To not confound liability proxy bias with differences for environmental (or population) thresholds it would be necessary to base inferences on the true liability, or, when information is lacking, on the liability proxy but as expressed at the true sensitive period. This leads to the question how close we got to the true period. The developmental transition is inducible by shortening daylength from 24 to 12 h for 6 weeks [63] and may occur when daylength is shortened to between 15 and 12 h [30]. For our experimental setting, a 6-week period after daylengths got shorter, or daylengths between 15 and 12 h, occurred shortly before, or at the beginning of, the size recording period at the end of the summer and the middle of the feed restriction, respectively (**figure 1** ***c***). Because the feed restriction reduced specific growth rates by about 50% but not migrant probability, it appears likely that the sensitive period preceded the feed restriction, as similarly inferred for amago salmon [64]. Future experiments destined to identify the sensitive period - a required prior for sound investigations on the physiological or genetic mechanisms determining the developmental transition - may thus be worthwhile if covering the entire summer.

Data accessibility

Underlying data and R-scripts are available on the Dryad Digital Repository: https://datadryad.org/stash/share/clrV_-cHbjDel14IKMf9BnLUZiqublsJeny0seSzvX8

Authors' contributions

P.D. and C.P. conceived the study, C.P. and J.E. contributed materials, C.P. coordinated genotyping, P.D. and N.P. performed experiments and recorded data, P.D. analysed data and wrote the manuscript. All authors critically revised the article and contributed to its final version.

Competing interests

We declare no competing interests.

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Tables

Table 1. Model mean posterior estimates (on the liability scale), lower and upper 95% credible intervals, and number of effective samples (N_{eff}) for the initial univariate generalized animal model on migration phenotype binaries of 663 Atlantic salmon individuals from 32 half-sib families. Variances were modelled as either diagonal (tanks, residuals) or unstructured (dams, animals) covariance matrices for the two temperature environments. Residual variance was fixed to 1 in each environment, resulting in scaling of all components relative to the residual variance. Effects or variances different from zero (i.e., credible interval not including zero) are in bold.

term	mean	lower	upper	N_{eff}
mean effects				
model intercept	-0.467	-1.325	0.423	15392
temperature (cold - warm)	**1.942**	**0.988**	**2.920**	13242
feed (full - restricted)	-0.010	-0.687	0.677	14000
maturation (immature - mature)	1.147	-0.892	3.161	14669
feed:temperature	-0.514	-1.607	0.537	14000
feed:maturation	-1.130	-3.536	1.151	14000
variance effects				
tank cold	0.127	0.000	0.455	13432
tank warm	0.166	0.000	0.608	14000
dam cold	0.182	0.000	0.691	13359
dam cold,warm	0.046	-0.139	0.319	13601
dam warm	0.188	0.000	0.694	14000
animal cold	**2.677**	**0.641**	**4.766**	14000
animal cold,warm	**1.709**	**0.496**	**3.145**	13430
animal warm	**1.709**	**0.319**	**3.322**	14000

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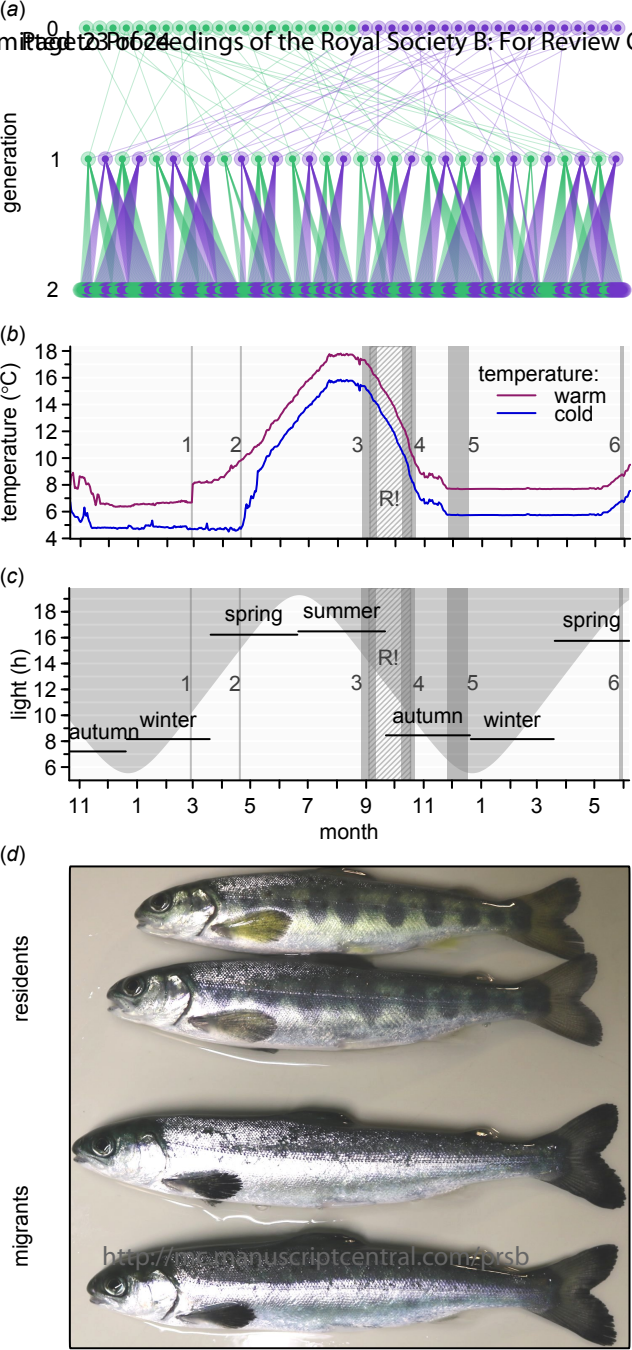

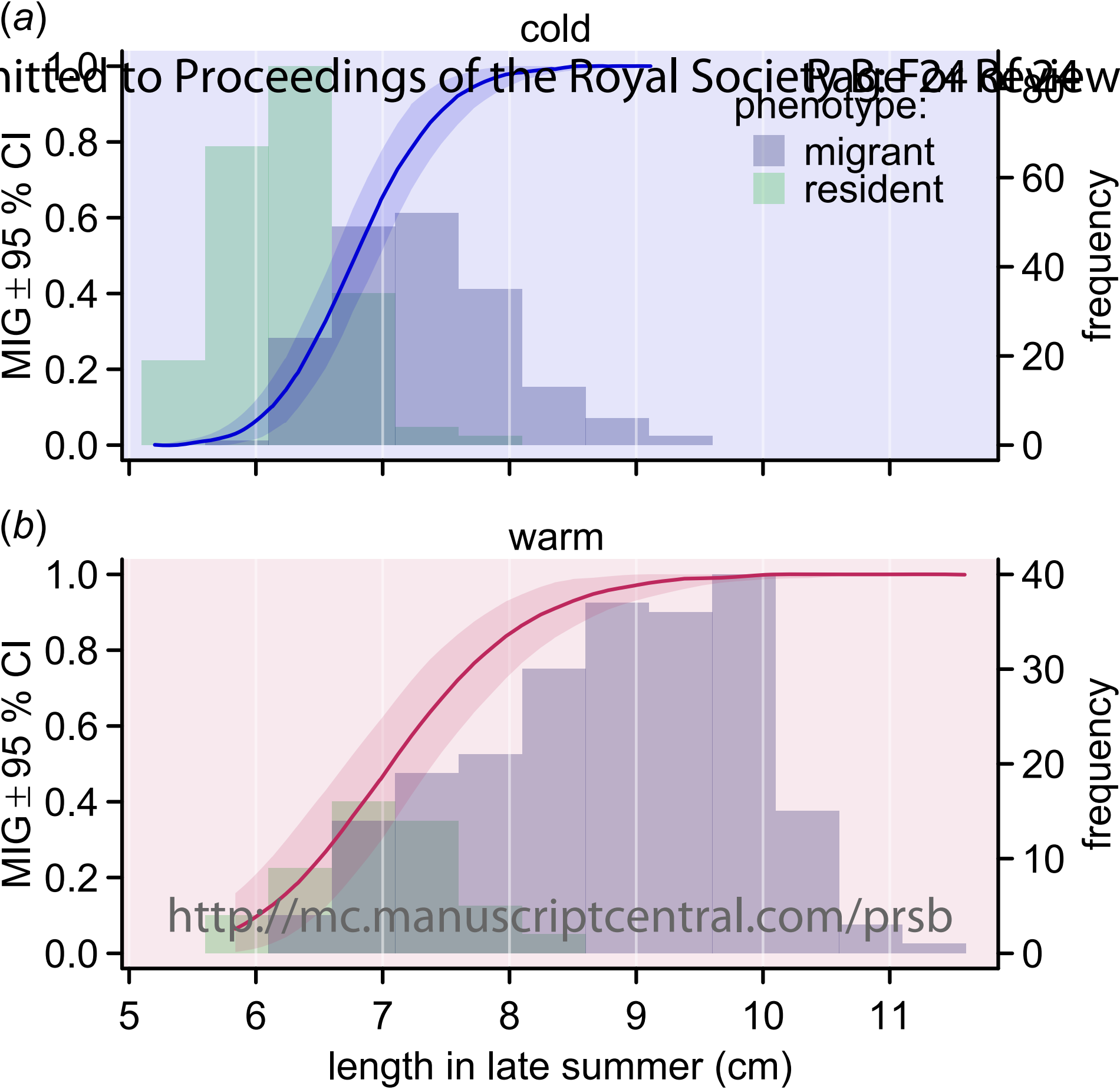

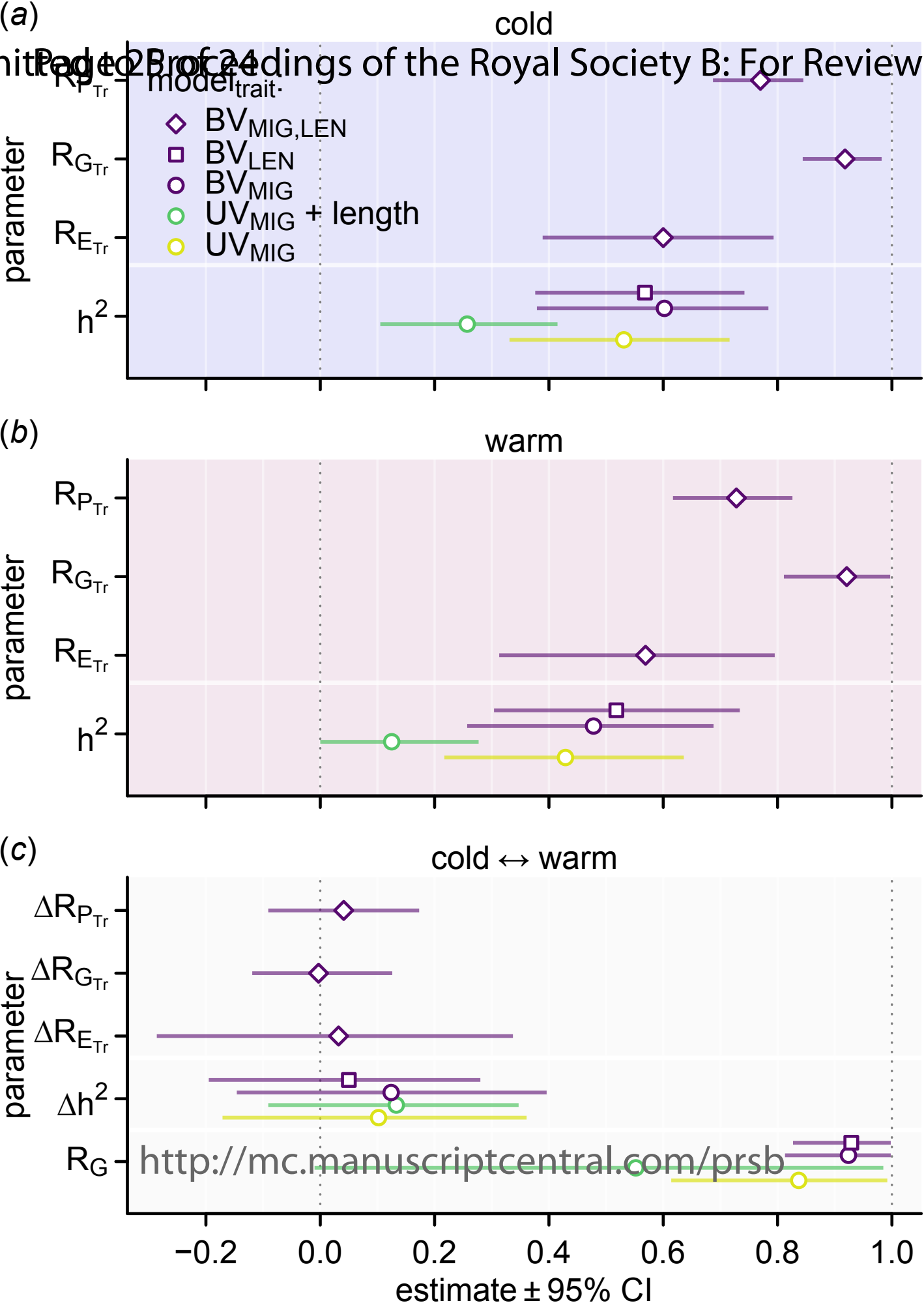

Electronic supplementary material to

Genetic growth potential, rather than phenotypic size, predicts migration phenotype in Atlantic salmon (doi: 10.1098/rspb.2020-0867)

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Contents:

Table S1. Model mean posterior estimates (on the liability scale), lower and upper 95% credible intervals, and number of effective samples (N_{eff}) for the reduced univariate generalized animal model on migrant phenotype binaries of 663 Atlantic salmon individuals from 32 half-sib families, including the effects of a locus with major effects on age at sexual maturation (*vgll3*, alleles: E = early, L = late maturation). Results for the binary response of migrant (1) or resident (0) are based on models not controlling for phenotypic length (model 1) or controlling for phenotypic length (model 2; length = length in late summer; mean centred and variance scaled).

Term	mean	lower	upper	N_{eff}
model 1				
model intercept	-0.338	-1.271	0.551	14516
temperature (cold - warm)	1.701	1.025	2.439	14564
Vgll3 (EE - EL)	-0.139	-0.780	0.512	14000
Vgll3 (EE - LL)	-0.276	-1.328	0.806	14000
model 2				
model intercept	1.287	0.616	1.913	7000
length (continuous)	2.946	2.435	3.507	7000
temperature (cold - warm)	-0.984	-1.660	-0.305	7000
temperature:length	-1.192	-1.864	-0.532	6317
Vgll3 (EE - EL)	0.164	-0.299	0.642	7000
Vgll3 (EE - LL)	0.213	-0.489	0.883	7000

Description of the general linear mixed to test for feed restriction treatment effects

To test for effect of the feed treatment (see methods for details) on growth rate, we fitted a general linear animal model under restricted residual maximum likelihood (REML) using asreml-R [1], to the (log) of length before and after the temporary feed restriction treatment. To estimate specific growth rate (i.e., proportional increase of length per unit time), we included date of measurement as a continuous covariate (date.integer; first measurement date taken as 0, days have a value of 1). We allowed the intercept (at the first measurement) and the growth rates to vary by temperature (cold, warm), feed restriction (full, restricted), and by migrant phenotype (resident, migrant). We also fitted all interactions among these terms. To account for the randomisation of treatments to tanks, we fitted random regression effects for tanks (i.e., a 2x2 covariance matrix for tank intercepts and date slopes with covariance between them). We accounted for the non-independence of, and among, individual data by including animal effects with additive genetic variance estimated via the inverse of the relationship matrix [2]. Using likelihood ratio tests between nested models, we determined that variances for animal effects (X_1^2 = 17.79, P < 0.001) and residuals (X_1^2 = 17.79, P < 0.001), but not tanks effects (X_3^2 = 1.97, P = 0.580), differed between temperature environments. We thus fitted these former two effect terms conditional on temperature environment, and with between-temperature covariance for the genetic effects. We found that the feed restriction had affected the specific growth rate of both residents and migrants and in both the cold and the warm environments (**table S2, figure S1**).

Table S2. ANOVA table for model terms of a univariate general animal model on body length phenotype in late summer of 663 Atlantic salmon individuals from 32 half-sib families. Intercept effects were estimated at the first measurement date (date.integer = 0). Statistical significance has been estimated based on *F*-tests with denominator degrees of freedom, DDF, approximated according to [3].

term	DF	DDF	*F*	*P*
model intercept	1	51.6	6162.0	< 0.001
migrant phenotype (resident, migrant)	1	518.8	448.4	< 0.001
temperature (cold - warm)	1	22.7	146.1	< 0.001
feed restriction (full - restricted)	1	11.8	7.6	0.018
date.integer (continuous)	1	11.3	904.1	< 0.001
migrant phenotype:temperature	1	206.0	8.8	0.003
migrant phenotype:feed restriction	1	550.9	0	0.968
temperature:feed restriction	1	14.7	0.1	0.782
migrant phenotype:date.integer	1	1302.0	430.9	< 0.001
temperature:date.integer	1	1302.0	9.7	0.002
feed restriction:date.integer	1	1302.0	122.0	< 0.001
migrant phenotype:temperature:feed restriction	1	407.8	0.3	0.607
migrant phenotype:temperature:date.integer	1	1302.0	8.1	0.004
migrant phenotype:feed restriction:date.integer	1	1302.0	38.3	< 0.001
temperature:feed restriction:date.integer	1	12.0	0.1	0.757
migrant phenotype:temperature:feed restriction:date.integer	1	409.3	10.8	0.001

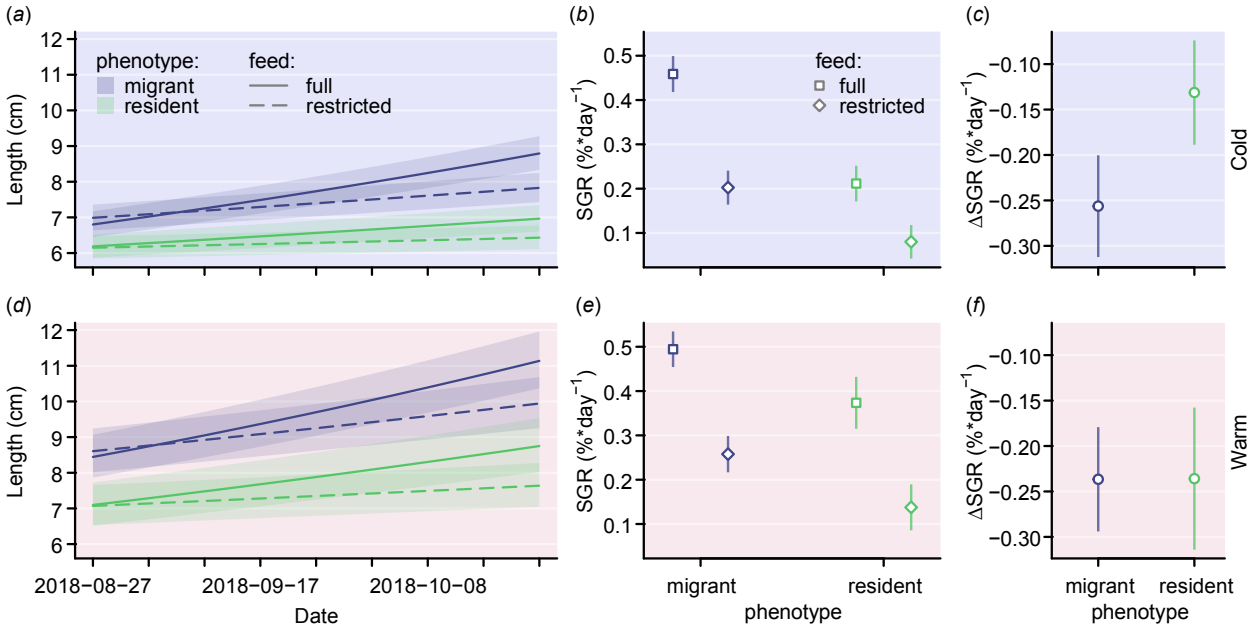

Figure S1. Model predicted average size trajectories (*a*, *d*) and associated specific growth rates (SGR; *b*, *e*) for prospective migrants or residents that were either fully fed or temporarily restrictedly fed in either a cold (upper row) or a 2°C warmer (lower row) environment, and the corresponding contrasts for SGR between the feeding treatments (ΔSGR; *c*, *f*). Estimates refer to the model as in **table S2**.

Description of the generalised linear mixed model to estimate male maturation rate

To estimate male maturation rate, we fit a generalised linear animal model with probit-link function to maturation binaries recorded during spawning time. We only fitted one overall mean effect (intercept). To account for the randomisation to tanks, we fitted random effects for tanks and to account for the non-independence among individual data, we including animal effects with additive genetic variance estimated via the inverse of the relationship matrix [2]. We used priors following [4] and methods as described for univariate Bayesian models in the main manuscript. We found that among tank effect variance was absent or negligible but that additive genetic variance was present (although methodologically inflated; **table S2**) and predicted a marginal overall maturation rate of males in the warm environment of 0.19 (95% CI: 0.08-0.33; **table S2**).

Table S3. Model mean posterior estimates (on the liability scale), lower and upper 95% credible intervals, and number of effective samples (N_{eff}) for the univariate generalized animal model on the sexual maturation phenotype binaries (0 = immature, 1 = mature) during the first year of 114 Atlantic salmon individuals from 30 half-sib families. Estimates were obtained for only males (no female matured) and only in the warm environment (no maturation occurred in the cold environment). Residual variance was fixed to 1, resulting in scaling of all components relative to the fixed residual variance. Variances different from zero (approximated by credible interval not including zero) are in bold.

Term	mean	lower	upper	N_{eff}
mean terms				
model intercept	-1.647	-2.615	-0.758	10000
variance terms				
Tank (V_C)	0.417	0.000	1.449	10210
Animal (V_A)*	**2.127**	**0.600**	**3.828**	10000

*Estimate is inflated because it is based on the coefficients of relatedness among individuals, but genotypes for the locus with major effects on maturation rate (*vgll3*) did not vary within families (leading to an absence of the expecting Mendelian sampling variance within families) due to the breeding design of using only *vgll3* homozygous parents as reported in [5].

Prior sensitivity analysis

We also assessed how prior specifications influenced the results. We focussed on the binary trait (MIG) because Bayesian heritability estimates for LEN closely matched estimates by residual maximum likelihood (REML), although the genetic correlation between environments was higher under REML (**figure S2**), whereby REML is the recommend method for animal models of continuous responses [4]. We compared the bivariate results (responses: MIG, LEN; including GxE) with univariate results (MIG per environment) obtained with recommended priors for binary animal models (following a X_1^2 distribution) [4]. We also separately assessed the effects, relative to the univariate models, of either including GxE for MIG or also fitting LEN per temperature environment. For the bivariate model, we varied prior specifications that resulted in different prior distributions for the variances and covariances. For the variances, we varied the parameter expansion variance scale by up to two magnitudes higher and lower than used (100, 10, 1, 0.1, 0.01), resulting in increasing prior densities for the proportions of the phenotypic variances towards one and zero, respectively. For the covariances, and thus correlations, we specified prior distributions that were either relatively flat for correlations (in *MCMCglmm*: nu = dimension of C, G, or R + 1) or showed higher densities towards -1 and 1 (in *MCMCglmm*: nu = dimension of C, G, or R). We found all investigated modelling variants to affect model estimates, but none strong enough to compromise our major inferences (**figures S2-S4**).

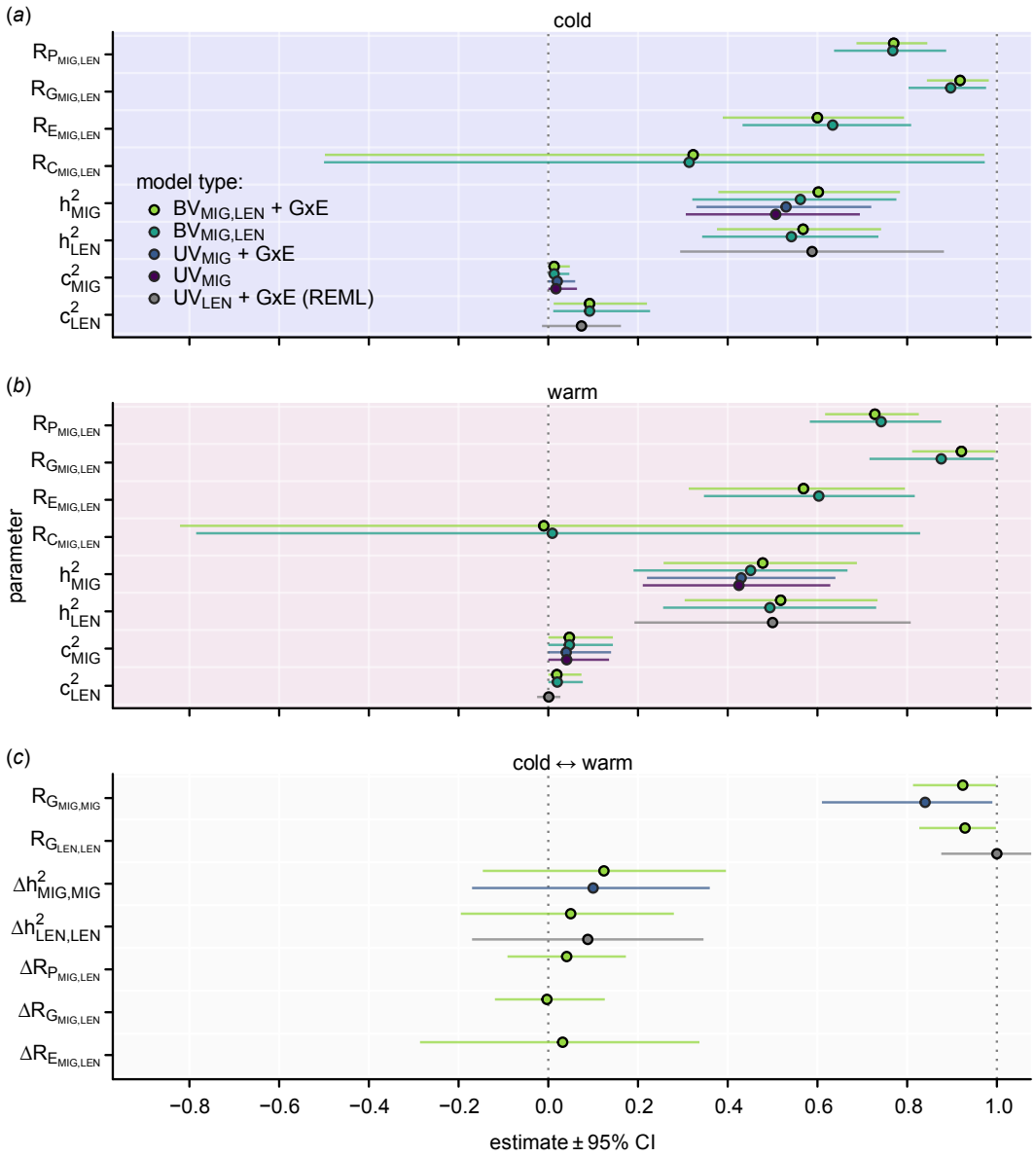

Figure S2. Comparison of the Bayesian model estimates for (co)variance components with increasing model complexity for the binary (MIG) and with REML estimates for the continuous trait (LEN). Heritability estimates for MIG increased in the following order: univariate single environment (UV) < univariate two environments (UV + GxE) < bivariate single environment (BV) < bivariate two environments (BV + GxE). Unfortunately, it is not clear whether model estimates in the absence of an explicitly modelled between-environment correlation (GxE) for the additive genetic effects should equal estimates in the absence of such modelled GxE. Furthermore, it is not clear whether model estimates for a single trait should equal those for estimates when a correlated second traits is modelled. Error bars show 95% credible intervals or approximate confidence intervals based on the delta method for REML estimates.

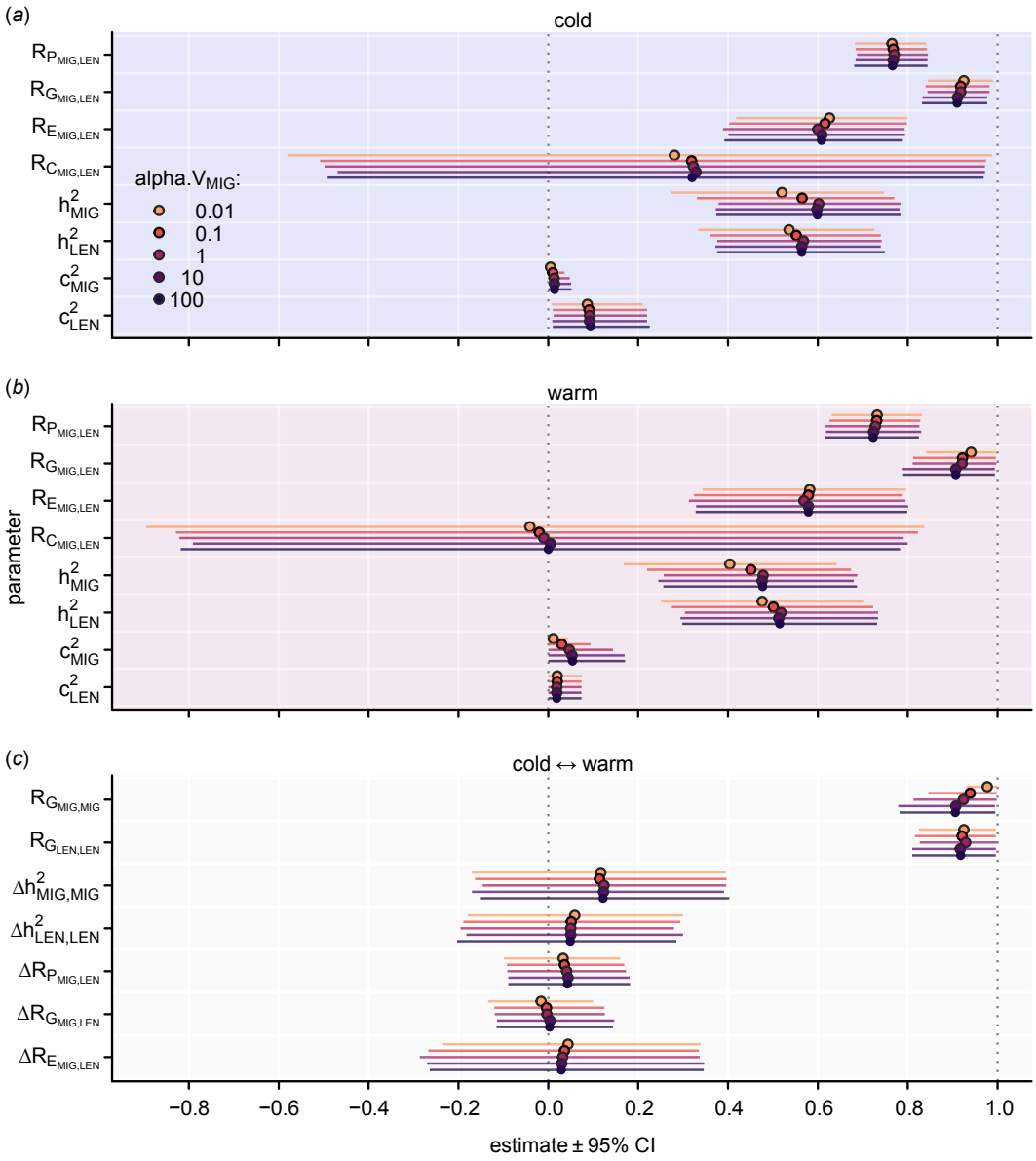

Figure S3 Comparison of the Bayesian model estimates for (co)variance components under different prior specifications for the parameter expansion variance scale (in *MCMCglmm* specified as "alpha.V") for the genetic and common environmental covariance matrices of the binary trait (MIG). For MIG heritability (h^2) and phenotypic proportion of the common environmental variance (c^2), the priors specify an increasing density towards 0 (alpha.V specified as either 100, 10, 1, 0.1, or 0.01). The parameter expansion variance scale specification for the continuous trait (LEN) was kept constant (alpha.V = 1). It could be noted that a smaller variance scale resulted in smaller heritability (h^2) and proportion of common environmental variance (c^2) of MIG when alpha.V fell < 1. Similar effects on h^2, but not c^2, were also observed for LEN.

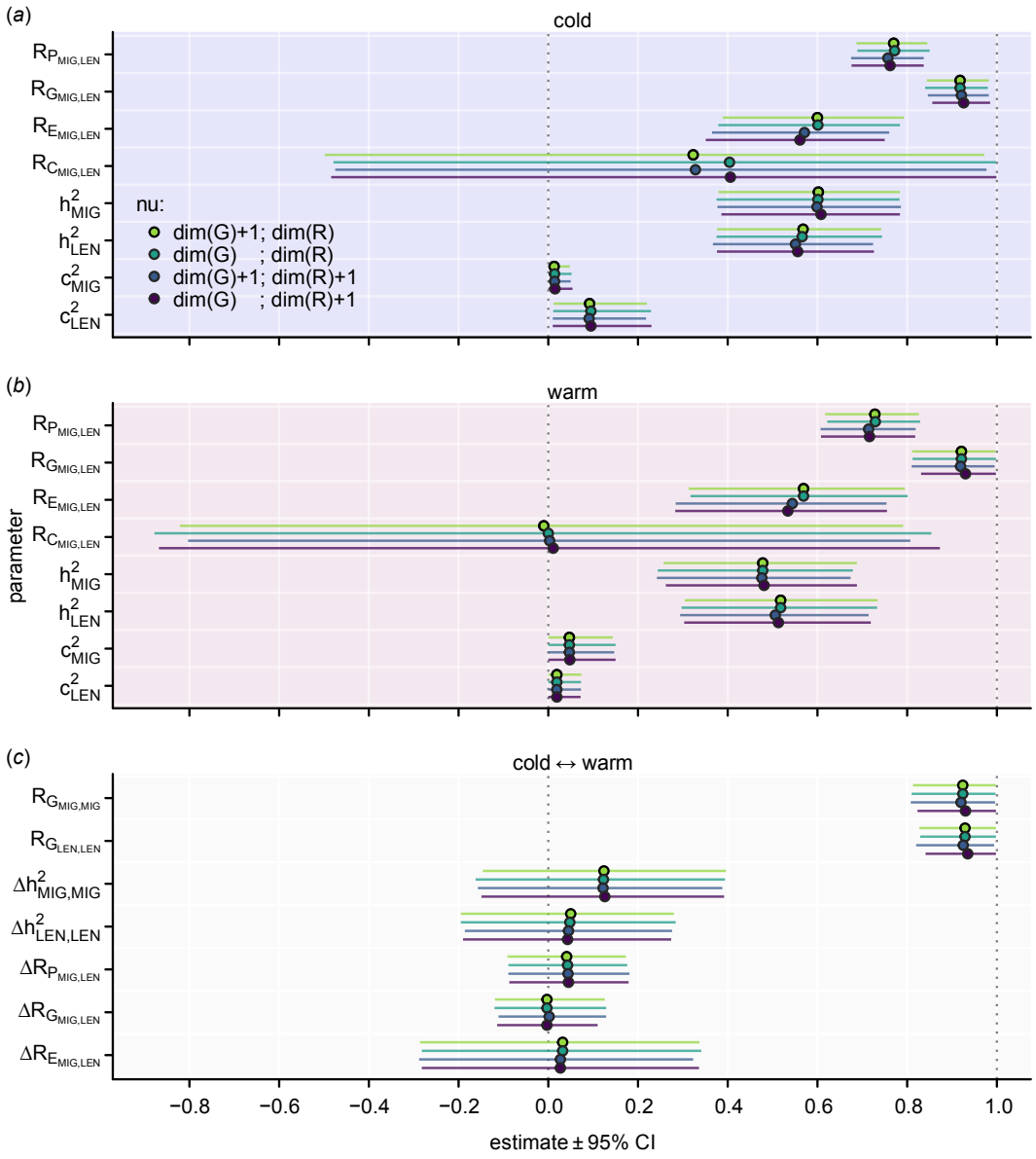

Figure S4. Comparison of the MCMC bivariate model estimates for (co)variance components varying the prior specification for the degree of believe parameter (nu) for combinations of the genetic (G, also applied for the environmental C) and residual (R) covariance matrices. The different priors result in either relatively flat distributions for the correlations (in *MCMCglmm*: nu = dimension [dim] of G or R + 1) or distributions with higher densities towards 0 and 1 (in *MCMCglmm*: nu = dimension [dim] of G or R). We noted an effect on the between-trait correlation for the common environmental effects within the cold environment ($R_{C_{MIG,LEN}}$; ***a***), which is unsurprising because common environmental effects were inferred as absent for one trait (c^2_{MIG}) but present for the other (c^2_{LEN}), thus lacking data information on correlations. As a result, we

refrained from making inferences about $R_{C_{MIG,LEN}}$, and effects of $R_{C_{MIG,LEN}}$ on $R_{P_{MIG,LEN}}$ were marginal because of the low or absent c^2. A second effect could be noted on the between-trait covariance for the residual environmental effects within both temperature environments ($R_{E_{MIG,LEN}}$; ***a***, ***b***). Given the prior knowledge of a suspected causal relationship between the two traits and for each trait between environments, but uncertainty whether this correlation is stronger at the environmental or genetic level, it is unclear which prior is more appropriate (i.e., whether assuming a between-trait correlation closer to zero or unity). However, this difference affects inferences only marginally and genetic correlation estimates appeared unaffected.

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