



# Polygenic and major-locus contributions to sexual maturation timing in Atlantic salmon

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## Abstract

Sexual maturation timing is a life-history trait central to the balance between mortality and reproduction. Maturation may be triggered when an underlying compound trait, called liability, exceeds a threshold. In many different species and especially fishes, this liability is approximated by growth and body condition. However, environmental vs. genetic contributions either directly or via growth and body condition to maturation timing remain unclear. Uncertainty exists also because the maturation process can reverse this causality and itself affect growth and body condition. In addition, disentangling the contributions of polygenic and major loci can be important. In many fishes, males mature before females, enabling the study of associations between male maturation and maturation-unbiased female liability traits. Using 40 Atlantic salmon families, longitudinal common-garden experimentation, and quantitative genetic analyses, we disentangled environmental from polygenic and major locus (*vgll3*) effects on male maturation, and sex-specific growth and condition. We detected polygenic heritabilities for maturation, growth, and body condition, and *vgll3* effects on maturation and body condition but not on growth. Longitudinal patterns for sex-specific phenotypic liability, and for genetic variances and correlations between sexes suggested that early growth and condition indeed positively affected maturation initiation. However, towards spawning time, causality appeared reversed for males whereby maturation affected growth negatively and condition positively via both the environmental and genetic effects. Altogether, the results indicate that growth and condition are useful traits to study liability for maturation initiation, but only until maturation alters their expression, and that *vgll3* contributes to maturation initiation via condition.

## KEYWORDS

feed restriction, genetic correlation, large effect locus, life-history trait, sexual maturation

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## 1 | INTRODUCTION

Sexual maturation timing is a central life-history trait that contributes to fitness by trading off individual survival and reproductive success, and contributes thereby to per capita population growth rate (Bernardo, 1993; Roff, 1992, 2002; Stearns, 1992; Wells et al., 2017). The binary response of maturation initiation may be controlled via one or several thresholds in an underlying "liability" (Falconer, 1965). As a statistical approach to binary responses, the liability may be assumed to be a normally distributed "trait", which consists of all combined (often unknown) environmental and genetic effects underlying the binary trait (Dempster & Lerner, 1950; Falconer, 1965; Wright, 1934). The liability underlying the initiation of maturation is often approximated by body size, growth, or condition (Andersson et al., 2018; Roff, 2002; Stearns, 1992; Taranger et al., 2010; Wells et al., 2017), which indeed may signal current energy status (Dupont et al., 2014; Koyama et al., 2020; Parker & Cheung, 2020; Shalitin & Phillip, 2003). However, disentangling cause and effect is a long-standing challenge in studies on maturation and its liability traits (Alm, 1959; Bell et al., 2018; Cousminer et al., 2013; Kause et al., 2003; Taranger et al., 2010). For example, as opposed to growth controlling maturation, ongoing maturation can temporally boost growth, such as the human puberty growth spurt. Likewise, the maturation process can reduce somatic growth by competing with resources and lower condition by depleting reserves, or affect both growth and condition, for example, via appetite (Andersson et al., 2018; Stearns & Koella, 1986; Taranger et al., 2010). Related to this problem, fundamental knowledge on the presence and relative importance of environmental versus genetic contributions to maturation timing and their link to liability traits remains limited (Dunlop et al., 2009; Enberg et al., 2012; Gjedrem & Baranski, 2005; Law, 2007).

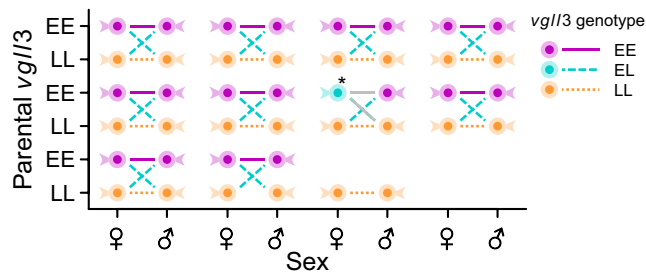
Contrary to previous assumptions that life-history traits are polygenic traits (Lande, 1982; Roff, 2002), usually coded by many loci with small effect (Hill, 2010; Lynch & Walsh, 1998), major locus effects on life histories are being identified in an increasing number of species (e.g. Cheng et al., 2018; Pearse et al., 2019; Wang et al., 2018). In Atlantic salmon (*Salmo salar*), a single locus (*vgll3*) explained a large proportion of phenotypic variation for maturation age (33%–39%; Ayllon et al., 2015; Barson et al., 2015), and for maturation probability at a specific age (Fjellidal et al., 2020). The *vgll3* locus has been reported to show sex-specific dominance for sea age at maturity in two previous studies (Barson et al., 2015; Czorlich et al., 2018), with dominance of the "early" allele (*vgll3*\*E) in males and dominance of the "late" allele (*vgll3*\*L) in females. However, dominance was not inferred by another study (Ayllon et al., 2019). Allele frequencies, and thus the expected *vgll3* contribution to the phenotypic variation, vary widely across wild and aquaculture salmon populations with generally lower contribution in North American-origin than European populations (Ayllon et al., 2015, 2019; Barson et al., 2015; Boulding et al., 2019; Czorlich et al., 2018; Kusche et al., 2017; Mohamed et al., 2019). However, in addition to major locus effects, an extensive polygenic background for maturation timing in Atlantic

salmon extends across at least 28 of the 29 chromosome pairs (Sinclair-Waters et al., 2020).

These findings not only offer opportunities for understanding life-history trait evolution in terms of compound effects of major loci and polygenic loci, but also hold implications for medium-term evolutionary predictions. Specifically, the expected selection response across several generations differs depending on whether a trait is governed by only polygenic or also major loci (Roff, 2002). For example, major loci may have strong effects on genetic variance (and the associated heritability) and on genetic correlations, and may briefly but greatly change the tempo of adaptation by allowing for large fitness surface steps (Agrawal et al., 2001). However, the evolutionary importance of major loci remains a subject of debate. One prediction for adaptive major loci is their rapid fixation by selection, especially when they contribute to an otherwise polygenic trait (Lande, 1983). In contrast, variation of adaptive major loci underlying threshold traits, such as maturation timing, may be expected to have longer-lasting consequences. This is because frequency-dependent selection and the cryptic genetic variance typical for threshold traits may retain genetic variation (Dempster & Lerner, 1950; Roff, 1996). Nonetheless, how major and polygenic loci may jointly shape a life-history trait, such as maturation timing, has rarely been considered empirically.

In Atlantic salmon, there is a long history of sexual maturation studies (Andersson et al., 2018; Marschall et al., 1998; Meerburg, 1986) and investigations of maturation as a threshold trait (Myers & Hutchings, 1986; Piché et al., 2008). This species offers features allowing for jointly assessing environmental and genetic effects of maturation and the liability traits growth and body condition. As one advantage, readily available pedigreed hatchery populations allow for planned breeding of highly fecund individuals. The many offspring with tightly connected pedigrees combined with common-garden experimentation followed by quantitative genetic analyses enable dissection of environmental from genetic effects on maturation and its presumed liability traits and estimation of environmental, genetic, and phenotypic correlations between maturation and liability traits. Perhaps the biggest advantage, however, is the observation that Atlantic salmon males, but rarely females, can mature during their first year (reviewed by Marschall et al., 1998). This biological peculiarity enables estimating environmental and genetic contributions to maturation in males and to growth and condition in nonmaturing females. By genetically relating male maturation to the maturation liability traits of their female relatives, it is possible to infer the presence and relative importance of genetic components of maturation timing and its maturation-unbiased liability traits.

Here, we implemented a quantitative genetic breeding design for 42 pedigreed Atlantic salmon parents with homozygous *vgll3* genotypes and studied 2,608 male and female offspring from 40 families (Figure 1), which allowed us to separate polygenic from major locus effects. The longitudinal study of these families in a common-garden environment, involving a temporary food restriction treatment, enabled estimates of environmental versus genetic contributions to



**FIGURE 1** Realized breeding design with the 11 anticipated  $2 \times 2$  factorials (22 dams  $\times$  22 sires, homozygous for *vgII3*). Two factorials were curtailed because of low egg survival, and for another, a dam (marked by asterisk) was determined to be *vgII3* heterozygote upon confirmatory genotyping. *VgII3* genotypes of breeders are represented by the colour of the fish shapes and *vgII3* genotypes of created families, which are represented as lines connecting the fish shapes, are represented by colours and line types corresponding to the legend

maturation timing of first-year Atlantic salmon and its presumed liability traits, which are expected to vary during development. To avoid reverse causality when associating maturation with liability traits that are known to be affected by maturation (growth, condition), we tested whether male maturation timing correlates genetically with maturation-unbiased female somatic growth or body condition. Further, we evaluated whether the known major locus effect on maturation (co)expresses via growth and/or condition by testing for its genetic architecture and quantifying its relative contribution to the phenotypic variance for maturation in males and growth and condition in males and females. By studying these relationships and contributions over a five-month period, we were able to describe how each of them changes between summer and spawning time in late autumn.

## 2 | MATERIALS AND METHODS

### 2.1 | Fish population, breeding and experimental design, data collection

The experimental salmon cohort was parented by pedigreed hatchery fish, maintained by the Natural Resources Institute Finland (Laukaa, Finland), which originate from the River Neva, Russia; draining into the Baltic Sea in the Gulf of Finland. In November 2017, we crossed 44 parents with known *vgII3* genotype as 11  $2 \times 2$  factorials of unrelated *vgII3* homozygous individuals; each factorial yielded four reciprocal same-*vgII3*-genotype offspring families (EE, EL, LE, LL; details on realized design in Figure 1).

We reared the experimental cohort in a water recirculation system at the University of Helsinki (see also Debes et al., 2020), controlled for parameters known to affect growth or sexual maturation timing, namely water temperature, oxygen, dissolved nitrate components, and natural light cycle (Andersson et al., 2018; Taranger et al.,

2010). We split each family by randomizing ~400 eggs across two egg-incubators (kept in darkness). At first feeding, we randomized per family an equal number of individuals from each incubator into eight similar tank replicates (due to differential egg mortality: mean = 5, range: 1–7, total per family across tanks: mean = 155, range: 21–234). Due to mortality, removal of sick individuals, unknown genotype or sex, culling of random individuals to reduce densities, and removal of individuals with mismatch between genotypic and phenotypic sex (see below), an average of 65 individuals (range: 9–114) for each of 40 half-sib families were available for analysis at the completion of the study (totalling 2,608 individuals). To maintain natural maturation schedules, water temperature followed a seasonal cycle (range: 6.3–17.7°C) and the light regime, controlled by a digital astronomical time switch, followed the natural cycle (latitude 61.054°; longitude: 25.042°). Fish were fed several times a day initially by hand and automatically thereafter ad libitum (except for a food-restriction period as described below) using a commercial salmon diet starting 09 March 2018. Feed pellet sizes matched fish-size compositions at all times. In August 2018, fish size allowed passive integrated transponder tagging to enable individual identification. We then anaesthetized (using tricaine methanesulphonate), fin clipped, and tagged individuals. The fin clips allowed for genotyping to assign family, determine molecular sex, and confirm *vgII3* genotype (see below). To allow for testing whether limited food availability was sufficient to halt the (already initiated) sexual maturation process, and whether this differs among *vgII3* genotypes, we applied a food-restriction treatment for a five-week period between September and October. The treatment consisted of either ad libitum feeding for seven days per week (full food; as before and after the treatment period) or ad libitum feeding for two days per week with no feeding for two or three days between feeding days (restricted food). We determined individual fork length ( $\pm 1$  mm) and wet mass ( $\pm 0.01$  g) five times between August and December in 4- to 5-week intervals. Before each measurement, we ceased feeding for 48 h to minimise measurement bias by ingested food. We determined maturity status in December (during spawning time) by checking for extruding milt while gently pressing the abdomen.

In December, we also culled and dissected 83% of all fish ( $n = 2,174$ ). Genotypic and phenotypic sex matched in 100% of the culled fish with determined genotypic sex, and we used phenotypic sex in 17 fish with undetermined genotypic sex. We confirmed maturity status in 100% of the culled fish. We defined condition as deviation from the slope of logarithmic mass on logarithmic length (Froese, 2006)—a close correlate of first-year salmon lipid content in nonmaturing females and, when assessed outside of the spawning season or accounting for maturation, also for males (Herbinger & Friars, 1991; Sutton et al., 2000). This condition index allows for expressing body condition as expected mass under a standardised length (here: geometric mean size of 11.19 cm) and for comparing condition as percentage mass difference under standardised length. Animal experimentation was conducted according to European Union Directive 2010/63/EU and under license ESAVI-2778–2018 by the Animal Experiment Board in Finland.

Genotypes and genotypic sex of the parental and the experimental cohorts were determined using a multiplex-PCR for 177 single nucleotide polymorphisms (SNPs) of a previously described panel (Aykanat et al., 2016), followed by Ion Torrent (984 broodstock individuals from which we drew parental individuals) or Illumina platform (MiSeq or Next-Seq) sequencing (parental individuals, experimental offspring cohort). Using a subset of polymorphic SNPs that did not show high linkage disequilibrium (all pairs with  $r < 0.5$ ), we reconstructed the experimental cohort's grandparents (with 131 usable SNPs), under maximum likelihood (Jones & Wang, 2010) (Supporting Information), and we assigned the experimental individuals to their 44 putative parents (with 141 usable SNPs) with a likelihood approach (Anderson, 2010). Merged information about grandparents and parents yielded a three-generation pedigree on which we based the additive relationship matrix utilized in animal-model analyses (Henderson, 1973).

## 2.2 | Data analysis

We fitted a series of uni- and multivariate general and generalized animal models to male maturation data scored as a binary trait in December ( $N = 1,280$ ), and male and female length or condition data recorded at five time points between August and December ( $N = 2,608$  per time point; 13,040 in total). To allow for estimating biologically meaningful proportional effects that hold across body sizes, and stabilise variances across sizes, length data were log-transformed, whereas condition data were already on the log scale, and length and condition data were both mean-centred and variance scaled to facilitate analysis.

In models, we included all variables of interest along with all meaningful interactions. Specifically, we were interested in sex-specific effects due to the temporary food restriction, *vgll3*, and their interactions, and in maternal (by dams), common environmental (by tanks), and polygenic (by relationship among individuals) effects. For *vgll3* we included additive and dominance effects in all models. We included dominance effects because dominance for *vgll3* has previously been identified (Barson et al., 2015; Czorlich et al., 2018) and including dominance, even in its absence, may not reduce accuracy of additive effect estimates (Duenk et al., 2017).

To estimate *vgll3* and feeding treatment effects and estimate maternal, common environmental, and polygenic variance for male maturation, we fitted a univariate model for male-specific maturation binaries (a cross-sectional trait) corresponding to:

$$Y = \mu + \beta_1 \text{Feed} + \beta_2 \text{Alpha} + \beta_3 \text{Delta} + \beta_4 \text{FeedAlpha} + \beta_5 \text{FeedDelta} + \text{tank} + \text{dam} + \text{animal} + \epsilon \text{error}, \quad (1)$$

where  $\mu$  is a constant,  $\beta_1 \text{Feed}$  the food restriction effect,  $\beta_2 \text{Alpha}$  the additive *vgll3* effect ( $\alpha_{\text{vgll3}}$ ; Alpha coefficients as -1, 0, 1 for *vgll3*\*LL, *vgll3*\*EL, *vgll3*\*EE, respectively),  $\beta_3 \text{Delta}$  the dominance *vgll3* effect ( $\delta_{\text{vgll3}}$ ; Delta coefficients as 0, 1, 0 for *vgll3*\*LL, *vgll3*\*EL, *vgll3*\*EE, respectively), and  $\beta_4 \text{FeedAlpha}$  and  $\beta_5 \text{FeedDelta}$  are the food restriction

effects on the additive and dominance effects, respectively. The terms *tank*, *dam*, *animal*, and  $\epsilon \text{error}$  refer to random tank (common environmental), dam (maternal), breeding value (additive polygenic), and residual (random environmental effects and measurement error) effects, respectively. Below, we commonly refer to residual effects as environmental effects. As is characteristic of animal models, we estimated the polygenic effects as inverse-relationship-matrix-predicted breeding values (Henderson, 1973). This model was fitted with a probit-link function and residual variance fixed to one under Bayesian Markov Chain Monte Carlo simulations, and using the R-package MCMCglmm v. 2.29 (Hadfield, 2010) in R v. 4.0.3. Generalized probit models can be interpreted like threshold models (Hadfield & O'Hara, 2015). The model was fitted using  $X_1^2$  priors for the variances of the random effects, as recommended for binary data animal models by de Villemereuil et al., (2013), and with four chains each for 3,000,000 iterations and sampled every 100th iteration. To avoid numerical errors, we invoked the MCMCglmm option for latent variable truncation between -7 and 7. Following Brooks and Gelman (1998), we diagnosed convergence as indicated by a scale reduction factor around one per chain, determined a burnin of 500,000 to reach a consistent scale reduction factor  $< 0.1$  between chains, determined a thinning of 1,000 to yield parameter autocorrelations at lag 2  $< 0.1$  per chain (leaving 10,000 samples), and confirmed whether MCMC resulted in sufficient mixing by visual examination of trace plots per chain.

To describe sex- and maturation-status-specific growth and condition trajectories, and also to test for feeding-treatment-effect and *vgll3*-effect trajectories, we fitted univariate longitudinal models to either length or condition data. The models were fitted for the continuous length or condition data (recorded longitudinally in both sexes). Specifically, we expanded the above equation (1) by adding sex effects (male, female), maturation effects for males (mature, immature), and a time covariate (to fit trajectories; average day of year for each sampling period), and all interactions, except that we did not interact *vgll3* effects with the maturation effect. The latter was due to an expected maturation bias of *vgll3* effects if obtained conditional on maturation status. Specifically, mature and immature males over-represent families with *vgll3*\*EE and *vgll3*\*LL genotypes, respectively. This may result in maturation-frequency biased samples per genotype and maturation group and thereby in loss of the polygenic background control provided by the breeding design. We fitted the models with (co)variances for random terms in (1) that we interacted with sex, except for tank effects that appeared equal between sexes. We allowed for different variances at each time point and for each sex, and for covariance(s) between sexes (except for residual effects that do not allow estimation of covariance between sexes) and across time (see detailed modelling approach in Supporting Information). The longitudinal models on length and condition data were fitted under REML, using ASReml-R v. 3 (Butler et al., 2009) in R v. 3.0.2

To estimate environmental and genetic variances for length or condition in males and females and correlations with maturation in males, and to investigate how these variances and correlations changed across development, we fitted a set of trivariate models.

The models were fitted for male-specific maturation binaries and either sex-specific length or sex-specific condition data per each of the five time points. All trivariate models followed equation (1) for each (sex-specific) response trait; we did not fit maturation effects for length or condition responses (because we were interested in the covariance with the maturation response). We allowed all variances to differ for each (sex-specific) trait and allowed for between trait covariance where possible. Specifically, we fitted  $3 \times 3$  unstructured covariance structures for dam, tank, and polygenic effects, and  $2 \times 2$  covariance structures for male-specific residual effects. Each trivariate model was fitted with probit-link function and residual variance fixed to one for the binary trait and identity link function for the continuous traits under Bayesian Markov Chain Monte Carlo simulation as described above. We here used parameter-expanded multivariate priors for the variances of the random effects. Following diagnostics as described above, we ran for each model two chains each for 1,050,000 iterations of 50,000, and sampled every 1,000th iteration, yielding 2,000 samples across both chains.

We estimated the proportions of phenotypic variance on the probit scale explained by the major locus ( $h^2_{vgll3}$ ), the presumed polygenic loci ( $h^2_{poly}$ ), and their sum ( $h^2_{total}$ ), which is, however, not straight forward for a single locus (Gianola et al., 2013; de Los Campos et al., 2015). We approximated  $h^2_{vgll3}$  by first assuming that the additive variance contributed by  $vgll3$  is  $V_{A,vgll3} = 2pq[\alpha_{vgll3} + \delta_{vgll3}(q-p)]^2$ , where  $p$  and  $q$  are the  $vgll3$  allele frequencies in the sample ( $vgll3^*E$ :  $p = 0.535$ ;  $vgll3^*L$ :  $q = 0.465$ ) and  $\alpha_{vgll3}$  and  $\delta_{vgll3}$  are the weighted averages of treatment-specific regression coefficient model estimates (on the probit scale). Similarly, we assumed that the dominance variance contributed by  $vgll3$  is  $V_{D,vgll3} = (2pq\delta_{vgll3})^2$ . We then calculated  $h^2_{vgll3} = V_{A,vgll3}/V_p$ , where  $V_p$  is the total phenotypic variance, including the variances contributed by  $vgll3$ . Likewise, we calculated the proportion of phenotypic variance explained by presumed polygenic additive genetic effects as  $h^2_{poly} = V_{A,poly}/V_p$ , where  $V_{A,poly}$  is the model estimate of additive genetic variance. Lastly, we estimated the total heritability as  $h^2_{total} = (V_{A,vgll3} + V_{A,poly})/V_p$ . It should be noted that we cannot exclude the possibility of large effect sizes of unknown genes contributing to the presumed polygenic variance.

We transformed probit scale estimates (and their 95% CI) to the probability scale for mean estimates using the “predict” function of the MCMCglmm v. 2.29 R-package and for variance and (polygenic) heritability estimates, while averaging over fixed effects, using the “QGparams” function of the QGglmm v. 0.7.4 R-package (de Villemereuil et al., 2016). To transform the major-locus probit variance to the probability scale, we first predicted the feeding-treatment-specific  $vgll3$  genotypic probability-scale means, while marginalising random effects, via the “predict” function. Across these genotypic means ( $Y_{obs}$ ) we then estimated probability-scale  $\alpha_{vgll3}$  and  $\delta_{vgll3}$  by multiple regression (weighted across feeding treatments;  $Y_{obs} = \mu_{obs} + \beta_{1obs}Alpha + \beta_{2obs}Delta$ ). To finally estimate the probability-scale major locus variances and heritabilities associated with these probability-scale regression coefficients, we proceeded

as reported above for probit-scale estimates but using the respective probability scale estimates.

We present estimates and their 95% credible intervals across posteriors (based on Bayesian models) or their approximate 95% confidence intervals (mean  $\pm 2$ \*standard error; based on REML models) on the probit scale for maturation and on back-transformed measured scales for all traits. To allow for wider applicability, we also report most effect sizes for length and condition on the proportional scale, which can be more meaningfully interpreted across body sizes.

## 3 | RESULTS

### 3.1 | Male sexual maturation is affected by $vgll3$

Of the 2,608 under-yearling Atlantic salmon, zero of 1,328 females and 455 of 1,280 males were mature by the December following hatching. The average probability to mature for males was estimated as 0.36 (0.22–0.51) in the full food and as 0.38 (0.24–0.54) in the restricted food treatment. The additive effects of  $vgll3$  ( $\alpha_{vgll3}$ ) on maturation timing, i.e. the effects of adding one  $vgll3^*E$  allele, were estimated on the modelled probit scale (which is, unlike the probability scale, the scale on which effects are linear) as  $\alpha_{vgll3,probit} = 0.94$  (0.50–1.43) in the full food treatment and as  $\alpha_{vgll3,probit} = 0.77$  (0.33–1.23) in the restricted food treatment ( $\alpha_{vgll3}$  probability scale effects in Figure S1). Their treatment-weighted average was  $\alpha_{vgll3,probit} = 0.86$  (0.43–1.30), which contributed a probit-scale variance of  $V_{A,vgll3} = 0.394$  (0.056–0.788). In contrast,  $vgll3$  dominance effects ( $\delta_{vgll3}$ ), i.e. the deviation of the heterozygous from the midpoint of the homozygous genotypes, were estimated to be smaller than the  $\alpha_{vgll3}$  effects, their credible intervals included zero in both feeding treatments (full food:  $\delta_{vgll3,probit} = -0.04$ , -0.34 to 0.27; restricted food:  $\delta_{vgll3,probit} = -0.11$ , -0.42 to 0.20), and also their treatment-weighted probit-scale variance was quite small ( $V_{D,vgll3} = 0.005$ , 0.000–0.019). The direction of dominance was, in contrast to previous study results (Barson et al., 2015; Czorlich et al., 2018), towards  $vgll3^*L$  (Figure 2a,b).

The food restriction contrasts showed little support for differences in maturation probability between feeding treatments or in  $vgll3$  effects. Specifically, all estimates were relatively small, the direction of the food restriction effect was inconsistent, with higher, equal, or lower maturation in the restricted relative to the full food treatment for  $vgll3$  LL, EL, and EE genotypes, respectively (Figure 2a,b), and all effect credible intervals included zero (probit scale treatment contrasts;  $vgll3^*LL$  genotype: 0.06, -0.29 to 0.42;  $\alpha_{vgll3,probit} = -0.17$ , -0.47 to 0.157;  $\delta_{vgll3,probit} = -0.07$ , -0.50 to 0.35). When we omitted dominance and the food restriction terms from the model, we estimated an overall  $\alpha_{vgll3,probit} = 0.84$  (0.44–1.24), which is close to the treatment-weighted average accounting for dominance reported above.

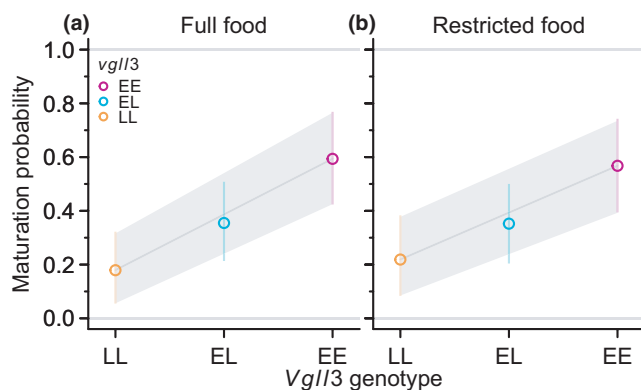
Using the weighted average across feeding treatments,  $\alpha_{vgll3}$  contributed about 13% phenotypic probit-scale variance for maturation ( $h^2_{vgll3,probit} = 0.133$ , 0.032–0.234) and on the observed (obs.) scale



about 7.7% ( $h^2_{vgll3,obs} = 0.077, 0.023-0.134$ ). However, polygenic effect contributions exceeded those by *vgll3* on both the probit and the observed scales. Specifically, additive polygenic effects contributed to the phenotypic variance on the probit-scale about 48% ( $h^2_{poly,probit} = 0.478, 0.259-0.684$ ) and on the observed scale about 27% ( $h^2_{poly,obs} = 0.267, 0.132-0.396$ ). In contrast, contributions to the phenotypic variance appeared negligible by both maternal effects ( $m^2_{probit} = 0.020, 0.000-0.074$ ) and by common environmental effects ( $c^2_{probit} = 0.005, 0.000-0.019$ ). The total heritability, i.e. based on both polygenic and *vgll3* effects, was estimated on the probit scale as  $h^2_{total,probit} = 0.611 (0.421-0.789)$  and on the observed scale as  $h^2_{total,obs} = 0.343 (0.230-0.450)$ . Averaged across posteriors, additive *vgll3* effects accounted for about one third to one quarter in the genetic variation of male maturation, specifically for 31.3% (2.8%–67.2%) of the total heritability on the probit scale and 23.3% (4.6%–42.9%) on the observed scale. It should be emphasized that these estimates are specific to the maturation rates and the experimental settings with near-equal *vgll3* allele frequencies.

### 3.2 | Temporal expression of body length and condition differs between maturing males and nonmaturing males and females

Using models on longitudinal data, we assessed the temporal expression of length and condition in maturing (mature) males and in nonmaturing males and females between August and December. Across time, nonmaturing males were 2%–3% shorter than females (Figure 3a,b,e,f). The body condition of nonmaturing males and females was much more similar (Figure 3c,d,g,h). Maturing males, in contrast, were initially about 5% and 6% longer (full food and restricted food) than nonmaturing males and about 2.5% and 3.5% longer (full food and restricted food) than females (Figure 3a,b,e,f).



**FIGURE 2** Model predicted maturation probability of first-year male Atlantic salmon ( $N = 1,280$ ) per major-locus (*vgll3*) genotype with the additive expectation in each of two feeding treatments (a,b). The slopes connecting the genotypic means in a and b represent back-transformed  $\alpha_{vgll3}$  estimates (modelled on the probit scale) and genotypic means and  $\alpha_{vgll3}$  have been adjusted for dominance major locus effects ( $\delta_{vgll3}$ ). Symbols represent mean estimates and error bars and bands show 95% credible intervals

However, the direction of this difference reversed towards autumn, when mature males were smaller than immature individuals in both feeding treatments (Figure 3a,b,e,f). These length differences in autumn were around 7% with immature males or 10% in immature females in the full food treatment, and 4% in immature males or 7% in immature females in the restricted food treatment (Figure 3e,f). Body condition of maturing males was, at all times, higher than that of nonmaturing males or females (Figure 3c,d,g,h). In detail, the condition difference of maturing males versus immature individuals was about 2% in summer, increased to 7% in early November, and declined thereafter (Figure 3g,h).

The temporary food restriction applied in September induced changes in body length and condition that were similar in nonmaturing males and females, but different in maturing males (Figure 3i,j). Specifically, when comparing treatment contrasts directly before and after the food restriction period (to account for differences prior to the food restriction), 6%–7% reductions in length and condition could be attributed to the food restriction for immature males and females (Figure 3i,j). For maturing males, these effects were smaller than for immature individuals (about 3%–5%; Figure 3i,j). The between-treatment size difference in length at the end of the study did not reach the same level as observed prior to the food restriction for immature males and females, but a similar level of difference was reached in mature males (Figure 3i,j). For condition, in contrast, presumed compensatory growth of all individuals during the four weeks following the food restriction led to similar treatment and sex differences as exhibited prior to the food restriction (Figure 3i,j).

### 3.3 | The major locus (*vgll3*) associates positively with body condition but not growth

Using the same models to assess length and condition trajectories, we estimated additive and dominance *vgll3* effects ( $\alpha_{vgll3}$  and  $\delta_{vgll3}$ , respectively) for each trait and sex between August and December. There was little evidence for a *vgll3* association with length. Specifically, the *vgll3* genotypes did not show any distinct pattern for size ranking (Figure 4a,b). Accordingly,  $\alpha_{vgll3}$  estimates varied between positive and negative values, whereby all confidence intervals included zero (Figure 4e,f). Furthermore, the length deviations of the heterozygote genotype from additive expectations for length were, although consistently negative in the full food treatment, inconsistent between feeding treatments (Figure S2). Accordingly, also most  $\delta_{vgll3}$  estimates were negative in the full food and positive or near zero in the restricted food treatment, and all confidence intervals included zero (Figure S2).

In contrast, there was mixed evidence for *vgll3* association with condition. Presence and magnitude of the estimates varied across time, between feeding treatments, and between sexes (Figure 4c,d). Nonetheless, for maturation-unbiased females in both treatments (and males in the full food treatment),  $\alpha_{vgll3}$  estimates were relatively similar across time and approaching 1% (Figure 4g,h). Confidence intervals covered zero in some but not all cases. However, males in the

restricted food treatment yielded lower  $\alpha_{vgll3}$  estimates (approaching zero) both at the start of the measurement period and after the food restriction, which both followed periods of temporary food limitation (unintentional, see tank effects below, and intentional, respectively). Interestingly, the  $\alpha_{vgll3}$  estimates of males in the restricted food treatment increased after each temporary food-limitation period, that is, during compensatory growth phases. The  $vgll3^*E$  allele associated with higher condition in both males and females, implying a positive  $vgll3$  covariance between maturation and condition. In contrast, the length deviations of the heterozygote genotype from additive expectations were relatively low ( $< 0.5\%$ ) and highly inconsistent between feeding treatments, sexes, and across time (Figure S2). Accordingly,  $\delta_{vgll3}$  estimates for condition fluctuated around zero, and all confidence intervals included zero (Figure S2).

### 3.4 | Sex-specific variances for body length or condition, and between-sex and within-sex correlations between maturation and length or condition

Using trivariate models, we estimated environmental and polygenic effect variances for each sex-specific liability trait at each time point. These models also allowed for correlation estimates between sexes for each liability trait, and between male maturation and each sex-specific liability trait. Polygenic effect variance for both length and condition appeared present throughout for both sexes, was generally higher for males (Figure 5a,c), and polygenic effects (breeding values) were strongly correlated between sexes (Figure 5e,g). However, the genetic correlation for length between sexes decreased during the study period (Figure 5e). Notably, polygenic effects for male maturation correlated with those for length and condition of both sexes, but in a sex-specific manner. More specifically, the correlation between male maturation and female length decreased but remained positive throughout, whereas the correlation with male length decreased and turned from positive to negative, and so did the residual correlation for male effects (Figure 5e,f). In contrast, for condition the polygenic correlation between sexes and the correlations between male maturation and male or female condition remained high throughout the study (Figure 5g). Interestingly, the polygenic variance for male condition and the polygenic correlation between male maturation and male condition, both peaked around November (Figure 5c,g), which coincided with the phenotypic condition peak of mature males (Figure 3c,d), and the residuals followed this pattern for the variance and the correlation (Figure 5d,h). Resulting polygenic heritabilities for length across the study ranged from  $h^2_{poly} = 0.284$  (0.074–0.491) for females in August to 0.371 (0.179–0.560) for males in October. Polygenic heritabilities for condition ranged from  $h^2_{poly} = 0.211$  (0.081–0.363) for females in August (which was affected by considerable common environmental effects, see below) to 0.454 (0.218–0.666) for males in November.

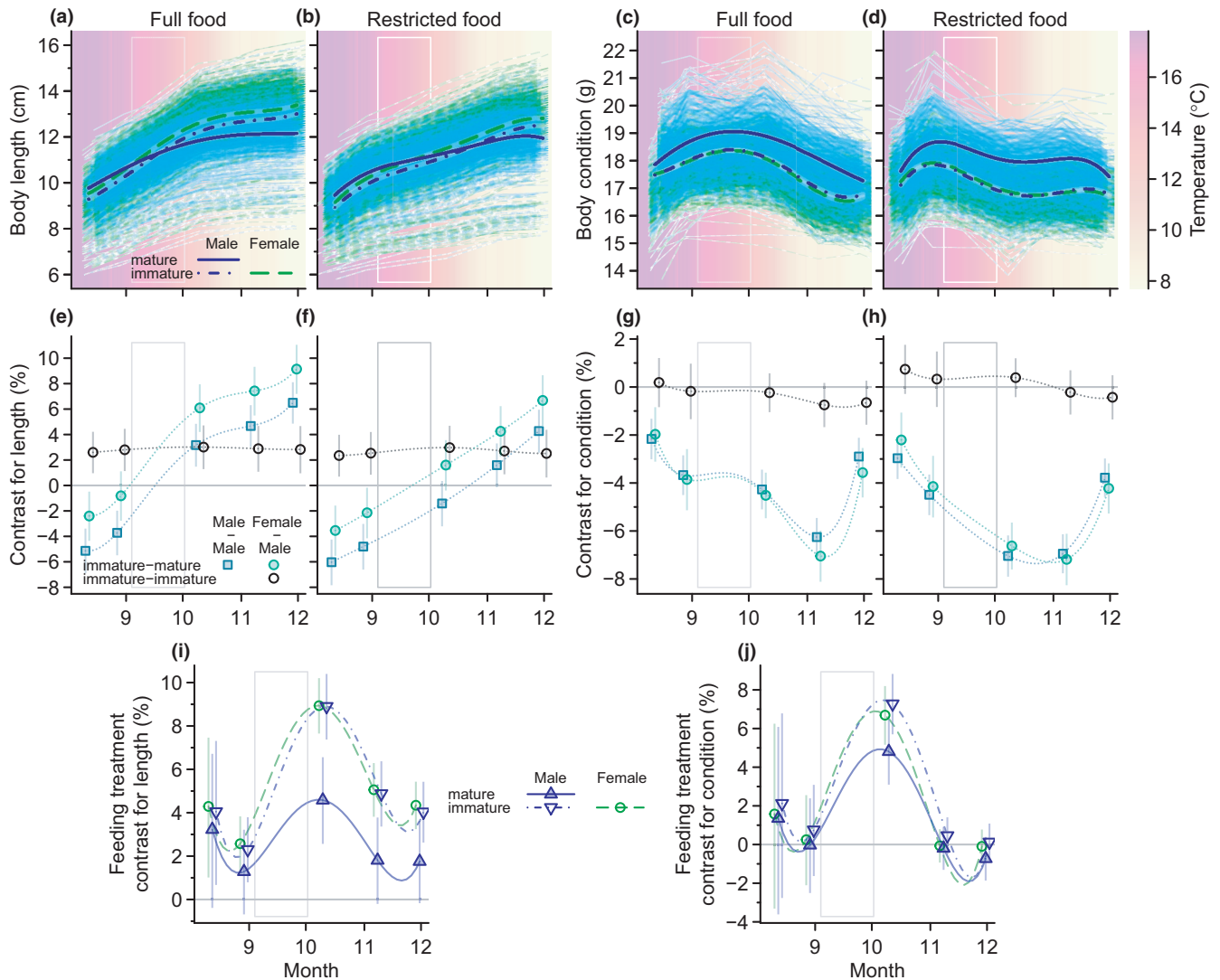
We also estimated common environmental (tank) and maternal (dam) effects. For length, we collected evidence for common

environmental effects mostly at the start of the study period in August (Figure S3a), which contributed 6% to the phenotypic variance ( $c^2_{male} = 0.06$ , 0.0–0.15;  $c^2_{female} = 0.06$ , 0.01–0.14) and were positively correlated between sexes (Figure S3e). However, common environmental variances and correlations between sexes for length approached zero otherwise. Across the entire period, common environmental correlation estimates between male or female length and male maturation fluctuated around zero with large credible intervals (Figure S3e). For condition, we estimated large common environmental effects at the start of the study (Figure S3c), which contributed around 31% to the phenotypic variance ( $c^2_{male} = 0.29$ , 0.11–0.52;  $c^2_{female} = 0.32$ , 0.12–0.16) and these effects were highly correlated between sexes (Figure S3g). The effects tapered off over the next two measurement periods with contributions to the total variance of 12% and 6%, and remained low (2%–3%) thereafter. With decreasing variance, the common environmental correlation estimates between sexes decreased while credible intervals increased (Figure S3g). As for length, common environmental correlation estimates between male maturation and male or female condition fluctuated around zero with large credible intervals (Figure S3g). Maternal effects for both length and condition appeared very low, exhibited large credible intervals covering zero (Figures S3b,d), and contributed ( $m^2$ ) 1%–4% to the phenotypic variance. The between sex correlation estimates were positive throughout but had large credible intervals (–0.61 to 0.99; Figures S3f,h). As for common environmental effects, maternal effect correlations between male or female and male maturation for both length and condition fluctuated around zero with large credible intervals (Figures S3f,h).

## 4 | DISCUSSION

### 4.1 | Control of maturation timing via liability traits

Mirroring the mechanistic assumptions for how maturation is initiated in many animal species (Dupont et al., 2014; Koyama et al., 2020; Parker & Cheung, 2020; Shalitin & Phillip, 2003), age- or season-specific size, growth rate, or available energy reserves (as reflected by condition) have all been suggested as liability traits determining the sexual maturation timing also in fishes (reviewed by Andersson et al., 2018; Good & Davidson, 2016; Taranger et al., 2010). However, environmental and genetic contributions to, and relative importance of, each liability trait to maturation remain largely unknown (Andersson et al., 2018; Good & Davidson, 2016; Mangel & Satterthwaite, 2008; Taranger et al., 2010) and several studies have questioned whether these traits specifically underly maturation timing variation (e.g., Gjerde, 1984; Skilbrei & Heino, 2011). Our study suggests that the causality of liability traits on maturation timing can be seasonally limited, and otherwise reversed to a causality of maturation timing on the presumed liability trait. These results stimulate critical assessments of previous, and rigorous design of future, studies on liability trait importance for binary traits. In contrast to previous studies that suggested a similar directional reversal of



**FIGURE 3** Recorded individual trajectories for length and condition of first-year Atlantic salmon ( $N = 2,608$ ) and the model-predicted average trajectories for combinations of sex and maturation status in two feeding treatments (a–d), the contrasts for the average trajectories among the combinations of sex and maturation (e–h), and the contrasts (full–restricted food) for the average trajectories of each of the combination of sex and maturation between the feeding treatments (i, j). The background colours in a–d indicate the average daily water temperature. The grey rectangles in all panels indicate the period of the temporary food restriction treatment. All estimates were back-transformed from the modelled log scale to the measured scale (averages) or percentage scale (contrasts). Symbols and error bars in e–j represent contrasts with approximate 95% confidence intervals at average measurement time points

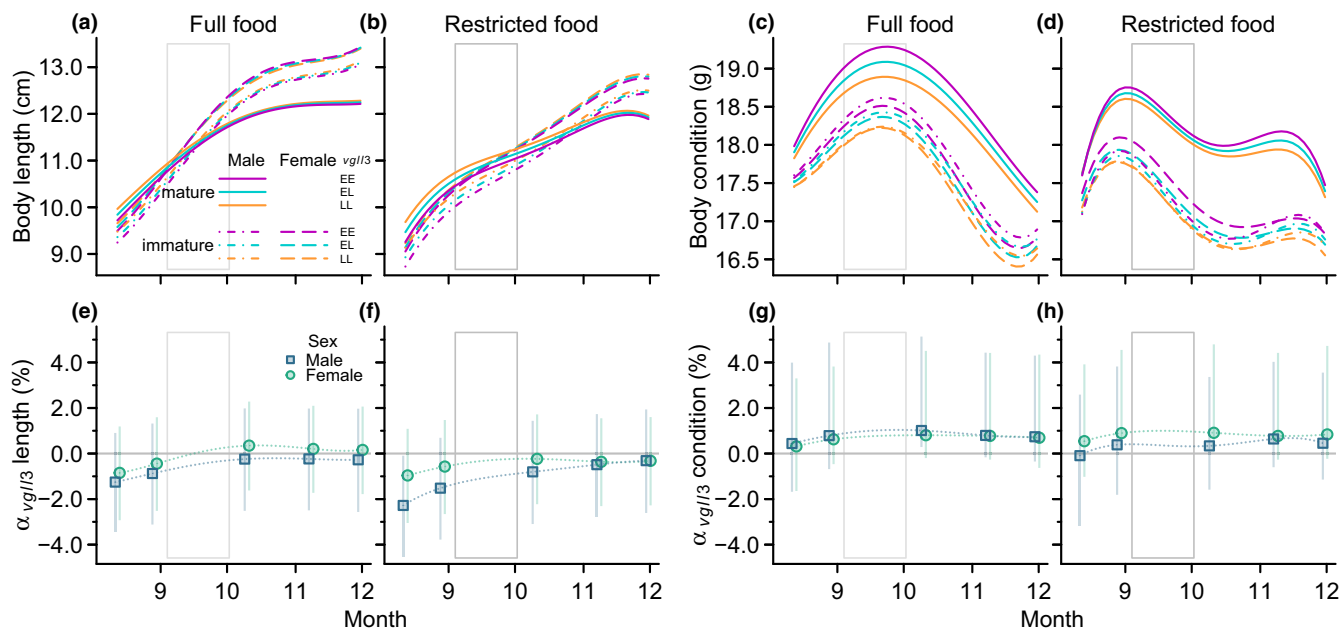
causality based on phenotypic relationships (e.g., Alm, 1959; Rowe & Thorpe, 1990), our study presents longitudinal expression patterns for the genetic relationships between maturation-affected males and maturation-unaffected females. Phenotypic relationships may be biased by reversed causality to different extents at initial (e.g., maturation-induced growth spurt) or later maturation stages (e.g., maturation-induced decline of somatic growth). The current study thereby allows for more confident support of initial causality direction and later causality reversal than studies on phenotypic or genetic relationships among only maturation-affected individuals. We also present results on how a major locus (*vgll3*) contributes to all of the aforementioned aspects. In light of the increasing number of studies identifying major loci affecting life-history traits, we thus

also provide a current empirical example of how a single locus relates to several key traits centring around maturation timing that exhibit complex caused-by and causal-on interactions.

## 4.2 | Association between growth and sexual maturation timing

The results of the current study support the general assumption that individuals with a larger body length during a sensitive period, representing higher prior growth rate, are more likely to initiate maturation (Andersson et al., 2018; Good & Davidson, 2016; Roff, 2002; Rowe & Thorpe, 1990; Stearns, 1992; Taranger et al., 2010; Wells

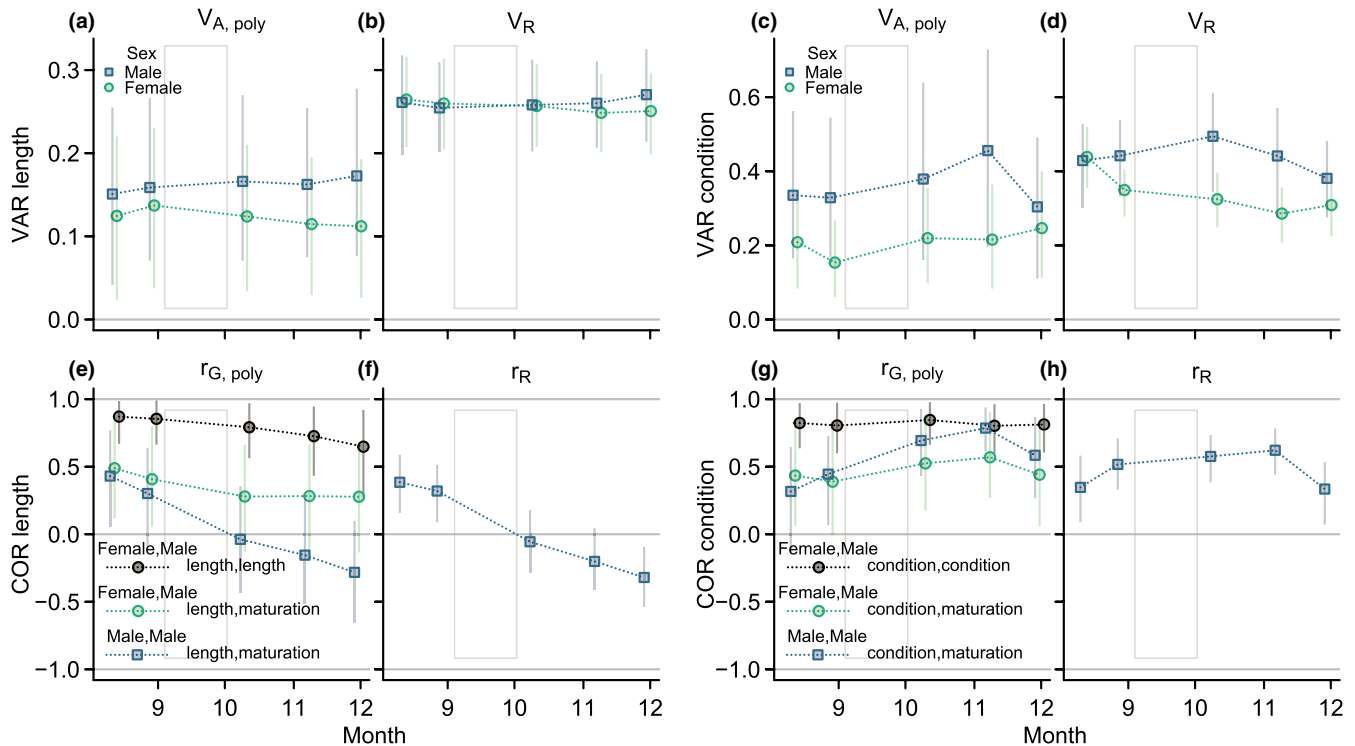




**FIGURE 4** Model-predicted average length and condition trajectories of major-locus (*vgll3*) genotypes under an additive genetic architecture in first-year Atlantic salmon ( $N = 2,608$ ) for combinations of sex and maturation status in two feeding treatments (a–d), and the corresponding additive *vgll3* effect estimates ( $\alpha_{vgll3}$ ; e–h). Additive effects have been accounted for maturation in males. The grey rectangles in all panels indicate the period of the temporary food restriction treatment. All estimates were back-transformed from the modelled log scale to the measured scale (averages) or percentage scale ( $\alpha_{vgll3}$ ). Symbols and error bars in e–h represent estimates with approximate 95% confidence intervals at average measurement time points

et al., 2017). This was suggested by the initial larger size of maturing males than immature males and females and by inferring that immature males were, with a consistent proportion across time, smaller than females (of which none matured) (Figure 3). This size pattern supports the hypothesis that the maturing males consisted of the initially more rapidly growing males, and the immature males of the initially slower growing individuals. This idea was supported by results about the variances and correlations in males and also between females and males (Figure 5); more specifically by initial positive environmental and genetic correlations between male length and maturation and by a similar positive genetic correlation between female length and male maturation. The latter genetic correlation reduces the possibility that maturing males grew initially only more rapidly as a result of an already initiated maturation process (e.g., by a puberty growth spurt) because female length was unaffected by maturation processes. Interestingly, the environmental and genetic correlations between male length and maturation, but not the genetic correlation between female length and male maturation, changed direction from positive in summer to negative towards winter. Concomitant with a decrease of the genetic correlation in males, the genetic variance for length in males increased relative to that of females and the genetic correlation between sexes decreased. Combined, these results support the presence of maturation-induced changes in gene expression that affected growth of maturing males, which increased the genetic variation for growth in males, and which effected a decreasing similarity in the gene sets underlying growth between sexes as a result. It appears very likely that the maturation process in males

altered the positive association of growth on maturation to become a negative association of maturation on growth that showed at both the environmental and the genetic levels. This inference is supported by observations that maturing males express slower growth towards reproduction, despite initially more rapid growth, presumably due to both re-allocation of energy from somatic to gonadal growth and a decreasing appetite (Alm, 1959). The finding that the genetic correlation between traits can change direction as a consequence of sexual maturation initiation, has implications for the applicability of genetic correlation estimates across studies and in selective breeding. The genetic correlation estimates can be expected to be—depending on extent of maturation-induced alterations—positive, neutral, or negative. We follow previous suggestions (Alm, 1959) and propose that the extensive phenotypic variation for the shape of the relationship between maturation probability and body size—positive, quadratic, or negative (e.g., Alm, 1959; Debes & Hutchings, 2014; Piché et al., 2008; Skilbrei & Heino, 2011)—may be explained by study differences reflecting a causal-on-maturation to caused-by-maturation continuum. Similar statements exist regarding inferences about fishery-induced evolution (Pauli & Heino, 2013). Comparable examples for conflicting inferences based on uncertain directions for maturation-timing-related causality also exist in humans (Bell et al., 2018; Prentice & Viner, 2012). We further propose that the positive genetic correlation between growth and maturation as exhibited prior to maturation-induced alterations of growth may be the most relevant to both natural and human multivariate selection. A direct relevance exists in aquaculture, where simultaneous selection for



**FIGURE 5** Time- and sex-specific variance estimates (VAR; a–d) for body length and condition, and correlation estimates (COR; e–h) between sexes for each trait and between each sex-specific trait and male maturation of first-year Atlantic salmon ( $N = 2,608$  for sex-specific length and condition and  $N = 1,280$  for male maturation). Variance and correlation estimates are shown for presumed polygenic effects ( $V_{A,poly}$  and  $r_{G,poly}$ ) and for environmental residual effects ( $V_R$  and  $r_R$ ). The grey rectangles in all panels indicate the period of temporary food restriction. Symbols and error bars represent mean estimates with 95% credible intervals at average measurement time points

late maturation and rapid growth is commonly practiced (Gjedrem & Baranski, 2005; Good & Davidson, 2016; Taranger et al., 2010).

There was little, if any, evidence in the current study that *vgll3* associates with body length (Figure 4). A previous study suggested that the *vgll3*\*E allele associates with smaller male spawner length within one of three studied salmon sea-age classes (Barson et al., 2015). However, a later study observed an opposite size trend where the *vgll3*\*E allele associated with larger mass within one age class (Fjelldal et al., 2020). These conflicting results in combination with results of the current study indicate uncertainty of the association between *vgll3* and length, and suggest that *vgll3* effects on size may be, if present at all, indirect or context dependent. It would not be surprising if *vgll3* exerts little or no effects on growth directly (i.e., prior to initiating maturation), because somatic growth is a highly multifactorial process (Enberg et al., 2012). A presumed absence of an association between *vgll3* and length is interesting. For example, a positive association for polygenic breeding values between length and maturation (genetic correlations up to 0.5 in the current study) may limit an effective simultaneous selection for late maturation and rapid growth. An absence of effects on somatic growth would make *vgll3* a very efficient means to select for later (earlier) maturation without compromising selection for larger (smaller) body size. In aquaculture, the expected selection response for later maturation may be considerable. Specifically, the generation interval could be

shortened by selecting for late maturation age in juveniles (via their *vgll3* genotype) and *vgll3* has a large effect size: *vgll3* accounted for up to one third of the total heritability for maturation timing of the age class studied here and for probably much larger proportions across several previously studied age classes (Ayllon et al., 2015; Barson et al., 2015).

### 4.3 | Association between body condition and sexual maturation timing

The results of the current study support the general assumption that individuals with higher body condition during a sensitive period, representing higher prior energy reserve acquisition or allocation rates, are more likely to initiate maturation (Andersson et al., 2018; Good & Davidson, 2016; Roff, 2002; Rowe et al., 1991; Stearns, 1992; Taranger et al., 2010; Wells et al., 2017). Reasons for this support were mostly similar to those reported for length above. The most compelling evidence emerges from the relatively high and positive genetic correlations between female condition and male maturation across the study period (Figure 5). In contrast, reverse causation appeared present in males, although no reversal of the direction of association between the traits was present as was present for growth. Instead, the association remained positive in maturing males and was

clearly increasing in strength relative to immature individuals during the study period with a peak around spawning time (November). Much of this increasing condition of maturing males may be due to increasing testes mass, and some increasing lipid reserves. The peak of the genetic correlation between male condition and male maturation coincided with that of genetic variance for male condition and with that of phenotypic differences between mature males and immature individuals, which supports the idea that maturation caused an increase in body condition and that this increased the genetic variance for male condition. As a result, we may assume that female breeding values represented the unknown maturation-unbiased male condition breeding values that contributed to the liability underlying maturation initiation (before and during an unknown sensitive period) more closely than the estimated maturation-biased male condition breeding values. However, the relatively high genetic correlation for condition between sexes remained, in contrast to that for length. This may not conflict with previous ideas if the maturation-induced increase in condition is proportional to that in the absence of maturation. In contrast, female phenotypic condition is unlikely to mirror the hypothetical maturation-unbiased male phenotypic condition. For example, lipid profiles of a maturation-unbiased experimental population at a similar age diverged between sexes, suggesting sex-specific lipid regulation (House et al., 2021). A strong biological link, nonetheless, exists in our study species between maturation timing and body condition in both sexes, which may be characterized as capital breeder, i.e. financing reproductive expenses using energy reserves (Jönsson, 1997).

Limiting inferences about condition, the study of body condition is much more challenging than that of growth. In the current study, body condition appeared to respond to environmental changes more rapidly and strongly than length. One example was provided by the much larger tank effects for condition than length, which we suspect to be caused by irregular feeding when transitioning from hand to automatic feeding. Another example is the shorter-lasting food restriction treatment effects for condition than length. More rapid responses to variation in food amount for condition than length or mass are well reported in fishes, which are suspected to serve, especially in salmonid fishes, in starvation avoidance during the freshwater phase (reviewed by Ali et al., 2003). However, body condition may, as a result, be a much more dynamic and less “memorising” parameter than body length. It may thus be more difficult to associate a phenotypic pattern of condition variation with maturation when it does not mirror the presumed condition that is causative on maturation initiation.

Some uncertainty exists whether higher body condition may underlie processes that are independent of somatic growth. Uncertainty is exemplified by for example, reviews on fish maturation timing by using phrases such as “high growth rates and/or high adiposity” (Andersson et al., 2018) or “high growth rate and/or lipid storage” (Taranger et al., 2010). Mechanisms have been identified in many animals that signal energy status and current metabolic challenges, control appetite and, eventually, control maturation timing (Dupont et al., 2014; Koyama et al., 2020; Parker & Cheung,

2020). Unfortunately, the same level of mechanistic understanding does not currently extend to fishes (Parker & Cheung, 2020). Nonetheless, our findings suggest it is plausible that maturation timing may be controlled via interactions between energy reserves and appetite-controlled growth, that is, that both traits interact but have some degree of independence. In contrast, Stearns and Koella (1986) and Thorpe (1986) suggested that the environmentally determined growth rate reflects overall energy acquisition rate and is an organismal predictor for resource availability. If condition variation indeed only underlies variation in somatic growth, condition would strongly covary with growth and may thus covary with maturation only indirectly. However, because the condition estimates presented here are accounted for by covariance with length, the detected positive genetic and possibly also environmental covariance between condition and maturation is unlikely to be explained via covariance with growth. Instead, our results indicate that not only somatic growth associates positively with maturation initiation, but also body condition, and that both have some degree of independence in their genetic control.

There was evidence that *vgll3* associates with body condition (Figure 4). This association showed a similar direction of association for females (and for males mostly) throughout the study duration. The *vgll3*\*E allele, which is associated with earlier maturation, is also associated with higher condition in both sexes. Known *Vgll3* functions suggest a mechanistic link with maturation via control of resource allocation between energy reserves and somatic growth. *Vgll3* inhibits adipocyte differentiation in favour of somatic growth processes (Halperin et al., 2013), which may lead to the expectation that *vgll3* would associate also with growth. Nonetheless, *vgll3* genotypes may differ by up to 2% in body mass at a given length, which we estimated across a considerable polygenic background of variation for condition. In comparison to the percentage of phenotypic variation explained by *vgll3* for maturation (7.7%), *vgll3* explained a smaller amount for condition at many time points (3%–8%) and all lower credible intervals were close to zero. However, male gonads at the investigated freshwater life stage constitute only 5–12% of body mass at spawning (Fleming, 1996; Rowe et al., 1991; Trombley et al., 2014). Therefore, the ~1% body mass increase that comes with the expected higher condition for each *vgll3*\*E allele may thus well be relevant for maturation because it adds markedly to the existing variation for body condition. These results indicate that *vgll3* effects on maturation may, at least partly, be mediated via *vgll3* effects on condition. The relatively weak signal may blur more easily for condition than maturation via larger contributions by environmental and polygenic effects to condition. In addition to underlying body condition variation may *vgll3* also *directly* be involved in gonadal development via inhibition (Kjaerner-Semb et al., 2018), whereby the different alleles associate with the expression variation of specific isoforms that may vary in their degree of maturation inhibition (Verta et al., 2020). Nonetheless, a final conclusion from our tentative results requires additional research, also because other plausible candidate genes close to *vgll3* have been identified, such as *akap11* (Ayllon et al., 2015; Barson et al., 2015; but see Sinclair-Waters et al., 2021).

#### 4.4 | Does one sensitive period exist to control age-specific maturation initiation?

An often-made assumption of many threshold characters is the presence of a sensitive period when the liability may control a binary character, but this has also been questioned for maturation initiation of fishes. Instead, maturation may initiate at fertilization with its *continuation* depending on environmental conditions thereafter (Sae-Lim et al., 2016; Thorpe, 2007; Thorpe et al., 1998). The consequence would be the absence of a typical sensitive period, further complicating the study of liability traits on maturation. For the current study, it appeared that the food restriction in September did not cause discontinuation of sexual maturation, despite the large detected effects on growth and condition. Also, maternal and common environmental effects appeared absent or very small for maturation, even though at least the latter were present for the liability traits at the start of the measurement period (which was closest to when we suspected a sensitive period). Furthermore, considerable maternal effects on length at first feeding have been reported in the study species (e.g., Debes et al., 2013), but these did not appear to affect the presumed maturation initiation in the current study. However, contributions by maternal effects may increase under specific environmental conditions (Páez & Dodson, 2017). For a more complete understanding of causality and the potential effects of liability traits on maturation, we propose a longitudinal multivariate approach covering the entire developmental period.

In summary, our study supports the hypotheses that (a) variation in growth and condition underlies variation for maturation initiation; and (b) that *vgll3* contributes to body condition, but hardly or not at all to growth. (c) However, once initiated, maturation affects variation in growth and condition, which highlights a challenge for studying phenotypic or genetic associations among these traits. We expect that growth, body condition, and maturation timing are traits that co-respond under selection for either trait via the polygenic correlations, and that body condition and maturation timing, but not growth, may co-respond to selection via the large-effect locus (*vgll3*) correlations.

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#### AUTHOR CONTRIBUTIONS

Conceptualization: Craig R. Primmer, Paul V. Debes; Data curation: Paul V. Debes, Tutku Aykanat; Formal analysis: Paul V. Debes; Funding acquisition: Craig R. Primmer; Investigation: Paul V. Debes, Nikolai Pivchenko, Annukka Ruokolainen, Outi Ovaskainen, Jacqueline E. Moustakas-Verho, Noora Parre, Craig R. Primmer; Methodology: Paul V. Debes, Craig R. Primmer; Project administration: Craig R. Primmer; Resources: Craig R. Primmer, Jaakko Erkinaro; Supervision: Craig R. Primmer, Paul V. Debes; Visualization: Paul V. Debes; Writing – original draft: Paul V. Debes, Craig R. Primmer; Writing – review & editing current version: Paul V. Debes, Craig R. Primmer, Jacqueline E. Moustakas-Verho; Writing – review & editing previous versions: Paul V. Debes, Craig R. Primmer, Jacqueline E. Moustakas-Verho, Tutku Aykanat, Jaakko Erkinaro.

#### DATA AVAILABILITY STATEMENT

The data have been made publicly available at the Dryad Digital Repository <https://doi.org/10.5061/dryad.jh9w0vt6k>.

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## SUPPORTING INFORMATION

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