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Genetic variation for upper thermal tolerance diminishes within and between populations with increasing acclimation temperature in Atlantic salmon

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Populations may counteract lasting temperature changes or recurrent extremes through plasticity or adaptation. However, it remains underexplored how outbreeding, either naturally, unintentionally, or facilitated, may modify a local response potential and whether genotype-by-environment interactions or between-trait correlations can restrict this potential. We quantified population differences and outbreeding effects, within-population genetic variation, and plasticity of these, for thermal performance proxy traits using 32 pedigreed wild, domesticated, and wild-domesticated Atlantic salmon families reared under common-garden conditions. Following exposure to ambient cold (11.6 °C) or ~4° and ~8° warmer summer temperatures, populations differed notably for body length and critical thermal maximum (CT_{max}) and for thermal plasticity of length, condition, and CT_{max}, but not for haematocrit. Line-cross analysis suggested mostly additive and some dominant outbreeding effects on means and solely additive outbreeding effects on plasticity. Heritability was detected for all traits. However, with increasing acclimation temperature, differences in CT_{max} between populations and CT_{max} heritability diminished, and CT_{max} breeding values re-ranked. Furthermore, CT_{max} and body size were negatively correlated at the genetic and phenotypic levels, and there was indirect evidence for a positive correlation between growth potential and thermal performance breadth for growth. Thus, population differences (including those between wild and domesticated populations) in thermal performance and plasticity may present a genetic resource in addition to the within-population genetic variance to facilitate, or impede, thermal adaptation. However, unfavourable genotype-by-environment interactions and negative between-trait correlations may generally hamper joint evolution in response to an increase in average temperature and temporary extremes.

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INTRODUCTION

The increase of both average and temporary extremes of environmental temperature are outcomes of climate change (IPCC 2014). When the environmental temperature exceeds population-specific limits for thermal plasticity, poikilothermic organisms have to emigrate or adapt to avoid extirpation. However, it remains uncertain whether adaptive potentials are sufficient to counteract environmental changes, and, whether gene flow between populations may improve or worsen adaptive or plastic potentials (Hoffmann and Sgrò 2011; Huey et al. 2012; Merilä and Hendry 2014; Catullo et al. 2019). This uncertainty is not surprising because studying evolutionary potential across changing environments that exert changing selection regimes involves several challenges. Individual phenotypes, the target of selection, underlie both genetic and non-genetic effects. Only the additive genetic proportion of the phenotypic variation, the heritability (h^2), reaches the next generation and allows for evolution (Lynch and Walsh 1998). However, across environmental conditions, the phenotype can vary via phenotypic plasticity (Bradshaw 1965), and the heritability can vary via differential gene expression (Hoffmann and Merilä 1999; Charmantier and Garant 2005). Thus, populations inhabiting changing environments may not only experience changing natural selection regimes and phenotypes but also changing environmental and genetic contributions to the phenotype, altogether complicating the study of evolution under climate change.

As a further complication in evolutionary research, selection rarely affects single traits because of phenotypic correlations among traits (Lande and Arnold 1983). The resulting multivariate evolution is often constrained, or facilitated, by genetic correlations (Walsh and Blows 2009) leading to dynamic links between different traits or the same trait in different environments (Morrissey et al. 2010). As a result, evolution can be constrained (e.g., Etterson and Shaw 2001) or accelerated (Falconer 1952; but see Agrawal and Stinchcombe 2009). Thus, understanding the evolutionary potential of thermal performance and extreme tolerance inevitably requires understanding how much genetic and non-genetic effects contribute to these traits as expressed in different thermal regimes. And furthermore, how these contributions are correlated between regimes, and how thermal performance and extreme tolerance traits are correlated (Kingsolver et al. 2015; Ørsted et al. 2019; Morgan et al. 2020).

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A mechanism that may be important for thermal plasticity and adaption is the influence of gene flow between populations (population outbreeding) (Hoffmann and Sgrò 2011; Moritz et al. 2012; Catullo et al. 2019). In the presence of population outbreeding, populations may reach adaptive optima faster, or optima that they cannot reach in its absence (Wright 1932). Under climate change, gene flow from warmer to colder adapted populations may thus aid in the thermal adaptation of the latter population (Aitken and Whitlock 2013; Bontrager and Angert 2019; Catullo et al. 2019). However, population outbreeding may also dilute local adaptation (Lenormand 2002; Edmands 2007). As a result, predictions about species responses to climate change might fail if outbreeding effects are neglected.

Small water bodies are especially affected by increasing temperature and thermal extremes because their temperature often co-varies with air temperature and solar radiation (Thompson et al. 2013). Within such habitats, life stages that are unable to escape thermal extremes may limit the thermal ranges of many poikilothermic species. Such a thermally fragile life stage occurs in the Atlantic salmon (*Salmo salar*), an anadromous salmonid that appears to not behaviourally avoid high temperatures during its first year of life in freshwater (Breau et al. 2007). Furthermore, the globally expanding aquaculture industry poses additional challenges to the adaptive potential of wild populations. Domesticated escapees, which have been selectively bred for many traits, including rapid growth, can successfully outbreed with wild individuals. This results in wild-domesticated hybrids that exhibit largely intermediate traits including growth (reviewed by Glover et al. 2017). However, an elevated growth potential may trade-off with a decreased thermal performance breadth (Huey and Kingsolver 1989; Kingsolver et al. 2004; Kingsolver et al. 2015), which may then compromise the degree of the evolutionary potential of admixed populations under climate change. Thus, comparing wild vs. domesticated salmon provides an excellent opportunity to assess the relevance of gene-flow between genetically differentiated populations on thermal performance (e.g., Solberg et al. 2016), thermal extreme tolerance, and plasticity of both and to understand the general dynamic link between temperature-specific performance and thermal extreme tolerance.

Here, we conducted a common-garden experiment using first-year wild, domesticated, and wild-domesticated Atlantic salmon and quantified genetic and environmental effects for thermal performance and extreme tolerance proxy traits under cold, optimal, and warm summer temperature regimes. Specifically, we quantified for each trait the population means within, and plasticity between, temperature regimes and assessed how means and plasticity change by population outbreeding, which also revealed the underlying genetic trait architecture. Furthermore, we quantified genetic, maternal, and environmental variation within populations and genetic correlations across regimes. Lastly, we explored whether traits exhibit genetic and phenotypic correlations among them. Due to uncertainty about traits relevant to thermal adaptation under climate change (Clark et al. 2013; Sinclair et al. 2016; Kingsolver and Buckley 2017), we chose four traits that are candidates for adaptation to climate change. These traits approximate either whole-organism performance (body length; body condition) or thermal extreme performance (critical maximum temperature, CT_{max}; haematocrit, hct). Our study, altogether, allows for a multivariate evaluation of the evolutionary potential of outbred populations to climate change.

MATERIALS AND METHODS

Breeding and experimental design

To minimize the confounding of environmental with genetic effects and equalize acclimation (Kellermann et al. 2017), we crossed, raised, and phenotyped all experimental fish under standardized conditions (i.e., a common-garden experiment). On 22 November 2016, we generated 32 salmon families: eight wild (Etne River, Norway) and eight domesticated (Mowi, Norway) full-sib families, and 16 first-generation hybrid families at the Matre Research Station, Norway (Fig. 1A). The breeding design, crossing every parent both within and between populations, allows for estimating the between-population genetic architecture while controlling for parental effects. It further allows for relatively crude quantifications of within-population variance based on additive genetic effects (averaged across populations and assuming a negligible segregation variance, which may hold in many cases; Muff et al. 2019) and maternal effects. Fin clips from broodfish and offspring enabled, via established DNA protocols (12 or 17, if required, microsatellite loci; Solberg et al. 2016), both the reassignment of offspring to their parents using the programme FAP 3.6 (Taggart 2006) and the reconstruction of grandparents using the programme Colony 2.0.5.0 (Jones and Wang 2010). The fertilized eggs were incubated in family batches under standard hatchery conditions until reaching the eyed-egg stage. Thereafter, on 2 February 2017, all families were mixed equally into three prospective temperature regimes; each randomized to three round-tank replicates (totalling nine tanks; each holding 0.4 m^3) and each tank contained 20 fish of each family (totalling 5760 fish).

On 18 July 2017, 56 days after the first feeding, we initiated three seasonally varying water-temperature regimes with anticipated 4 °C steps between them to mimic a range of present and potential future conditions that lasted 2 months (Fig. 1B). The "cold" (mean, 95% confidence interval: 11.6, 10.4–13.5 °C), reflects many contemporary Norwegian rivers (Solberg et al. 2016) and is below the optimum *S. salar* growth temperature. The "optimum" (15.6, 14.5–17.3 °C), represents the temperature that maximizes

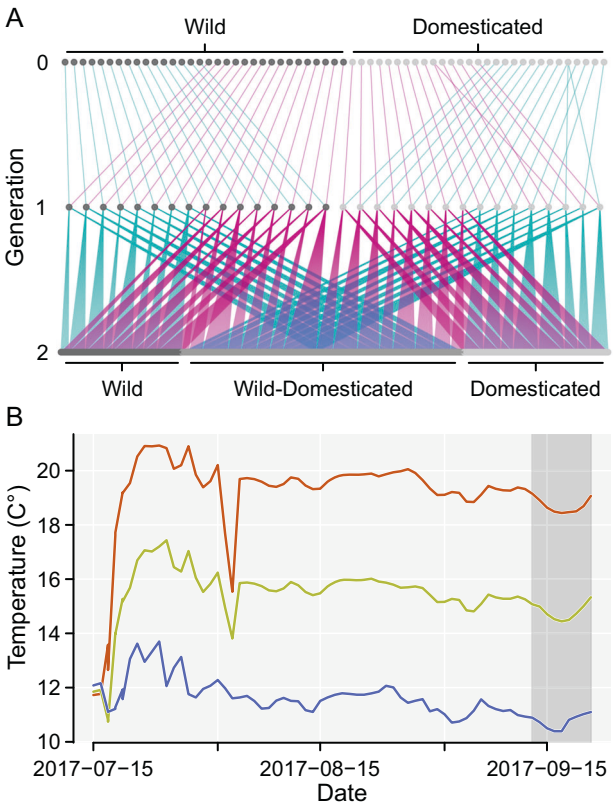

Fig. 1 Pedigree depicting breeding design and line plot showing experimental temperatures. The pedigree (**A**) is limited to experimental individuals with data on all four investigated traits ($n = 538$; 32 families in generation 2) and shows relationship ties via their reassigned parents (generation 1) up to their reconstructed grandparents (generation 0). Offspring are connected to their mother by turquoise lines and to their father by violet lines. The line plot (**B**) shows the average daily water temperatures for each thermal regime, which are differentiated by blue (ambient; cold), yellow (optimum), and red lines (warm). The phenotyping period is indicated by the grey vertical bar.

S. salar growth (Elliott and Elliott 2010). The "warm" (19.5, 18.2–20.9 °C) represents conditions above optimal growth for many salmon populations (Jonsson et al. 2001). Fish were constantly fed *ad libitum* (ensured by periodically adjusting feed-producer guidelines for mass- and temperature-specific feed rations to result in some excess feed at tank bottoms) with mixed pellet sizes to accommodate all fish sizes (Nutra XP, Skretting, Norway). Oxygen was kept automatically >70% saturation by adding O_2, and the temperature was adjusted daily for the warmer regimes based on the average previous-day cold regime temperature. Artificial lighting followed the regional natural cycle. Between 13 and 21 September 2017, we quantified (i) somatic growth by body length (length) and (ii) lipid reserves by body condition (condition), (iii) thermal extreme tolerance by critical maximum temperature (CT_{max}), and (iv) a component of oxygen transport capacity by haematocrit (hct). Because we wanted to link CT_{max} and hct, whereby determining the latter is lethal, hct was determined after CT_{max} trials. Whole-organism performance traits, such as growth and condition, directly link to climate-change evolution via several selective forces (Pörtner and Farrell 2008; Franks and Hoffmann 2012; Sinclair et al. 2016). For example, salmon condition, which reflects surplus energy (Sutton et al. 2000), and growth both associate with maturation and migration schedules and overwintering survival (Thorpe 1994; Taranger et al. 2010). CT_{max} may be relevant under thermal extremes (Lutterschmidt and Hutchison 1997; Pörtner and Peck 2010). Hct, the proportion of red blood cells in the blood, may relate to oxygen transport capacity (Gallaugher and Farrell 1998), but its importance on thermal tolerance is under current debate (Pörtner et al. 2017; Jutfelt et al. 2018). Animal experimentation followed the Norwegian Animal Welfare Act under license 12109 by the Norwegian Food Safety Authority.

Phenotyping and CT_{max} trials

We conducted 18 CT_{max} trials, six per regime and two per tank. To not confound tank or regime effects with temporal or diurnal effects, respectively, we conducted trials in random order, but on different days and at different hours of the day per tank. Due to our split-plot design (regime-specific tanks containing all families), all families were affected equally by potential tank and trial effects. For each trial, we randomly sampled ~30 fish (27–31) that had been fasted for 24 h, and let them get accustomed for 12–20 h (overnight) to a trial tank. Random sampling resulted in an average of 11 (range 3–23) offspring from each of the 32 parents per regime (see also Table S1). The trial tanks and their initial and overnight accustom-period temperatures mirrored holding tanks but had lower water levels (40 cm), and received flow-through water and diffuser aeration. During the trial, we followed the established Atlantic salmon-specific protocol of Anttila et al. (2013) and heated from the regime-specific temperature at a rate of 0.3 °C min^{-1} until 22 °C, and at 0.1 °C min^{-1} thereafter. The trial-tank-water temperature was recorded in real-time in 5 s intervals with a resolution of 0.001 °C and an accuracy of 0.01 °C using a CTD probe (SD204, SAIV A/S, Bergen, Norway). The probe was connected to a computer running a custom-made R-script to visualise real-time temperatures and heating rates. The anticipated tank heating rate was ensured by an automatically heated (Normatic Web Server 3.3) flow-through water source and manually fine-tuning by varying hot water inflow from an in-house designed water boiling tank (400 L, 9 kW). Oxygen across trials ranged between 8.0 and 8.8 mg L^{-1} (87–95% saturation) during CT_{max} recordings and was thus not environmentally limited.

During trials, we sampled fish that had permanently lost the righting response, recorded the corresponding CT_{max}, and euthanized the fish (using metacain). To quantify individual hct, we took blood sample duplicates with ammonium-heparised 18 µL glass capillaries (Hirschmann Laborgeräte, Eberstadt, Germany) after tail ablation. We centrifuged the capillaries for 4 min at 12.2 g (Heraeus Pico 17, Thermo Fisher Scientific, Waltham, Massachusetts, USA) and determined hct using manufacturer-provided tables. Using duplicates, we estimated measurement precision as the ratio of the among-individual to the total variance by a mixed model with fish identification as a random term and fixed effects as reported below for the hct model. Measurement precision for hct was 0.83 ± 0.02 (mean ± standard error). For subsequent analyses, we used the average hct for each individual. We measured fork length (±1 mm) and wet mass (±0.1 g), fin-clipped each trial fish ($n = 538$) and also a subset of fish that was not trialled for CT_{max} (starting when having transferred fish for the last trial for a given tank, sampling every second fish in random order of the cold and warm regimes and all fish of the optimum regime; $n = 3938$). We estimated fish condition as residuals of the regression of LN of mass on LN of length (see below; Sutton et al. 2000).

Data analyses

To jointly estimate mean and plasticity effects and (co)variances for each trait, we fitted animal models, which estimate additive genetic variance (V_A) via the inverse of the additive genetic relationship matrix (Henderson 1950; Henderson 1973). Each model contained fixed effects for Regime, Population, and Population-by-Regime terms, some models contained additional continuous covariate terms (and potential interactions; Covar, mean centred, see below). No differences were observed between the reciprocal hybrids for any trait and we thus fitted a common effect. Because unaccounted length heterogeneity among populations may mimic population effects, we tested for (and accounted for, if relevant) covariance between length and CT_{max} or hct. Further, for CT_{max} we tested for diurnal effects (trial hour), which affect CT_{max} in many species (Lutterschmidt and Hutchison 1997). For hct, we also tested for seasonal effects (trial date) and whether these differed among populations because we observed such temporal trends during data exploration. As random effects, we initially included additive genetic (*Animal*; estimating V_A), maternal composite (*Female*; estimating V_M), common environmental (*Tank* and *Tank:Trial*; estimating V_C), and residual environmental effects (estimating V_R, heteroscedastic for regimes). By design, the nine tank effects were nested within regime effects and the 18 trial effects within tank effects. The tank-by-trial term was only fitted for CT_{max} and hct. Models followed the general equation:

$$y \sim 1 + Regime + Population + Regime - by - Population + Covar + Animal + Female + Tank/Trial + Residual \quad (1)$$

To test whether genetic or maternal effects vary with the thermal regime, observed as variance heterogeneity or effect re-rankings, we compared models representing a series of nested covariance structures (Table S2). To achieve a condition response based on LN-transformed body mass, we added LN-transformed length (mean centred) as a covariate to the model in (1). This results in an analysis of length-standardized mass and provides estimates for body conditions that correlate with lipid content in first-year Atlantic salmon (Sutton et al. 2000).

Because CT_{max} and hct exhibited phenotypic covariance with body length (see "Results"), we also estimated genetic, environmental (residual), and phenotypic correlations among these traits. To do so, we fitted bivariate models (a multivariate model did not converge) that were similar to univariate models (but omitted the length covariate when fitting length as a response) and allowed for genetic between-trait covariances within and between regimes, and for residual between-trait covariances within regimes.

We estimated the between-population genetic architecture by line-cross analysis on trait means (Mather and Jinks 1982). For two populations and their first-generation hybrids, additive (α) and dominant (δ) outbreeding effects can be estimated. To minimize estimation bias and fully account for the experimental design and all relatedness (Komender and Hoeschele 1989), we implemented the line-cross analysis to the univariate animal models (1) but replaced the Population term (also in the Population-by-Regime term) by coefficients for α and δ (in the order W, $W \times F$, F; α: −1, 0, 1; δ: 0, 1, 0). We only fitted δ if a sequential Wald's test for a population lack-of-fit term was significant. The α-by-Regime level contrasts represent the expected change of thermal plasticity by first-generation population outbreeding, which we refer to as α_p.

Model fitting proceeded with residual maximum likelihood using ASReml-R v. 3 (Butler et al. 2009) under backwards model selection for nested covariance structures using likelihood ratio test (LRTs). We tested fixed terms thereafter using Wald's F-tests with denominator degrees of freedom approximated after Kenward and Roger (1997). We regarded fixed terms and covariances with $P < 0.05$ and positively constrained variances with $P < 0.10$ as significant. For pairwise comparisons, we used t-tests with (denominator) degrees of freedom approximated by the abovementioned F-tests and adjusted P-values using the false discovery rate (FDR) following Benjamini and Hochberg (1995). We approximated correlation and heritability standard errors using the delta method (Lynch and Walsh 1998) and 95% confidence intervals as means ± 2 standard errors.

We defined population-level thermal plasticity as the population-specific contrast between regime-pair means relative to the warmer regime. This approach yields unbiased plasticity estimates, regardless of the shape of the underlying reaction norm (Morrissey and Liefting 2016). We defined differences in thermal plasticity between populations as thermal plasticity population contrasts, which are provided by Population-by-Regime term-level contrasts. We defined within-population thermal plasticity as the

Table 1. ANOVA table for the fixed effects in the univariate animal models to test for population (Pop: wild, wild-domesticated, domesticated), temperature regime (Reg: cold, optimum, warm), population-by-regime effects, and additional covariates (hour of day, date, body length), for responses of either length, condition, CT_{max}, or hct (left columns) or to test for the corresponding genetic architecture between populations (right columns; a = additive; δ = dominant, a_p = additive plasticity).

Term	DF, DDF	*F*	*P*	Term	DF, DDF	*F*	*P*	[a]Lack of fit
Length								
Pop	2, 8.3	133.5	<0.001	a	1, 8.0	500.6	<0.001	$\chi^2_1 = 81.4$ $P < 0.001$
				δ	1, 3701	81.3	<0.001	
Reg	2, 9.9	135.9	<0.001	Reg	2, 11.0	127.3	<0.001	
Pop:Reg	4, 74.8	4.8	0.0017	a_p	2, 18.9	5.9	0.010	$\chi^2_2 = 2.24$ $P = 0.327$
Condition								
Length	1, 3816.9	102,600	<0.001	Length	1, 3817.0	102,600	<0.001	
Pop	2, 67.1	2.9	0.056	a	1, 25.7	1.1	0.305	$\chi^2_2 = 5.0$ $P = 0.029$
				δ	1, 3776.2	4.6	0.031	
Reg	2, 8.1	140.5	<0.001	Reg	2, 8.1	140.8	<0.001	
Pop:Reg	4, 120.1	2.5	0.049	a_p	2, 41.1	4.2	0.021	$\chi^2_2 = 1.0$ $P = 0.490$
CT_{max}								
Hour	1, 14.6	13.2	0.003	Hour	1, 25.8	13.3	0.002	
Length	1, 279.6	17.1	<0.001	Length	1, 17.9	17.9	<0.001	
Pop	2, 32.9	1.4	0.247	a	1, 40.7	2.7	0.013	$\chi^2_1 = 0.2$; $P = 0.667$
Reg	2, 20.7	1023	<0.001	Reg	2, 20.7	1025	<0.001	
Pop:Reg	4, 73.5	3.3	0.015	a_p	2, 54.3	6.6	0.004	$\chi^2_2 = 0.0$; $P = 0.274$
Hct								
Date	1, 13.9	29.4	<0.001	Date	1, 14.1	28.2	<0.001	
Length	1, 402.0	0.7	0.391	Length	1, 415.2	1.2	0.284	
Pop	2, 75.0	1.1	0.349	a	1, 31.6	0.9	0.360	$\chi^2_1 = 1.2$ $P = 0.540$
Reg	2, 17.6	165.3	<0.001	Reg	2, 17.6	162.6	<0.001	
Pop:Reg	4, 196.2	2.9	0.024	a_p	2, 104.0	5.2	0.007	$\chi^2_2 = 7.9$ $P = 0.096$
Date:Pop	2, 467.7	8.8	<0.001	Date:α	1, 474.1	17.9	<0.001	$\chi^2_2 = 1.4$ $P = 0.503$
Length:Reg	2, 178.1	4.9	0.009	Length:Reg	2, 184.0	4.6	0.011	

[a]For a model without δ; significance indicates that δ may improve the model fit to the genotype means

compound effect of additive genetic variance heterogeneity and genetic correlations between regimes (Falconer 1952).

RESULTS

Population means, between-population genetic effects, and their thermal plasticity

We first tested covariates for CT_{max} and hct. For CT_{max}, we identified the experimental variable of trial hour and the biological variable of fish length as phenotypic predictors (Table 1, Figs. S1 and S2). A one-hour delay in trial incurred a CT_{max} increase of 0.027 ± 0.008 °C (mean ± standard error) and a one-cm length increase associated with a CT_{max} decrease of 0.024 ± 0.006 °C. For hct, we identified the experimental variable of trial date, which varied in effect among populations, and the biological variable of fish length as phenotypic predictors (Table 1, Figs. S3 and S4). However, the latter varied only significantly for fish exposed to the cold regime. A one-day delay in trial incurred an increase for hct of 2.38 ± 0.37% for wild, 1.15 ± 0.27% for hybrid, and 0.51 ± 0.35% for domesticated salmon. A one-cm increase in individual length associated with a hct increase of 0.58 ± 0.20% in the cold, a decrease of −0.23 ± 0.21% in the optimum, and a trend estimate close to zero of 0.01 ± 0.13% in the warm regime.

Controlling for covariates as reported above and using *F*-tests, we detected genotype-by-environment interactions, i.e., we estimated that population differences depend on acclimation temperature, for all four traits (Table 1). However, controlled for the false discovery rate, genotype-by-environment interactions only remained significant under a 5% *P*-value threshold for length and CT_{max} (Fig. 2). Specifically, the average length was estimated to be 26.1 ± 2.1, 23.6 ± 1.6, and 28.4 ± 2.4% lower for wild than domesticated salmon in the cold, optimum, and warm regime, respectively (Fig. 2A). Furthermore, length-controlled CT_{max} was estimated to be 0.31 ± 0.01 and 0.23 ± 0.01 °C higher for wild than domesticated salmon when trialled for fish from the cold and optimum regime, respectively, but negligible population differences of 0.01 ± 0.05 °C for CT_{max} were estimated in the warm regime (Fig. 2C). Thus, CT_{max} differences between populations decreased with acclimation temperature and appeared absent at the highest temperature. For body condition or hct, we did not estimate population differences that were significant under a 5% *P*-value threshold in any temperature regime. However, condition estimates were consistently higher with increasing allelic contribution of the domesticated population across temperature regimes (Fig. 2B). Following the strongest differences detected between wild and domesticated salmon, hybrids were different from both parental populations in all regimes for length, and in the cold and optimum regimes for CT_{max} (Fig. 2).

We also assessed for each trait in each temperature regime the underlying genetic architecture between populations (additive vs. additive and dominant) and estimated the associated effects. The additive effect (a) is here defined as the expected change in the mean of first-generation hybrid relative to wild salmon based on an additive genetic architecture between populations.

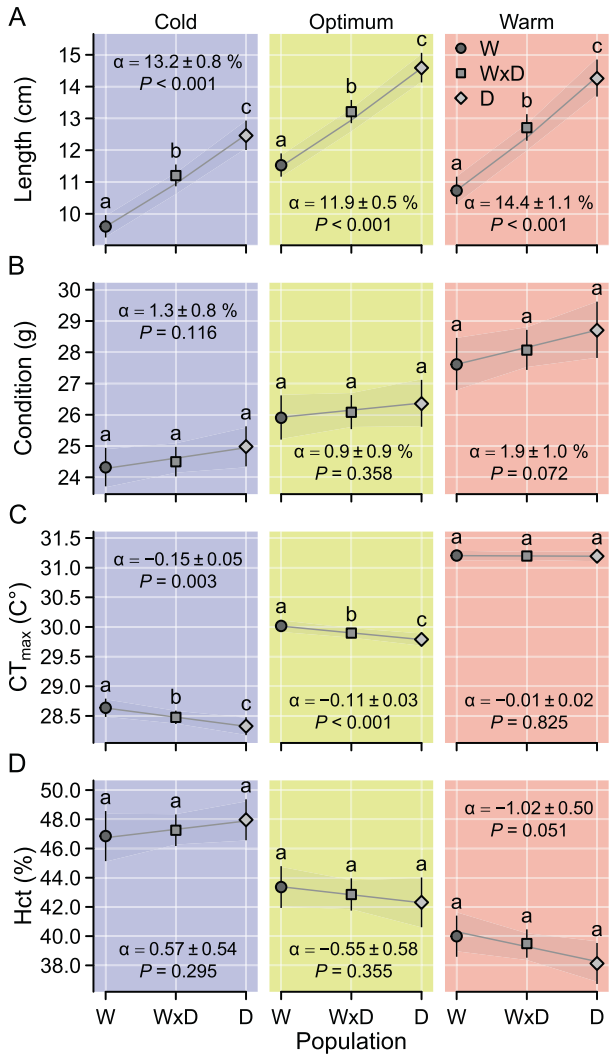

Fig. 2 Mean estimates and associated temperature-regime-specific additive outbreeding effect estimates among wild (*W*), reciprocal wild-domesticated (*W* × *D*), and domesticated (*D*) Atlantic salmon from three temperature regimes. Means are represented by population-specific symbols with 95% confidence intervals, whereas additive outbreeding effects are shown as grey lines with transparent 95% confidence bands (also given as: $\alpha \pm$ standard error; *F*-test-based *P*-value). Estimates are for three temperature regimes (cold, optimum, warm) and for the four traits of length (**A**; $n = 3932$), condition (**B**; expected mass of a geometric-mean-sized fish of 12.1 cm; $n = 3932$), CT_{max} (**C**; $n = 538$), and hct (**D**; $n = 538$). The same letters above means indicate within-regime comparisons with no support for differences at FDR ≤ 0.05.

The dominant effect is here defined as the deviation of the wild-domesticated outbred trait mean from the additive expectation. We observed the greatest statistical support for additive outbreeding effects for length in all temperature regimes, and for CT_{max} in the cold and optimum regime (but not in the warm regime) (Fig. 2). Population outbreeding led to hybrids that appeared intermediate to both parental populations for CT_{max} and hct, but not for length and condition (Fig. 2). Specifically for length, we estimated a small dominant effect towards domesticated salmon with a magnitude of about one-hundredth of the additive effects ($\delta = 0.144 \pm 0.016\%$). For condition, we estimated a dominant effect towards wild salmon that was about one-quarter of the additive effects ($\delta = -0.33 \pm 0.15\%$). All additive-by-regime effects sufficiently fit the population-by-regime means, which indicated undetectable or absent dominant-by-regime effects (Table 1).

Using the same models to estimate means, we also quantified thermal plasticity, i.e., the change in trait mean between temperature regime pairs with a 4 °C difference. We estimated that most traits exhibited strong thermal plasticity (Fig. 3; *F*-test in Table 1). Across populations, length increased from cold to optimum and declined from optimum to warm, but less for domesticated than wild salmon. This indicated a higher optimal performance temperature, or wider performance breadth, of the domesticated population. Condition and CT_{max} both increased from cold to warm, whereas hct decreased from cold to warm (Fig. 3).

When we compared thermal plasticity between the cold and the optimum regimes between populations, the contrasts for all traits included zero within their 95% confidence intervals, i.e., we were unable to detect significant differences for plasticity between wild and domesticated salmon, although relatively consistent intermediate means of the hybrid salmon indicated that larger sample sizes may lead to stronger statistical support (Fig. 3). Nonetheless, between the optimum and the warm regimes, we estimated statistically significant population differences for three of the four traits. Specifically for wild relative to domesticated salmon, we estimated a larger growth reduction, a smaller condition increase, and a smaller CT_{max} increase (Fig. 3A–C). The plasticity of hybrid salmon fell largely in between that of wild and domesticated salmon and was statistically different for thermal plasticity between the optimum and warm regimes for length and CT_{max} of wild but not domesticated salmon (Fig. 3A, C). It should be noticed that both the relatively larger (negative) thermal plasticity for length and the relatively smaller (positive) thermal plasticity for the condition of wild salmon relative to hybrid and domesticated salmon suggest a relatively lower growth performance of wild salmon under a warm temperature. Furthermore, despite a higher thermal plasticity for CT_{max} in hybrid and domesticated salmon, both showed very similar absolute CT_{max} values like wild salmon in the warm environment. Put differently, a higher domesticated-origin thermal plasticity for CT_{max} mitigated the lower domesticated salmon performance as expressed at colder temperatures and led to a similar performance among populations at the highest temperature (Figs. 2C and 3C).

We also quantified the additive genetic between-population effects for thermal plasticity (a_p) for three of the four investigated traits (Fig. 3; Table 1). Specifically, we estimated values for a_p that were different from zero between the optimum and the warm regimes for length, condition, and CT_{max}. We did not detect dominant effects on thermal plasticity (Table 1).

Within-population maternal, common environmental, and genetic effects, and their thermal plasticity

Using the same models, we had also quantified—although with lower precision and accuracy—maternal, common environmental, and additive genetic effects, and thermal plasticity for maternal and genetic effects. We inferred the presence of maternal effects for length ($\chi^2_1 = 7.7$, $P = 0.006$), but not for other traits (condition and CT_{max}: $\chi^2_1 = 0.0$, $P = 1.00$; hct: $\chi^2_1 = 0.02$, $P = 0.887$), and did not find indications that maternal effects varied among thermal regimes (Table S2). The average relative contribution of maternal to the phenotypic variance (m^2) for length was with $m^2 = 0.201 \pm 0.080$ relatively high using a univariate model, but lower or higher using bivariate models between length and either CT_{max} or hct, respectively, rendering m^2 and the confounded h^2 estimates for length somewhat uncertain (Fig. S5).

We inferred the presence of common environmental effects in terms of the tank (length: $\chi^2_1 = 26.6$, $P < 0.001$; condition: $\chi^2_1 = 74.3$, $P < 0.001$) or trial effects (CT_{max}: $\chi^2_1 = 54.1$, $P < 0.001$; hct: $\chi^2_1 = 4.5$, $P = 0.033$). Relative phenotypic variance contributions (c^2) were relatively low for tank effects with, on average,

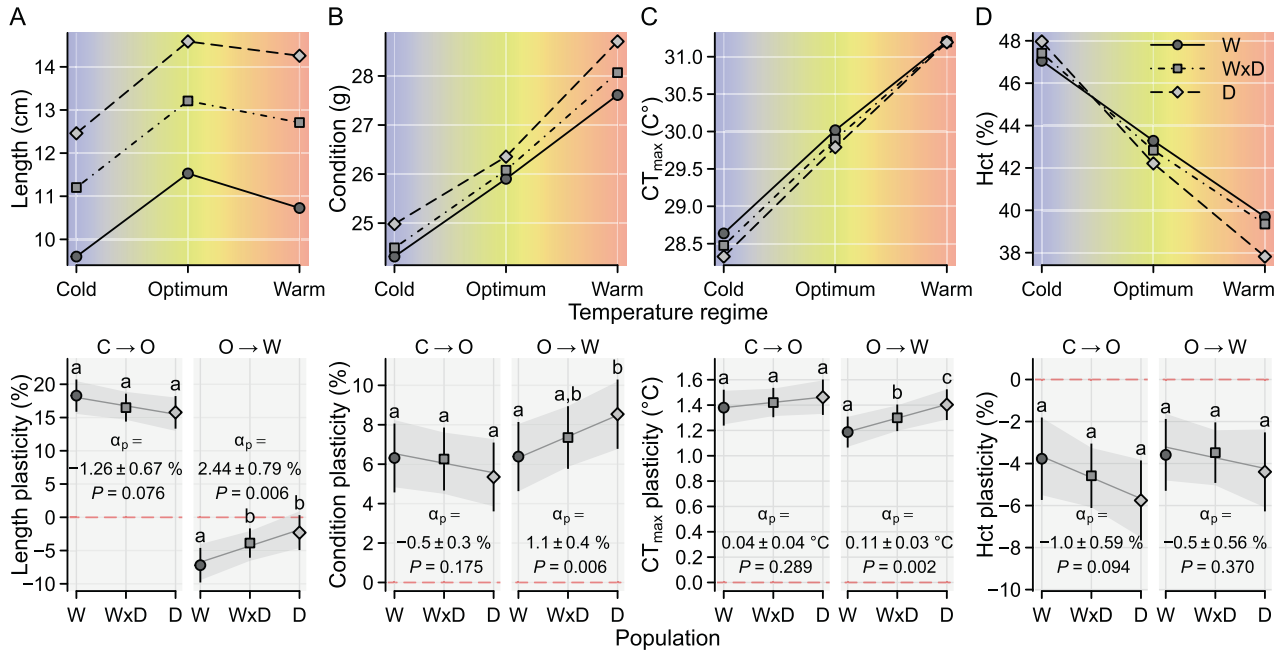

Fig. 3 Temperature-regime-pair-specific thermal plasticity estimates and associated additive genetic outbreeding effect estimates for thermal plasticity (α_p) among wild (*W*), reciprocal wild-domesticated (*W* × *D*), and domesticated (*D*) Atlantic salmon from three temperature regimes. Thermal plasticity per population is represented in the upper panels by slopes between temperature-regime-specific means (observed trait scale) and in the lower panels by the corresponding slope estimates with 95% confidence intervals (proportional scale except for CT_{max}), whereas additive genetic outbreeding effect estimates for thermal plasticity between populations are shown in the lower panels as grey lines with light grey 95% confidence bands (also given as: $\alpha_p \pm$ standard error; *P*-value). Estimates are for temperature pairs with a 4 °C difference (indicated above the lower panels: C → O, optimum—cold; O → W, warm—optimum) and for the four traits of length (**A**; $n = 3932$), condition (**B**; $n = 3932$), CT_{max} (**C**; $n = 538$), and hct (**D**; $n = 538$). Same letters above thermal plasticity estimates in the lower panels indicate within-regime-pair comparisons with no support for differences at FDR ≤ 0.05.

$c^2_{Tank} = 0.012 \pm 0.008$ for length and $c^2_{Tank} = 0.020 \pm 0.012$ for condition. In contrast, relative phenotypic variance contributions for trial effects were, on average, $c^2_{Trial} = 0.111 \pm 0.046$ for CT_{max} but only $c^2_{Trial} = 0.027 \pm 0.020$ for hct. For CT_{max}, removing the hour-of-trial covariate resulted in $c^2 = 0.185 \pm 0.063$, and for hct, removing the day-of-trial covariate resulted in $c^2 = 0.115 \pm 0.046$. Thus, the hour-of-trial and day-of-trial trend effects accounted for 7.4 and 8.8% of the phenotypic variance for CT_{max} and hct, respectively, emphasizing the need to account for methodological biases.

We had estimated within-population thermal plasticity as the difference between temperature regimes in relative contribution of the additive genetic variance (V_A) to the phenotypic variance (heritability, h^2; for evolvability estimates see Tables S3 and S4), and also as the re-ranking of breeding values as indicated by low genetic correlations (R_G) between regimes. We had estimated the V_A and h^2 differences between regimes and R_G because these parameters affect the selection efficiency across temperatures (Falconer 1952). Controlled for other model effects, we estimated h^2 for all four traits, which ranged widely among traits between 0.14 and 0.77 (Fig. 4E–H). We inferred the presence of V_A differences for all traits (tests for heteroscedastic V_A: length, $\chi^2_2 = 14.3$, $P = 0.001$; condition, $\chi^2_2 = 7.0$, $P = 0.030$; CT_{max}, $\chi^2_2 = 6.4$, $P = 0.042$; hct, $\chi^2_2 = 4.8$, $P = 0.089$). However, based on more rigorous pairwise h^2 contrasts, we identified differences only for condition and CT_{max} and only between the cold and optimum regimes (condition $\Delta h^2 = 0.198 \pm 0.079$, $t_{32} = 2.51$, $P = 0.017$; CT_{max} $\Delta h^2 = -0.379 \pm 0.079$, $t_{32} = 5.41$, $P < 0.001$). For CT_{max}, we observed a re-ranking of breeding values between the optimum and warm regimes (Fig. 4C), which was also indicated by a relatively low between-environment genetic correlation ($R_G = 0.33 \pm 0.28$) compared to those of other traits ($R_G > 0.8$; Fig. 4E–H).

Correlations among length, CT_{max}, and hct

We had quantified pairwise correlations among length, CT_{max}, and hct at the genetic (R_G), environmental (R_E), and phenotypic (R_P) levels based on bivariate animal models. We estimated for trait pairs involving length opposing correlation signs at the genetic and environmental (residual) levels, but confidence intervals were different from zero only for genetic and phenotypic correlations (Table 2). Specifically, between length and CT_{max} relatively high negative genetic correlations, which were consistently estimated both within and among regimes, were opposed by lower positive environmental correlations that resulted in low to moderate net negative phenotypic correlations within regimes. Between length and hct from the cold regime, a moderate positive genetic correlation was opposed by a low negative environmental correlation that resulted in a low net positive phenotypic correlation. Lastly, between CT_{max} and hct (both here controlled for length), we estimated a net negative phenotypic correlation in the cold regime that underlaid a negative genetic correlation (Table 2). Not controlling for length did not change this latter result (not shown).

DISCUSSION

We present parameter estimates on traits commonly employed in thermal tolerance studies and used as performance proxies under both warming trends (growth, body condition) and thermal extremes (CT_{max}, hct). For all traits, we quantified population-specific effects for, and gene flow (i.e., outbreeding) effects on, temperature-regime-specific expression and thermal plasticity between regimes. We also provide somewhat crude but nevertheless valuable insights into the presence of within-population maternal, genetic, and environmental effects, and genetic correlations between regimes, and lastly of correlations between

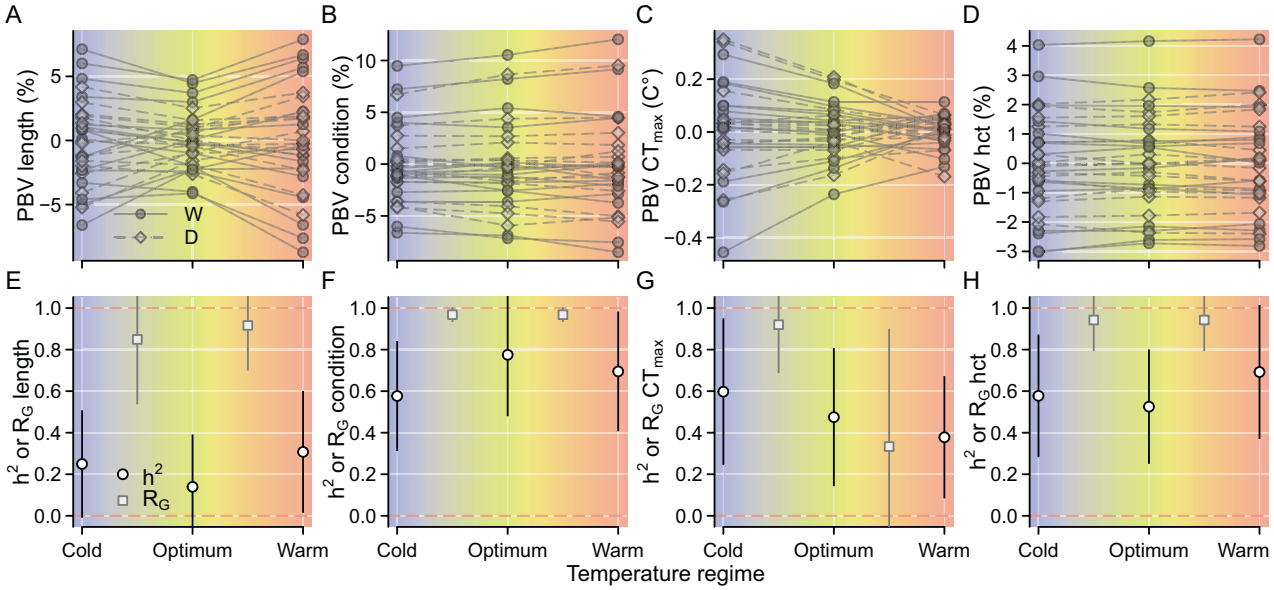

Fig. 4 Temperature-regime-specific predicted breeding values (PBV; i.e., additive genetic effects) and their thermal plasticity. PBV estimates (**A**–**D**) are controlled for fixed effects as shown in Table 1, for four traits and 16 wild (*W*) and 16 domesticated (*D*) Atlantic salmon breeders and based on their progeny performance records in three temperature regimes. Thermal plasticity in the upper panels is depicted by breeder-specific slopes for PBVs between temperature-regime pairs with a 4 °C difference. The lower panels (**E**–**H**) show corresponding estimates (with 95% confidence intervals) for the regime-specific proportion of the additive genetic to the total phenotypic variance (h^2) and for thermal plasticity among genotypes between temperature regime pairs with a 4 °C difference as estimated by genetic correlations (R_G). Please note that estimate confidence intervals per trait are highly correlated between most regimes. Estimates are for the four traits of length (**A**, **E**; $n = 3932$), condition (**B**, **F**; $n = 3932$), CT_{max} (**C**, **G**; $n = 538$), and hct (**D**, **H**; $n = 538$).

some traits. Collectively, our results provide a rare multivariate perspective on the evolutionary potential of populations to different components of climate change while considering outbreeding effects.

We inferred different responses of length and condition to temperature increase above the optimum, with stagnant or decreasing growth for length but an increasing condition. This result may reflect a difference in the thermal performance for processes related to somatic growth and energy storage. We also present evidence that acclimation temperature has population and family-specific effects on the critical thermal maximum (CT_{max}). We, furthermore, present evidence that population- and family-specific CT_{max} estimates (and their differences) preceded by colder acclimation temperature do not substitute estimates preceded by warmer acclimation temperatures. These results on CT_{max} have considerable implications for interpretations of CT_{max} studies that are based on single populations, few families, or a single acclimation temperature. Lastly, growth rate and CT_{max} were negatively correlated both at the genetic and phenotypic levels. This result leads to the expectation for a correlated, but antagonistic, response of growth rate and CT_{max}, which may limit the evolutionary change of both traits.

Gene flow may modify a local response potential and the standing genetic variance signifies adaptive potential

We estimated that temperature-regime-specific performance and extreme tolerance proxy traits, and their thermal plasticity, differ between populations. We also estimated that temperature-specific trait expression and thermal plasticity inherit largely and completely additively between populations, respectively. By estimating these differences between populations in a common garden experiment, and estimating that between-population hybrids expressed largely intermediate (whereby an absence of reciprocal differences indicated a lack of parent-of-origin effects), we provide strong evidence that the population effects are genetic. Thus, gene flow may influence thermal performance and its evolution. The effects may be expected to be largely additive and thus predictable via average performance differences between outbreeding populations.

We also detected considerable genetic variance for both temperature-regime-specific expression and thermal plasticity within populations. Thus, the genetic variation both segregating between, and present within populations, bears potential to respond to selection and aid in thermal adaptation to increase in either average temperatures or the occurrence of extreme temperatures. These results add to existing genetic parameter estimates for thermal performance and tolerance traits that promise adaptive potential (Munday et al. 2017; Catullo et al. 2019; Kelly 2019; Stillman 2019). However, our results also suggest that the response to selection may be impeded by both genotype-by-environment interactions for between- and within-population genetic effects and by correlations between-traits that may be unfavourable under some selection regimes.

Genotype-by-environment interactions for populations and breeding values

Between-population differences depended on the temperature regime for somatic growth and CT_{max}, i.e., both traits exhibited genotype-by-environment interactions. Expected average phenotypic contributions from genetic effects via gene flow on both traits may therefore depend on the prevailing temperature (see limitations for CT_{max} below). Notably, the estimated genotype-by-environment interaction for growth may reflect a larger (upper) temperature performance breadth of the more rapidly growing domesticated population. We thereby collected indirect evidence against the hypothesis that growth rate trades off with thermal performance breadth (Huey and Kingsolver 1989; Kingsolver et al. 2004; Kingsolver et al. 2015), although domesticated salmon may still underperform in their relative growth rate change compared to wild salmon when approaching lower temperatures. Nonetheless, growth rate has been suggested to correlate with the thermal performance breadth in wild Atlantic

Table 2. Regime-specific between-trait (partial) correlation estimates among length, CT_{max}, and hct, at either the genetic (R_G), environmental (R_E), or phenotypic (R_P) levels, and between regimes for R_G, based on bivariate animal models that account for population (wild, wild-domesticated, domesticated), temperature regime (cold, optimum, warm), population-by-regime effects, and additional covariates (for CT_{max}: hour of day; for hct: date, population-by-date).

Regime	R_G (95% CI)	R_E (95% CI)	R_P (95% CI)
Length, CT_{max}			
Cold	**−0.72 (−1.12 to −0.32)**	0.24 (−0.37–0.84)	**−0.53 (−0.96 to −0.10)**
Optimum	**−0.86 (−1.37 to −0.34)**	0.51 (−0.17–1.19)	−0.42 (−0.85–0.01)
Warm	**−0.71 (−1.05 to −0.37)**	0.18 (−0.39–0.74)	**−0.40 (−0.68 to −0.13)**
Cold.Optimum	**−0.78 (−1.18 to −0.39)**		
Optimum.Warm	**−0.78 (−1.16 to −0.40)**		
Cold.Warm	**−0.68 (−1.08 to −0.28)**		
Length, Hct			
Cold	**0.55 (0.07–1.02)**	−0.18 (−0.90–0.54)	0.45 (−0.01–0.91)
Optimum	0.15 (−0.97–1.27)	−0.12 (−0.37–0.14)	−0.02 (−0.36–0.33)
Warm	0.29 (−0.28–0.86)	−0.03 (−0.42–0.35)	0.19 (−0.15–0.54)
Cold.Optimum	−0.01 (−0.75–0.72)		
Optimum.Warm	0.27 (−0.41–0.96)		
Cold.Warm	**0.67 (0.26–1.08)**		
CT_{max}, Hct			
Cold	**−0.51 (−0.97 to −0.04)**	−0.08 (−0.51–0.36)	**−0.46 (−0.9 to −0.02)**
Optimum	−0.24 (−0.88–0.40)	0.74 (−0.56–2.04)	−0.02 (−0.63–0.60)
Warm	0.22 (−0.39–0.83)	−0.27 (−0.88–0.35)	0.13 (−0.3–0.56)
Cold.Optimum	−0.27 (−0.87–0.33)		
Optimum.Warm	0.09 (−0.58–0.76)		
Cold.Warm	−0.24 (−0.83–0.34)		

Correlations between CT_{max} and hct are also controlled for length. Bolded values signify correlations with an approximate 95% confidence interval excluding zero.

salmon populations (Jonsson et al. 2001), which supports the hypothesis of a general vertical performance shift between the populations studied here. These results together suggest that growth rate and performance breadth correlate positively in Atlantic salmon. The question remains whether growth rate and thermal performance breadth, both higher in the domesticated population, underlie a shared set of genes. The answer may be important because growth rate and a third trait, namely CT_{max}, showed strong negative genetic correlations across regimes, indicating that genes may be shared (or in linkage disequilibrium) between CT_{max} and growth. Thus, it remains to be evaluated whether growth rate, thermal performance breadth, or both, trade off with CT_{max}. If (upper) thermal performance breadth and CT_{max} correlate negatively, this may imply that Atlantic salmon are unlikely to adapt simultaneously to increasing water temperature (by increasing their upper thermal performance breadth) and to thermal extremes.

Within populations, genotype-by-environment interactions were—with one exception—small or absent, indicated by both relatively similar heritability and relatively high (>0.8) genetic correlation estimates across regimes. Such low or absence of genotype-by-environment interactions within populations indicate that useful predictions of selection responses adhere to general heritability estimates (with the usual limitations: Robertson 1959; Hill 2010), although our crude estimates may only indicate trends. For CT_{max}, however, we inferred with increasing acclimation temperature a decreasing heritability and considerable genotype re-rankings between the optimum and warm regimes, which encompassed a re-ranking of both the population means and the breeding values within populations. However, CT_{max} is—by definition—an upper-temperature response and may thus be in nature preceded by a warm, rather than cold or optimal, acclimation temperature. Thus, genotype-by-environment interactions for CT_{max} may matter for theoretical considerations but may have little biological relevance to estimating adaptive potential to thermal extremes. This makes CT_{max} estimates preceded by the warmest acclimation temperature the most relevant. Notably, under the warmest acclimation temperature, populations did not show differences for size-controlled CT_{max} and its heritability was the lowest estimated. Thus, even though thermal performance breadth for somatic growth appeared to differ between populations, their CT_{max} did not when preceded by warmer acclimation temperature.

Although populations did not differ for CT_{max} under the warmest acclimation temperature and heritability was lower than under the other acclimation temperatures, we still estimated a moderate heritability for CT_{max}. Thus, CT_{max} can be expected to respond to selection via within-population genetic variation, but which is unlikely to be as excessive as expressed following colder acclimation temperatures (e.g., Anttila et al. 2013). Many of these ideas based on salmon are corroborated by selection experiments using zebrafish (a tropical fish) where warm acclimation slowed down the selection response towards higher CT_{max}, and which indicates a lower heritability for CT_{max} at higher acclimation temperature also in zebrafish (Morgan et al. 2020). Together, these results suggest the presence of a diminishing genetic variation for upper thermal tolerance with increasing acclimation temperature (i.e., a hard ceiling) in both cold- and warm-water fishes.

Unfavourable correlations between-traits

Growth rate covaried negatively with CT_{max}, at both the population and individual levels. Similar negative but

phenotypic correlations were estimated in same-aged cutthroat trout (Underwood et al. 2012) and different-aged coral-reef fish (Messmer et al. 2017). Negative genetic correlations were also estimated in several bird species (Bowen and Washburn 1984) and suspected in rainbow trout (Hartman and Porto 2014). Previously estimated negative genetic correlations between body size and maximum metabolic rate or net aerobic scope in a reef fish suggest an underlying physiological mechanism (Munday et al. 2017). In contrast, correlation estimates between growth and maximum temperature tolerance traits were positive at the phenotypic level, but not different from zero at the genetic level in both turbot (Zhang et al. 2014) and cutthroat trout (Robinson et al. 2008). Thus, the sign of the relationship in cutthroat trout, and possibly other species, may depend on additional unknown parameters, which deserves additional research, although negative relationships may be recovered more often (reviewed by McKenzie et al. 2021).

Nonetheless, we estimated high positive genetic correlations for length across temperature regimes in combination with high negative genetic correlations between length and CT_{max} (as discussed above). Together, this may indicate that a set of genes, stably expressed across temperature environments, jointly underlie growth and CT_{max}, but with antagonistic effects on each trait. It remains to be evaluated whether our inferences apply to only *ad libitum* settings or whether the negative relationship may be cryptic otherwise. Furthermore, under environmental feed limitation, different growth genotypes (and the genetically correlated CT_{max} genotype) may not be fully exposed to selection. For example, even though the higher ranking of domesticated vs. wild genotypes for growth is usually phenotypically realized in nature, differences are much smaller than in the laboratory (Glover et al. 2017, 2018)

Differences in the thermal performance for somatic growth and energy storage may affect life history

We inferred increasing growth for all populations between the cold and optimum regimes, but stagnant or decreasing growth between the optimum and warm regimes, leading to an expectation of a reduced physiological efficiency at too warm temperatures. Conversely, body condition still increased consistently with water temperature. A positive relationship between body condition and the water temperature has been observed previously in Atlantic salmon (Dwyer and Piper 1987; Jonsson et al. 2013; Tromp et al. 2018). Together, these results make way for future research on the temperature dependence of either resource allocation between somatic growth and energy storage or their physiological differences. In salmonids, rapid growth in freshwater propagates both reproduction prior to a feeding migration in males and the initiation of the feeding migration (Hutchings and Myers 1994; Morita et al. 2014; Debes et al. 2020; Debes et al. 2021). Sexual maturation is further accelerated with increasing body conditions (Rowe et al. 1991; Taranger et al. 2010; Debes et al. 2021). With increasing water temperature and under unlimited feed, Atlantic salmon may thus be expected to show higher early male maturation rates and younger migration ages, which agrees with present latitudinal patterns (Klemetsen et al. 2003) and earlier maturation ages observed under increased production temperature in aquaculture (Good and Davidson 2016). The inferred environmental effects, thereby, support a scenario of 'shrinking fish' under climate change (Sheridan and Bickford 2011; Cheung et al. 2012), because maturing fish slow down growth, which however, would not result from evolutionary (Siepielski et al. 2019) but environmental effects. These results may stimulate research on the largely unresolved phenomenon that increasing growth rate via either feed or temperature has differential effects on age and size at maturity (Berrigan and Charnov 1994).

The uncertain role of hct

Hct in stressed fish, such as after CT_{max} trials, may be increased (Gallaugher and Farrell 1998), limiting comparison of our estimates with other studies, but enabled the study of associations between CT_{max} and hct. However, a potential bias applies to all individuals and renders our reported within-study estimates valid. Here, hct *decreased* consistently by 3–4% per 4 °C of temperature *increase*. Assuming that O_2 transport capacity *increases* with temperature, our results may indicate, somewhat counter-intuitive, that a hct decrease may serve this demand. Specifically, a hct decrease increases blood velocity, which increases cardiac output (Gallaugher and Farrell 1998). Alternatively, a hct *increase* with temperature *decrease* may contribute to maintaining performance at slower physiological rates. However, effect estimates of temperature on hct have been very inconsistent in both presence and direction among salmon studies (summarised by Gamperl et al. 2020). In addition, we found little support that regime-specific hct, or its plasticity, differed strongly between fish populations and estimated relatively high heritabilities across temperature regimes. This indicates that hct is neither under strong natural or human selection, because selection may elevate between-population differences and lower heritability.

Under a theory on thermal vulnerability, CT_{max} is expected to correlate positively with hct (Pörtner et al. 2017). This theory was supported by Muñoz et al. (2018) who estimated higher CT_{max} at higher acclimation temperatures in concert with *higher* hct and a *positive* phenotypic correlation between traits in Chinook salmon. However, we estimated partly the opposite: higher average CT_{max} values at higher acclimation temperatures in concert with *lower* average hct values and *negative* phenotypic and genetic correlations between CT_{max} and hct under colder acclimation temperature and none otherwise. Helping in explaining disparities, we inferred that population differences between CT_{max} and hct can depend on acclimation temperature and that both traits were affected by methodological biases. The opposing environmental and genetic correlations between CT_{max} and hct estimated here, and relatively high heritabilities, make a bias in phenotypic correlation estimates possible if relatedness is not statistically controlled for. Altogether, these results make it possible that haemoglobin variation (reviewed by Andersen 2012) or cardiac output (Muñoz et al. 2014b), rather than hct variation, may be more important to blood-oxygen-carrying capacity under increasing temperature.

Study limitations for genetic and maternal variance estimates

Unlike mean estimates, our (co)variance estimates are likely imprecise. Even though sample sizes were relatively high for studies on CT_{max} or hct, they were relatively low for studies on genetic parameters. Using relatively few breeders, we inferred the presence of maternal effects for body length but not for other traits. In contrast, a previous study on Chinook salmon inferred maternal effects for CT_{max} (Muñoz et al. 2014a). However, using relatively few females in the studies by Muñoz et al. (2014a) ($n = 5$) and ours ($n = 16$) makes it unlikely that maternal effects were reliably disentangled from genetic effects in either study. This was here also indicated by a disagreement between univariate and bivariate model estimates for maternal and genetic variances (Fig. S5). Strong maternal effects exist in Atlantic salmon on size at first feeding (Debes et al. 2013; Solberg et al. 2014) but may disappear rapidly (Van Leeuwen et al. 2016). Thus, maternal effects on growth in our study (or on CT_{max} by Muñoz et al. 2014a) may have been small or absent and the genetic effect contribution larger than reported.

We estimated all (co)variance components across populations with one having a human selection history (Glover et al. 2009). If the investigated traits deviate from the infinitesimal model, population-specific estimates may deviate from those reported here. Furthermore, if the non-additive genetic variance is

important, but which appears rare (Hill 2010), between-population outbreeding may convert population-specific additive genetic variance to non-additive genetic variance (Whitlock et al. 1993). Lastly, we assumed that fitting population means removed genetic effect variance segregating between populations, which would make within-population estimates more general, and which is likely but not guaranteed (Muff et al. 2019). Any of the above may limit our estimates to a between-population outbreeding scenario before reaching equilibrium, which, however, may fit the scenario when increased gene flow is present under climate change as occurring either naturally, by re-current aquaculture escapes, or by facilitated gene flow.

CONCLUSIONS

Our results suggest that population outbreeding affects thermal performance and plasticity via segregating predominantly additive genetic effects. In addition, all traits (growth, condition, CT_{max}, hct) showed moderate to high heritabilities and all but CT_{max} showed high genetic correlations across environmental temperatures, indicating a predictable evolutionary potential across changing temperatures. However, possibly shared genetic effects for upper thermal performance breadth and thermal extreme tolerance may act antagonistically and growth rate consistently appeared to trade-off with CT_{max} both genetically and phenotypically, which may restrain simultaneous evolution of both traits. Our results further stress the importance of genotype-by-environment interactions both between and within populations, whereby genetic variation for thermal extreme tolerance diminishes with increasing acclimation temperature.

DATA AVAILABILITY

Underlying data is available at the Dryad repository: https://doi.org/10.5061/dryad.mgqnk98x9.

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AUTHOR CONTRIBUTIONS

Conceptualization: PVD, KAG, MFS; Methodology: PVD; Formal analysis: PVD; Investigation: MFS, PVD, IHM, LD; Resources: KAG, IHM, LD; Data Curation: PVD; Writing—original draft preparation: PVD; Writing—review and editing: PVD MFS, KAG, IHM, LD; Visualization: PVD; Project administration: KAG, MFS; Funding acquisition: KAG.

COMPETING INTERESTS

The authors declare no competing interests.

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