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Exposure to warmer water and prenatal cortisol independently alter transcriptional plasticity during early development in Chinook salmon (*Oncorhynchus tshawytscha*)

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Abstract

Freshwater ecosystems are changing rapidly from ongoing human-induced environmental change. Determining whether organisms have the adaptive capacity to respond to stressors and by which mechanisms remains a challenge. Within the environmental-match framework, we experimentally manipulated a maternal signal of environmental stress to see whether it primed gene transcription plasticity to allow developing Chinook salmon (*Oncorhynchus tshawytscha*) to respond to water temperatures expected from climate modelling. Our OpenArray® chip approach across four functional groups (growth, metabolism, immune, and stress) revealed elevated temperatures induced a diversity of effects on gene transcriptional profiles at different early life stages for candidate genes at all levels of the hierarchical evaluation (complete gene sets, functional gene groups, and individual gene transcriptional phenotypes). However, contrary to predictions, prenatal cortisol did not alter these transcriptional effects further and did not rescue temperature-driven increases in mortality. Results suggest that while exposure to signals of maternal stress have the capacity to induce complex life stage- and system-specific responses, they may not further mitigate existing responses induced by elevated water temperatures in Chinook salmon (possibly other species).

Key words: Chinook salmon (*Oncorhynchus tshawytscha*), climate change, gene transcription, water temperature, cortisol, maternal effects

Introduction

Human-induced rapid environmental change (HIREC) is generating multiple novel stressors, as well as combinations of stressors, at extremes that wildlife have not experienced in their evolutionary history (Sih et al. 2011; Gissi et al. 2021). Anthropogenic impacts are directly, and indirectly, affecting habitat degradation (Poff et al. 2002), species invasions (Salo et al. 2007), pollution (Dudgeon et al. 2006), and of course global climate change (Romero-Lankoa et al. 2014). Anthropogenic factors have already had a serious impact on global temperature changes over all continents for more than a 50-year period (Bindoff et al. 2019). In aquatic ecosystems as a whole, average global surface water temperatures are increasing (Wigley and Raper 2001) and these sustained increases are having a diversity of abiotic and biotic impacts, including increases in the intensity of extreme weather events (Walsh et al. 2016), habitat degradation (both ecologically and chemically; Vollenweider et al. 1974; Mallin et al. 2006; Butchart et al. 2010), reducing annual ice coverage (Karl and Trenberth 2003), resulting in impacts on biodiversity ranges (Cheung et al. 2009), and changes to ecosystem functioning (Malhi et al. 2020). Alarmingly, in freshwater lakes and rivers, decadal increases in average water temperatures are more than twice that of the oceans (Kintisch 2015; Islam et al. 2019). As a result, many fish species are now facing chronic and acute temperature fluctuations beyond their species' preferred temperature ranges (Ficke et al. 2007; Gunderson et al. 2016), and these fluctuations are expected to become more frequent as climate change persists (Jeffries et al. 2014). However, determining whether these organisms have the adaptive capacity to respond to HIREC (Huo et al. 2023), and if so, by which mechanisms, remains an ongoing challenge (Beever et al. 2016; Funk et al. 2019).

Since environmentally induced phenotypes may have a greater potential to result in adaptive responses than mutations (West-Eberhard 2005; Moczeck et al. 2011), quantifying the degree of developmental plasticity in response to HIREC is increasingly seen as critical to determine whether organisms possess the adaptive capacity (Beever et al. 2016; Funk et al. 2019) necessary to respond to environmental change (Desmond 2018). In response to elevated environmental stressors, developing offspring of ectothermic fish could

respond plastically in at least two ways. First, there is increasing evidence that environmental stressors experienced by the mother (i.e., such as impacts on maternal physiology induced by direct and indirect effects of climate change) can be transmitted to offspring via the egg in the form of elevated stress (glucocorticoid (GC)) hormones (Meylan et al. 2012; Taylor et al. 2016), inducing developmental plasticity that may benefit (or even hinder) the offspring in that same stressful environment (Nettle and Bateson 2015; Capelle et al. 2016; Sopinka et al. 2016; Warriner et al. 2020*a*, 2020*b*). Second, it is known that eggs developing under warmer water temperatures experience a suite of developmental responses in fish (Murray and McPhail 1988; Zuo et al. 2012; Paaijmans et al. 2013; Wilder et al. 2024). While plasticity in phenotypic traits such as body size, growth, and behaviour is increasingly being examined within this more integrative, multi-stressor (i.e., pre- and post-natal stress) framework (Warriner et al. 2020*a*), fewer studies have examined the underlying mechanisms (e.g., gene expression) by which these interactions produce variation in offspring phenotypes (Colson et al. 2019; D'Agostino et al. 2019; Venney et al. 2020, 2021). Given the pleiotropic nature of the genes that underscore environmentally initiated phenotypes (e.g., Chesler et al. 2005; Kim et al. 2016), and the advancements in high throughput genomics (Lowe et al. 2017; Connon et al. 2018; Houde et al. 2019), more and more studies are increasingly focussing on transcriptional mechanisms that quantify the degree to which an organism can respond to an environmental stressor within their current genotypic constraints (Harder et al. 2020). Through approaches such as quantitative real time polymerase chain reactions (qRT-PCR), researchers can target specific genes of interest to use as biomarkers to identify the degree to which organisms alter their gene expression in response to environmental conditions (Olsvik et al. 2013; Akbarzadeh et al. 2018). The ability to quantify variation in transcriptional profiles for genes across multiple functional groups (e.g., growth, metabolism, immune function, stress response) at multiple time periods through development is what makes qRT-PCR an indispensable tool when performing a "Time-Series" approach for identifying how and why environmentally stressful conditions alter early, and transient, responses to environmental stressors (e.g., chronically elevated temperatures, prenatal stress; Aubin-Horth and Renn 2009). The goal of this type of mechanistic approach is to examine the underlying early developmental mechanisms that respond to differential prenatal stress signals to ultimately alter an individual's response to an overall decline in environmental quality (Venney et al. 2020, 2021). As such, examining variation in gene expression in response to interacting stressors that developing offspring experience may help to determine the role that prenatal stress signals play during embryonic development: perhaps a larger role than previously appreciated in modifying future (possibly adaptive) responses to chronically elevated thermal regimes that offspring will face under climate change.

Using an experimental approach in a semelparous Pacific salmonid—Chinook salmon (*Oncorhynchus tshawytscha*), our goal was to examine stage-dependent (i.e., eyed egg, alevin, and fry) developmental plasticity in gene transcription in response to altered environmental conditions resulting from exposure to (i) elevated rearing water temperature and (ii) maternal stress signals (i.e., prenatal exposure to elevated egg GCs). Specifically, within the framework of the environmental-match hypothesis (Sheriff and Love 2013; Warriner et al. 2020*a*), we examined whether exposure to a maternally derived stress signal (elevated prenatal GCs) induced predictable phenotypic and fitness benefits (Gluckman et al. 2005) in offspring that better matched developmental phenotypes to expected future environmental stressors (i.e., elevated water temperature). Examining the mechanisms underlying this potential adaptive capacity of Pacific salmon to current ambient and future climatic changes in thermal conditions using this predictive framework is relevant for multiple reasons. From an applied (conservation) perspective, North American populations of Pacific salmonids are experiencing severe population declines, many of which have been attributed to the direct and indirect effects of increasing water temperatures (Crossin et al. 2008; Crozier et al. 2010; Isaak et al. 2012). From a mechanistic perspective, Pacific salmonids are excellent models for testing the effects of maternal and temperature stressors during development (Warriner et al. 2020*a*, 2020*b*; Sturba 2021) as they are ectothermic and therefore highly susceptible to the effects of elevated water temperatures (McCullough et al. 2009). Beyond the resource-limitation and migratory stress experienced by female Pacific salmon leading up to spawning, environmental stressors can further increase maternal circulating plasma GCs (Carruth et al. 2000; Cook et al. 2014; Currier 2021; Sturba 2021) and corresponding egg GCs (Mingist et al. 2007; Currier 2021; Sturba 2021). Finally, maternal spawning grounds and the offspring's early developmental environment overlap (i.e., a maternal stress signal has the potential to act as an honest indicator of the quality of the offspring's future environment; Love et al. 2013; Currier 2021; Sturba 2021). Using previous research in salmonids examining developmental responses to environmental stressors as a guide, we chose a set of 26 genes across four key biological processes (i.e., growth, metabolism, stress response, immune function) to examine these transcriptional responses. The direct and indirect effects of elevated water temperature play a key role in cellular processes for aquatic ectotherms and have been linked to important effects on fish growth, metabolism, immune function, and stress response (Gillooly et al. 2001; Forster et al. 2012; Smith et al. 2013; Yang et al. 2015; Venney et al. 2020; Banousse et al. 2024). We expected elevated temperature exposure to accelerate cellular and metabolic functions, since metabolism increases exponentially with increasing temperature in fish (Schulte 2015), which often drives offspring to reach developmental stages earlier, and at reduced body size for early life history stages (Zuo et al. 2012). We also expected that genes associated with innate stress responses (e.g., chaperonins, peroxidases, superoxide dismutase; Liu et al. 2018) would be upregulated to aid/rescue cellular activity due to elevated temperatures and the possible free radical accumulation associated with this stressor. We anticipated that immune gene expression would be reduced at elevated temperatures given that chronically elevated temperatures

often impair immune function (Dittmar et al. 2014). As a result of this impairment, we also expected overall survival under elevated water temperatures to be lower (Daufresne et al. 2009; Whitney et al. 2013). However, based on predictions of the environmental-matching hypothesis (Sheriff and Love 2013), we expected preparatory prenatal (egg GC) signalling to aid in mitigating possible negative effects associated with development in elevated thermal environments. Under elevated incubation temperatures, we would therefore predict that exposure to elevated egg GCs would result in a "rescue effect" of transcriptional responses in the key biological processes examined compared to fish raised under elevated water temperatures that did not receive the predictive prenatal stress signal, ultimately leading to increased survival. Overall, within the context of this experimental design we aimed to answer three main questions: (1) do chronically elevated thermal conditions significantly alter gene transcription of important biological processes during early development?; (2) does a prenatal stress signal, potentially predictive of future stressful (i.e., warm water) conditions, modulate gene transcription within those same biological processes to help mitigate some of the negative aspects of those deteriorating thermal environmental conditions?; (3) does this prenatal stress signal help to rescue offspring (i.e., reduced mortality) from the negative effects of warm water conditions during early development? The importance of determining how chronic hyperthermal stress modulates gene transcription profiles, and whether a prenatal stress signal can mitigate these responses, is an important step in evaluating how predicted future environmental conditions will affect the underlying cellular processes during sensitive developmental stages in freshwater fishes (Semeniuk et al. 2022).

Methods

Study system

Intensive stocking of Chinook salmon in Lake Ontario, Canada, began in 1967 to naturalize the species to the ecosystem to increase biodiversity, manage predator–prey interactions, and for recreational fishing (OMNRF 2015). We focussed on the Credit River breeding population (43°34′40.0″N, 79°42′06.3″W), a tributary of Lake Ontario, during the fall (October–November) when mature fish migrate upstream to spawn. This population is relevant for this study because global climate change is one of the main challenges that aquatic species in the Laurentian Great Lakes is already facing (Collingsworth et al. 2017), a situation that is predicted to worsen based on global climate models (Wigley and Raper 2001; Austin and Colman 2007; Woolway et al. 2021). It is therefore important to evaluate how this population responds to chronic temperature increases, with the goal of eventually determining the effects of temperature stressors on the naturalized Great Lakes Chinook salmon as well as for Pacific salmon populations in their native ranges. Using electrofishing, we collected eggs and milt from eight females and eight males on 17 October 2017. The gametes were maintained at river temperature and transported (Warriner et al. 2020*a*) to the Great Lakes Institute for Environmental Research in Windsor, Ontario, for fertilization, cortisol exposure, and incubation.

Egg fertilization and egg cortisol manipulation

Each female's eggs were separated into eight containers (90 g or ∼300 eggs per container, containing ovarian fluid from each female) for fertilization, for an approximate total of 2400 eggs per female. Milt from the eight males was pooled in equal volumes to give each male similar likelihood of fertilizing each egg. We then added ∼0.5 mL of the pooled milt to each egg container and gently mixed the eggs. To activate the sperm, 60 mL of the collected river water was added to each container (Lehnert et al. 2018). After a 2 min incubation period, 2 mL of 400 mg·L^{-1} concentration of cortisol (H4001, Sigma–Aldrich Canada Co) dissolved in 90% ethanol (HPLC grade, Sigma–Aldrich Canada Co) was added to half of the containers, along with 740 mL of Credit River water, to generate a final cortisol concentration of 1000 ng·L^{-1} (Sopinka et al. 2014; Capelle et al. 2016). The other half of the containers were exposed to a vehicle only solution (ethanol and river water) to act as a control treatment. All the containers were exposed to their respective treatment solutions for 2 h. The cortisol concentrations and incubation times were based on the literature that produced environmentally relevant egg cortisol concentrations (Auperin and Geslin 2008; Burton et al. 2011; Capelle et al. 2016; Sopinka et al. 2016). After the 2 h incubation period, the eggs were removed from the solutions and rinsed with dechlorinated water. A subset of three eggs was removed from each female both prior to fertilization and after cortisol treatment, to determine the effectiveness of the cortisol treatment compared with each female's background egg cortisol concentration.

Water temperature treatment

To determine the effects of elevated water temperature on offspring development, we used two identical incubation systems with two different temperature regimes based on methods outlined in Warriner et al. (2020*a*). The first egg incubation stack was maintained at the current ambient river temperatures the offspring would currently be experiencing (referred to hereon in as "current T_w"), based on monthly records collected from stations close to the spawning grounds of the Credit River (Provincial Water Quality Monitoring Network). The second egg incubation stack was consistently maintained at +3 °C above the current ambient river temperature incubation (referred to hereon in as "elevated T_w"), an elevation that is consistent with climate projections for the region (van Vliet et al. 2013). Both incubation stacks were adjusted throughout the rearing process to mimic seasonal fluctuations that they would naturally experience in the current river system, maintaining the +3 °C difference (see Warriner et al. 2020*a*). Temperatures were tracked every minute within each stack using HOBO Pendant MX Water Temperature Loggers (Onset Inc.). Each incubation stack was equipped with seven trays that were subdivided into 16 (10.2 cm × 7.6 cm) cells. From each female, two cortisol and two control treated repli-

cates were randomly assigned to each of the two incubation stacks. We then divided the ~300 eggs from each cortisol/control treatment container, for each female, into three random replicate cells (i.e., ~100 eggs/cell) within their respective incubation stacks. This two-by-two design resulted in four treatment groups (control:current T_w; cortisol:current T_w; control:elevated T_w; cortisol: elevated T_w), which was replicated within each treatment per female, per stack to control for fertilization-container effects, with three replicates within each of these two female-treatment replicates to further control for any positional effects within the incubation trays.

Rearing and sampling

During egg incubation, we monitored developmental progress and mortality every third day through visual inspection (Warriner et al. 2020*a*). Any eggs that had died between checks were removed, placed in labelled containers (with tray and cell ID), and stored in Stockard's solution (5% formaldehyde, 4% glacial acetic acid, 6% glycerin, 85% water) to quantify fertilization rates. We also assessed survival rates of fertilized eggs by developmental stage (i.e., number of offspring that died between fertilization and fry sampling by assessing at what stage death occurred for fertilized eggs up to fry—see below for stage descriptions) that were used in subsequent survival analyses. Once individuals reached the eyed stage, death was visually evident.

Since ectotherms develop at faster rates under elevated temperatures (Alderdice and Velsen 1978; Zuo et al. 2012), we used accumulated thermal units (ATUs) to identify when the current T_w group had reached the same developmental stage as the elevated T_w group (Neuheimer and Taggart 2007). We monitored the daily temperatures that offspring experienced using temperature probes in the incubation stacks (accurate to ±0.2 °C) to identify how many ATUs were required for the elevated temperature group to reach the desired developmental stage. To determine the effects of incubation temperature and prenatal cortisol exposure on gene transcription in juvenile Chinook salmon, we focused on three important developmental stages during their early life: eyed egg (304/317 ATU elevated T_w and current T_w, respectively), alevin (537/541 ATU elevated T_w and current T_w, respectively), and fry (868/848 ATU elevated T_w and current T_w, respectively). When offspring had reached these developmental stages, we euthanized three individuals from each cell in the incubation stack using an overdose solution of clove oil, and placed them in a tube filled with 10 mL of a high salt preservative (700 g ammonium sulfate, 935 mL ddH_2O, 40 g sodium citrate, 25 mL 0.5 M EDTA, adjusted to pH of 5.2 using 1 mol·L^{-1} sulfuric acid, filtered using 0.45 μm membrane filters) per gram of sample. We stored tubes at room temperature for 24 h to allow the solution to permeate the tissue, and then moved the samples to −20 °C to preserve RNA for future transcriptional analyses. Eyed eggs were perforated to allow the high salt solution to interact with the tissue, and the alevins had their yolk-sacs removed to increase the solution:tissue ratio and to reduce the presence of proteins during future extraction.

Egg cortisol assays

We used enzyme-linked immunosorbent assays (Cayman Cortisol kits; Capelle et al. 2016) to measure egg cortisol levels to ensure that the cortisol treatment successfully increased egg cortisol levels to mimic an environmentally relevant maternal stress signal. For each female, we measured cortisol in eggs sampled both prior to fertilization and after receiving the cortisol or control treatment ($N = 24$). Eggs ($n = 3$) from each treatment group within each female were weighed, homogenized in 1.2 mL of assay buffer, and cortisol was extracted using 3 mL of diethyl ether (Capelle et al. 2016). Samples were then agitated by vortexing for 30 s, centrifuged for 5 min at 3500 rpm, and allowed to rest for 30 min, after which they were flash frozen at −80 °C for 30 min. The supernatant was decanted, and the remaining sediment was left overnight to allow the rest of the liquid to evaporate. Samples were then reconstituted in 1.2 mL of assay buffer and stored at −80 °C for future analysis. Samples were run in triplicate at a 1:50 dilution and the cortisol concentration was measured at a wavelength of 412 nm. Intra- and inter-plate coefficients of variation for a control replicated within plates (three cells per plate) and across plates (one control per plate) were 5.0% and 22.5%, respectively.

Primer and probe development

Due to temperature having such broad effects, we included candidate genes from four key physiological processes for transcriptional profiling. Most of our Taqman© primer and probe sequences were from Toews et al. (2019). However, several primer/probe combinations were also developed for this study using data from the National Center for Biotechnology Information for Chinook salmon transcript sequence or transcript sequences from species within the same genus (*Oncorhynchus*) or family (*Salmonidae*). Primer and probe sequences were designed using Primer Express 3.0 software and optimized for amplicon length, melting temperature, and minimal dimerization. Primers were tested for successful amplification using a gradient PCR to confirm that primers functioned at target annealing temperatures (i.e., ≥61 °C) for PCR, and generated the proper amplicon length, using gel electrophoresis. See Supplementary Materials for full list of candidate gene primer and probe sequences.

mRNA extraction and cDNA synthesis

A total of 417 fish samples (140 eyed egg, 138 alevin, and 139 fry) were included in the gene transcription analysis. RNA extraction from whole body samples (average mass; eyed = 10 mg, alevin = 38 mg, fry = 347 mg) was performed using TRIzol™ Reagent (Ambion) following manufactures' protocols (Invitrogen) with slight modifications. First, while eyed and alevin samples followed the protocol exactly, fry were too large to homogenize in a single 2.0 mL graduated tube (UltiDent Scientific) with 1 mm silica beads using an unequal centrifugation bead breaker (Biospec Products). As such, each fry sample was dissected into approximately three equal portions, each section placed in three separate tubes, and each underwent homogenization in TRIzol (following manufacturer's protocol) mixed into a single 15 mL Fal-

con tube, and then 750 μL of the mixed solution was added to a new 2.0 mL tube to complete the protocol. Total RNA samples were resuspended in water and RNA concentration was measured by spectrophotometry on a NanoVue spectrophotometer (General Electric Company; average RNA concentration = 945 ng·μL^{-1}). We synthesized cDNA from 250 ng total RNA for each sample using High Capacity cDNA Reverse Transcription Kits (Applied Biosystems) using half reactions of the manufacturer's protocol. All cDNA samples were stored at −20 °C until qRT-PCR analysis.

Quantitative real time PCR

We used Taqman® OpenArray® chips from Applied Biosystems to quantify variation in gene transcription using the QuantStudio 12K Flex Real-Time PCR System (Applied Biosystems). Each chip consisted of 48 subarrays, consisting of 56 wells that were loaded with a solution consisting of 1.2 μL cDNA solution, 1.3 μL ddH_2O, and 2.5 μL Taqman® OpenArray® Real-Time PCR Master Mix (Applied Biosystems) using the QuantStudio 12K Flex Accufill System (Applied Biosystems) to reduce intra-assay variation. The 56 wells were preloaded with primers and probes, as described above (please see Supplementary Materials), to quantify gene transcription at all target genes within each sample. Each chip could therefore process 48 samples with each of the 28 candidate genes run in duplicate.

General statistical approach

We plotted all statistical models for visual inspection of homogeneity (residual vs. fitted graphs), and normality (residual quantile–quantile plots). All datasets that did not meet these assumptions underwent Box–Cox transformation and were then reassessed. All statistical analyses were completed using R software, version 3.6.0 (R Core Team 2019).

Egg cortisol analysis

We tested for differences in egg cortisol concentrations from our cortisol versus control treatments. The data underwent a Box–Cox power transformation (Osborne 2010), using the *boxcox* function in the *MASS* package ($\lambda = 0$; Venables and Ripley 2003), to normalize the dataset. The effect of the cortisol treatments on post-fertilized eggs was then analyzed using a linear model (LM) with the cortisol treatment groups as fixed effects.

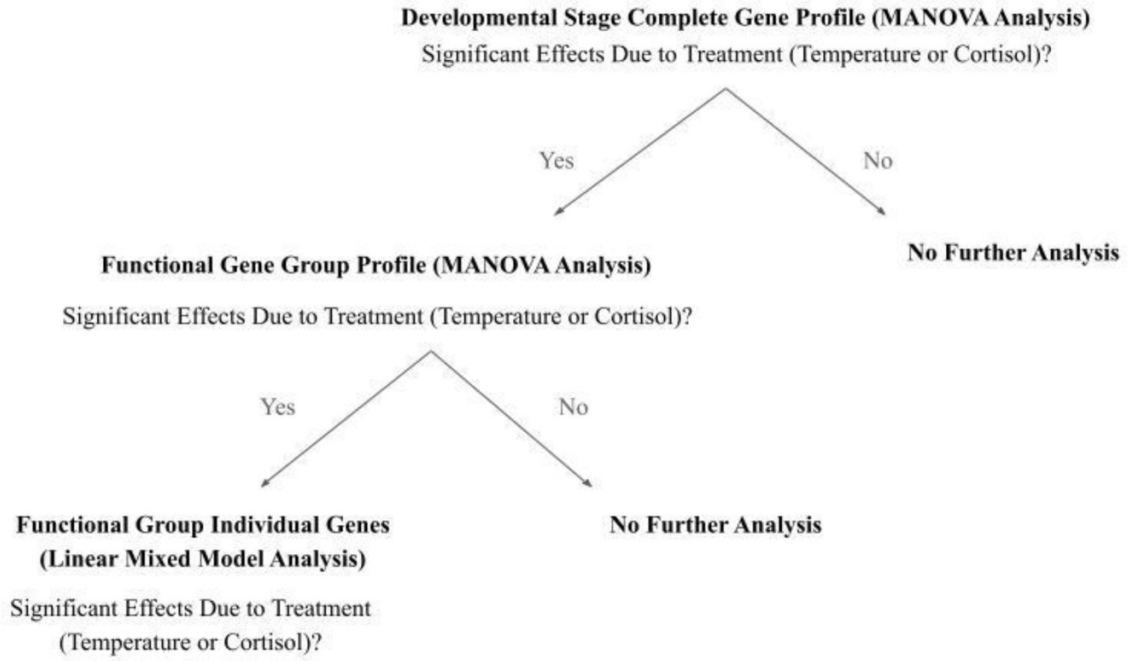
Treatment analysis of candidate gene transcription

Prior to all gene transcription analysis, the target gene transcription data were standardized based on relative primer efficiencies using LinRegPCR software, to determine relative individual gene Cq (Tuomi et al. 2010). All target gene transcription levels underwent a Box–Cox power transformation to normalize the data (Osborne 2010) for linear mixed models (LMMs), to ensure that data were as normal as possible within individual candidate genes. All target gene transcription data underwent the previously described Box–Cox transformation ($\lambda = 0$) for multivariate Analysis of variance (MANOVA) to ensure variance was due to similarities in response and not variation due to differing transformations. Sample sizes differed across genes due to mortality, or due to samples being removed if they exceeded the acceptable maximum for Cq (i.e., >30 cycles; Wellband et al. 2018).

To analyze the effects of our environmental stressors on gene transcription of our candidate genes, we took a multistage approach. First, to assess the interactive effects of both elevated T_w and cortisol treatments were evaluated within the developmental stages against the controls for both treatments. Second, to assess the impacts of elevated T_w on development we examined the effect of rearing temperature on gene transcription within developmental stages within the control group (i.e., eggs that did not receive cortisol exposure). Finally, to assess the context-dependent impact of exposure to an early maternal signal of stress in relation to water temperature we examined the effects of cortisol exposure at specific developmental stages within each water temperature treatment. This combined approach allowed us to first establish whether any interactive or individual treatment effects existed, to what degree, if any, elevated rearing temperature could induce changes in our candidate gene transcription, and then the potential for exposure to prenatal cortisol to alter the transcriptional responses within each of the two temperature regimes.

Taking a hierarchical approach to evaluating the effects of elevated rearing temperatures and prenatal cortisol (see Fig. 1), we performed separate MANOVA for all target genes ("profile") within each developmental stage. To test for temperature effects, all non-cortisol (control) individuals, across both current ambient and elevated rearing temperatures, were subdivided by developmental stage. To test for cortisol effects, all developmental stages were divided into two separate datasets based on rearing temperatures to evaluate cortisol's capacity to modulate gene transcription under current ambient and elevated thermal environments. All candidate genes with sufficient numbers of samples were then included in the MANOVA as dependent variables to be compared for correlational effects of our independent variable (i.e., temperature treatment or prenatal cortisol). Female ID and tray ID nested within female ID were not significant independent variables, and were therefore removed from the final models. Any developmental stages that showed significant responses to temperature or prenatal cortisol then underwent MANOVA for each functional gene group (i.e., growth, metabolism, immune, and stress) to identify whether one or more functional gene groups were being significantly more affected by the independent variable than others. Any functional gene groups significantly affected by temperature or cortisol then underwent individual candidate gene analysis, using linear mixed modelling (LMM), to determine whether their transcriptional phenotypes were being significantly modulated by temperature or cortisol. Each LMM contained either temperature or cortisol, depending on which treatment was significantly altering correlational responses at the functional gene group level, as a fixed effect. To account for residual effects due to possible maternal differences, incubation position, and treatment effects, the random effects were all nested within female ID and included tray ID, cortisol treatment replicate nested within cortisol treatments, and cell replicate. Random

Fig. 1. Hierarchical protocol design evaluating the individual treatment effects on gene transcriptional variation in Chinook salmon (*Oncorhynchus tshawytscha*) offspring originating from the Credit River, Lake Ontario, Ontario, Canada. Only treatment effects from each hierarchical stage that significantly affected gene transcription carried on to the next level of evaluation. MANOVA, multivariate Analysis of variance.



effects found to account for no residual variance were kept in the model to reduce the risk of falsely removing a variance component that is approaching zero, but not equal to it (Matuschek et al. 2017).

Survival analysis

Mortality data collected throughout development were converted to a binary dataset using the buildBinary function in the *fullfact* package (Houde and Pitcher 2016). The package assigned each deceased individual with a value of 0 and all living individuals with a value of 1. To analyze the effects of prenatal stress and rearing temperatures on survival during early development, we ran general linear mixed models using a binomial distribution with temperature treatment, cortisol treatment, and developmental stage as fixed effects, examining all of them individually and in three two-way interactions. The model also included female ID, egg treatment nested within female ID (to control for the replicates within female), and cell-position nested within tray as random effects. To test the fixed effects, we used likelihood ratio tests (LRT) by fitting full models for survival with maximum likelihood (ML) estimation to determine significance of the interactions by comparing the full models, with the interactions, to one that did not contain the interaction (Zuur et al. 2009). If no significant interactions were found ($p > 0.05$), we removed the interactions from the model and we sequentially tested the fixed effects again using LRT with ML estimation. All significant interactions ($p \leq 0.05$) remained within the model and underwent restricted maximum likelihood estimation, and pairwise analysis within each developmental stage using false discovery rates (FDRs) in the *emmeans* package (Pike 2011).

Results

Egg cortisol treatment

Cortisol treated eggs ($n_{cort} = 8$) had significantly higher cortisol concentrations than the non-cortisol treated control eggs (LM: $t = -6.7$, $p < 0.001$; cortisol: 46.6 ± 12.8 ng·g^{-1}, control: 8.1 ± 4.4 ng·g^{-1}; mean ± SEM). Cortisol levels in the dosed group were comparable to literature-reported levels examining relevant environmental maternal stress signals in Pacific salmonids (Capelle et al. 2016; Sopinka et al. 2016; Currier 2021; Sturba 2021).

Temperature and prenatal cortisol effects on candidate gene transcription

Examination of complete gene sets for our three developmental stages of interest (i.e., eyed, alevin, and fry) indicated that no interactive effects of the cortisol and elevated water temperature treatments existed at any developmental stage (Table 1). However, elevated temperature significantly impacted candidate gene transcriptional responses at every developmental stage in the interactive model. Additionally, the cortisol treatment had persistent marginal significance at the alevin and fry developmental stages. These results warranted further analysis. To increase the power of the model to allow for greater specificity in evaluating the impact of these individual treatments, the interaction was removed.

Table 1. Inter-stage differences in the interactive effects of an elevated egg cortisol and water temperature treatment (vs. controls) on the overall gene transcriptional profile responses for Chinook salmon (*Oncorhynchus tshawytscha*) offspring originating from the Credit River, Lake Ontario, Ontario, Canada.

Fixed effect	MANOVA results	Eyed	Alevin	Fry
Cortisol	Pillai trace	0.16	0.41	0.38
	F-value	0.48	1.59	1.56
	df and error df	(1, 17)	(1, 20)	(1, 21)
	p-value	0.95	0.097	0.097
Temperature	Pillai trace	0.72	0.63	0.51
	F-value	6.41	3.79	2.72
	df and error df	(1, 17)	(1, 20)	(1, 21)
	p-value	**<0.001**	**<0.001**	**0.0016**
Cortisol:temperature	Pillai trace	0.14	0.42	0.24
	F-value	0.40	1.66	0.79
	df and error df	(1, 17)	(1, 20)	(1, 21)
	p-value	0.98	0.080	0.72

Note: MANOVA, multivariate Analysis of variance.

Table 2. Inter-stage differences in the impacts of an elevated water temperature treatment (vs. current (control) water conditions) on the all gene transcriptional profile responses for Chinook salmon (*Oncorhynchus tshawytscha*) offspring originating from the Credit River, Lake Ontario, Ontario, Canada.

Fixed effect	MANOVA results	Eyed	Alevin	Fry
Temperature	Pillai trace	0.81	0.77	0.70
	F-value	4.40	3.26	1.56
	df and error df	(1, 18)	(1, 18)	(1, 15)
	p-value	**0.0070**	**0.0080**	0.19

Note: MANOVA, multivariate Analysis of variance.

Examination of complete gene sets for our three developmental stages of interest for elevated water temperature treatment alone significantly impacted candidate gene transcriptional responses at the eyed and alevin stages compared to control temperatures (Table 2). At the eyed stage, all functional gene groups (i.e., growth, metabolism, immune, and stress) were significantly affected by the elevated temperature treatment (Table 3). Following this hierarchical evaluation, examination of individual gene transcriptional analysis for the candidate genes used to evaluate functional gene group responses to the elevated temperature treatment indicated that at least one candidate gene from each functional group was significantly affected at the eyed stage (Table 4). Overall, development under elevated water temperatures reduced all candidate gene transcriptional responses at the eyed-egg developmental stage compared to current ambient water temperature conditions. Similarly, gene transcriptional profiles for the metabolic functional gene group at the alevin stage were significantly affected by the temperature treatment (Table 3). Investigating the transcriptional responses of the metabolic functional group's individual candidate genes revealed that only the transcription of cytochrome P450 1A (CYP1A) was significantly affected by the elevated temperature treatment for the alevin developmental stage (Table 4).

To confirm that the cortisol treatment impacted gene transcription, and then examine whether exposure to elevated prenatal cortisol mitigated the effects of the elevated water temperature treatment, we examined complete gene set transcriptional profiles for all the developmental stages within each experimental temperature regime (i.e., current T_w or elevated T_w). Under the current T_w treatment, exposure to elevated egg cortisol had a significant effect on the complete gene set profile at the alevin developmental stage, and a marginal effect at the fry developmental stage (Table 5). However, under predicted elevated T_w rearing conditions the same exposure to elevated egg cortisol did not significantly affect any developmental stage's complete gene set profile (Table 5). Further evaluation of functional gene groups within the elevated T_w treatment group for both the alevin and fry developmental stages identified no significant differences within any functional gene group in response to the prenatal cortisol treatment (Table 6).

Survival analysis

Survival analyses indicated that there was no significant interaction effect between temperature and cortisol treatments (LRT: $\chi^2 = 0.0038$, $p = 0.95$; Fig. 2), or between cortisol treatment and developmental stage ($\chi^2 = 0.82$, $p = 0.84$). However, there was a significant interaction between temperature treatment and developmental stage on survival ($\chi^2 = 9.8$, $p = 0.0074$). Reviewing the summary of the model, there were significant differences in survival across the three developmental stages ($p < 0.001$ all stages). After performing a FDR post hoc analysis on the temperature effects within each developmental stage, there was a significant difference in survival due to temperature at the eyed stage ($q = 0.015$) with lower survival under elevated compared to current ambient thermal conditions.

Table 3. Inter-stage differences in the impacts of an elevated water temperature treatment (vs. current (control) water conditions) on transcriptional responses of functional gene groups in eyed-egg and alevin Chinook salmon (*Oncorhynchus tshawytscha*) offspring originating from the Credit River, Lake Ontario, Ontario, Canada.

Fixed effect	Functional group	MANOVA results	Eyed	Alevin
Temperature	Growth	Pillai trace	0.18	0.085
		F-value	4.42	1.23
		df and error df	(1, 39)	(1, 40)
		p-value	**0.019**	0.31
	Metabolism	Pillai trace	0.52	0.33
		F-value	5.31	2.96
		df and error df	(1, 34)	(1, 42)
		p-value	**<0.001**	**0.013**
	Immune	Pillai trace	0.16	0.043
		F-value	5.46	1.28
		df and error df	(1, 59)	(1, 57)
		p-value	**0.0067**	0.29
	Stress	Pillai trace	0.41	0.22
		F-value	3.62	1.55
		df and error df	(1, 31)	(1, 33)
		p-value	**0.0078**	0.19

Note: MANOVA, multivariate Analysis of variance.

Discussion

Our study examining the impacts of early-life stressors on the transcriptional responses of developing Chinook salmon was an experimental approach to testing the environmental-matching hypothesis (Sheriff and Love 2013), and it had three primary goals. First, to examine whether exposure to elevated (compared to current ambient) water temperatures modified the transcriptional phenotypes of offspring at three distinct developmental stages. Second, to determine whether a prenatal stress signal had the capacity to further modify the transcriptional phenotypes of offspring already responding to the chronic hyperthermal challenge. Finally, we examined whether receiving a hormonal signal of future life stressors rescued thermally induced reductions in survival. We identified effects of chronically elevated temperatures on gene transcriptional profiles for candidate genes at all levels of the hierarchical evaluation (i.e., complete gene sets, functional gene groups, and individual gene transcriptional phenotypes). Under current ambient thermal conditions, exposure to prenatal cortisol generated both significant and marginally significant modifications to the complete gene set profiles at the alevin and fry stages, respectively. However, at elevated water temperatures this same prenatal cortisol exposure did not result in any of these transcriptional responses. Finally, while elevated temperatures significantly increased mortality at the eyed stage, exposure to prenatal cortisol failed to "rescue" this temperature-induced offspring mortality, in contrast to predictions of the environmental-matching hypothesis (Sheriff and Love 2013). Overall, our results examining transcriptional plasticity in response to early-life stressors confirm the important effects of temperature on transcriptional responses and survival, and also offer new insight into the potential for exposure to maternal stress to alter these responses. Below we examine these responses within the framework of environmental change in general, the environmental-matching hypothesis specifically, and compare our results to those of recent studies to shed light on the capacity of Chinook salmon (and possibly other temperate-water fish species) to respond to climate change.

Predicted increases in water temperature significantly modulate gene transcriptional profiles

As expected, we found gene transcriptional profiles across multiple functional gene groups showed significant responses to elevated temperature at the eyed-egg and alevin developmental stages. Within these stages, the candidate genes used as biomarkers for alterations to the functional gene groups metabolism (both eyed and alevin), growth (eyed only), immune function (eyed only), and stress (eyed only) had significantly different overall gene transcriptional profiles. When examining the candidate genes at the individual level for variation in transcriptional phenotypes, we found significant decreased transcription of at least one candidate gene within each functional group under elevated T_w treatment. These results could indicate a more energy-efficient (i.e., possibly adaptive) cellular response to the thermal challenge to reduce the allostatic load of the individual (Warriner et al. 2020*a*). Elevated thermal rearing temperatures may be acting as a priming signal for these early developmental stages, altering overall gene transcription to optimize the intensity of the response, reflected by alterations to individual candi-

Table 4. Stage by functional gene group differences in the impacts of an elevated water temperature treatment (vs. current (control) water conditions) on individual candidate gene transcription in Chinook salmon (*Oncorhynchus tshawytscha*) offspring originating from the Credit River, Lake Ontario, Ontario, Canada.

Developmental stage	Functional group	Candidate gene	*F*-value	*p*-value
Eyed	Growth	GH-R	$F_{(1,39}$= 0.24	0.63
		p53	$F_{(1,35}$= 22.5	**<0.001**
	Metabolism	COI	$F_{(1,25}$= 5.24	**0.031**
		CPT1	$F_{(1,47}$= 7.76	**0.0076**
		CYP1A	$F_{(1,10}$= 14.2	**0.0036**
		ELOVL7	$F_{(1,42}$= 2.30	0.14
		FAS	$F_{(1,50}$= 6.82	**0.012**
		HK1	$F_{(1,4}$= 12.9	**0.022**
		INSIG1	$F_{(1,5}$= 0.30	0.61
	Immune	CAL	$F_{(1,6}$= 5.15	0.058
		NKEF	$F_{(1,36}$= 5.84	**0.021**
	Stress	GPx1	$F_{(1,10}$= 0.087	0.77
		GR-2	$F_{(1,31}$= 8.41	**0.0068**
		hsp70	$F_{(1,52}$= 9.35	**0.0035**
		hsp90a	$F_{(1,44}$= 5.96	**0.019**
		metA	$F_{(1,11}$= 10.5	**0.0072**
		TrxR3A	$F_{(1,47}$= 13.7	**<0.001**
Alevin	Metabolism	COI	$F_{(1,9}$= 2.90	0.12
		CPT1	$F_{(1,48}$= 0.088	0.77
		CYP1A	$F_{(1,59}$= 9.40	**0.0033**
		ELOVL7	$F_{(1,41}$= 0.24	0.63
		FAS	$F_{(1,9}$= 0.056	0.82
		HK1	$F_{(1,5}$= 2.16	0.20
		INSIG1	$F_{(1,7}$= 1.37	0.28

Note: All candidate genes that were significantly different due to the elevated water temperature treatment showed reductions in gene transcription.

Table 5. Inter-stage differences in the impacts of an elevated egg cortisol treatment (vs. current (control) water conditions) on the overall gene transcriptional profile responses in relation to an elevated water temperature treatment for Chinook salmon (*Oncorhynchus tshawytscha*) offspring originating from the Credit River, Lake Ontario, Ontario, Canada.

Fixed effect	MANOVA results	Eyed (current)	Alevin (current)	Fry (current)	Eyed (elevated)	Alevin (elevated)	Fry (elevated)
Prenatal cortisol elevation treatment	Pillai trace	0.25	0.70	0.66	0.33	0.68	0.54
	F-value	0.27	2.46	2.00	0.38	0.54	0.96
	df and err df	(1, 14)	(1, 21)	(1, 17)	(1, 13)	(1, 5)	(1, 17)
	p-value	0.99	**0.023**	**0.076**	0.97	0.85	0.54

Note: MANOVA, multivariate Analysis of variance.

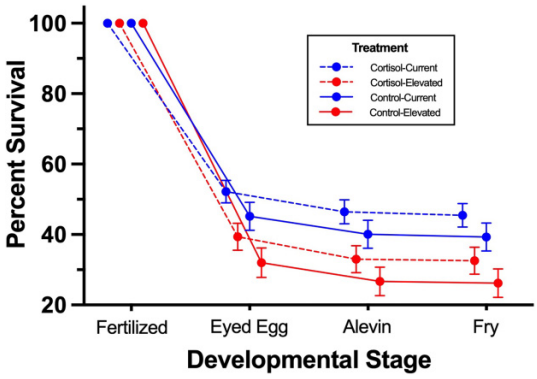
date gene transcriptional profiles. While more work linking responses to performance and fitness is needed, these group differences could represent an example of an adaptive response across functional gene groups since offspring sampled at these stages would have survived the initial high mortality in response to elevated temperatures at the eyed-egg stage, suggesting that they may be exhibiting greater thermal competency. Indeed, fish have the capacity to acclimate to their thermal environments (*Gambusa holbrooki*, *Danio rerio*, *Gasterosteus aculeatus*, Beaman et al. 2016; *Acanthochromis polycanthus*, Donelson et al. 2011; *Salmo clarki*, *Cyprinodon nevadensis amargosae*, *Etheostoma lepidum*, *Cyrinodon macalrius*, Hutchison and Maness 1979; *Notropis anogenus*, Potts et al. 2021), which may explain why the fry developmental stage did not show significant alterations to their gene transcription profiles in response to the thermal challenge. Once they had achieved that stage in their development, fish may have acclimated to the chronic increase in water temperature as a "normal" or "expected" environmental condition, and possibly no longer as an acute perturbation or stressor.

Table 6. Developmental stage by functional gene group differences of the impacts of the interaction between an elevated egg cortisol exposure (vs. control) and elevated water temperature (vs. current (control) water conditions) and on individual candidate gene transcription in Chinook salmon (*Oncorhynchus tshawytscha*) offspring originating from the Credit River, Lake Ontario, Ontario, Canada.

Fixed effect	Functional group	MANOVA results	Alevin (current)	Fry (current)
Prenatal cortisol elevation treatment	Growth	Pillai trace	0.014	0.059
		F-value	0.15	0.45
		df and error df	(1, 41)	(1, 36)
		p-value	0.96	0.81
	Metabolism	Pillai trace	0.15	0.16
		F-value	1.16	1.25
		df and error df	(1, 47)	(1, 46)
		p-value	0.35	0.30
	Immune	Pillai trace	0.019	0.031
		F-value	0.27	0.46
		df and error df	(1, 41)	(1, 43)
		p-value	0.85	0.71
	Stress	Pillai trace	0.080	0.12
		F-value	0.56	0.97
		df and error df	(1, 39)	(1, 41)
		p-value	0.76	0.45

Note: MANOVA, multivariate Analysis of variance.

Fig. 2. Inter-stage variation in survival of Chinook salmon (*Oncorhynchus tshawytscha*) offspring originating from the Credit River, Lake Ontario, Ontario, Canada, in response to the combined stressors of prenatal cortisol exposure and elevated water temperature. Red lines represent elevated thermal water conditions and blue lines indicate current ambient water conditions. Dashed lines indicate prenatal cortisol exposure treatment and solid lines indicate controls (vehicle only). Means ± SEM are represented.



Elevated egg cortisol did not alter transcriptional activity under elevated temperatures

We exposed Chinook salmon eggs to an exogenous cortisol treatment to mimic a natural prenatal stress signal that offspring might receive from a mother experiencing stressful migratory and/or breeding conditions (Warriner et al. 2020*b*; Currier 2021; Sturba 2021). Based on predictions of the environmental-match hypothesis (Sheriff and Love 2013), we expected the exposure to remediate transcriptional responses to elevated T_w conditions. We first confirmed that exposure to prenatal cortisol under current T_w conditions generated significant and marginally significant effects on gene transcription across all candidate genes in alevin and fry, respectively. These results support previous work that has identified significant alterations to early life phenotypes in fish in response to prenatal cortisol exposure but under benign (i.e., control) thermal conditions (McCormick 2006; Nettle and Bateson 2015; Capelle et al. 2016; Sopinka et al. 2016; Warriner et al. 2020*a*, 2020*b*). Interestingly, exposure to elevated egg cortisol under current ambient water temperatures had no impact on gene transcriptional profiles at the eyed-egg stage. However, there were significant effects observed at the alevin and fry stages. It is possible that maternal stress signal resulted in a pre-programmed response of the offspring under specific conditions to alter gene transcriptional responses based on the current developmental stage:environmental interaction (Lazaro-Côté 2023). The transcriptional effects of elevated cortisol seen under current ambient water conditions were not observed under elevated thermal conditions, with offspring exposed to the elevated T_w treatment exhibiting no significant differences in gene transcriptional profiles at any developmental stage regardless of cortisol exposure. These results therefore did not match predictions from the environmental-match hypothesis (Sheriff and Love 2013), and in contrast suggest exposure to elevated cortisol during early development did not play a role in allowing developing Chinook salmon offspring to better respond to elevated water temperatures.

Egg cortisol concentrations in Pacific salmonids (and other species) have been observed to significantly decrease 24 h

post-fertilization (Capelle et al. 2016; Sopinka et al. 2017). What may be more probable than transcriptional responses being altered due to a prenatal signal (that is no longer present at these later developmental stages) would be that functional physiological thresholds are being compensated for to overcome possible detrimental alterations produced at the earliest developmental stage (Eriksen et al. 2006; Connon et al. 2018; Thayer et al. 2018). These alterations may have occurred similarly for both eyed-egg developmental groups across current ambient and elevated water temperatures, since the maternal stress signal would have been identical, resulting in no significant difference at the eyed-egg stage. However, the surrounding environmental conditions (e.g., water temperature) would act as a more immediate indicator of the appropriate levels of response, which could explain the aforementioned transcriptional expression compensations at the alevin and fry developmental stages. As for the offspring developing under elevated thermal rearing conditions, we did not observe any significant effects of prenatal cortisol on all gene transcriptional profiles at any developmental stage. While the current work does not support a preparative role for elevated egg cortisol under the environmental-matching hypothesis, it is certainly possible that the maternal stress signal we used did not match the signal expected for the level of environmental stress for elevated thermal conditions we chose, meaning the cortisol exposure was at an appropriate level to alter gene transcriptional profiles in response to predictive future climactic conditions.

Exposure to prenatal cortisol did not rescue warm water effects on survival

Based on previous research, we expected a significant negative effect of the elevated T_w treatment on early survival across development stages, with exposure to elevated prenatal cortisol potentially buffering (and even rescuing) those effects. Indeed, there has been considerable research that has identified the negative effects of temperature on early survival in larval fish (Murray and McPhail 1988; Réallis-Doyelle et al. 2016; Campos et al. 2019). We confirmed that elevated T_w resulted in a significant increase in mortality rates, but only within the eyed stage. Eyed eggs are well known to be extremely sensitive to environmental perturbations (Myrick and Cech 2004; Pankhurst and King 2010), which may account for why we observed the highest mortality rates occurring at this stage in response to our elevated T_w treatment (Dahlke et al. 2020). Interpreting the impacts of elevated prenatal cortisol on survival is complex because prenatal stress signals have been shown to both positively and negatively impact early mortality rates across multiple taxa (Capelle et al. 2016; Love et al. 2005; McCormick 1998; Love and Williams 2008; Meylan and Clobert 2005). In Chinook salmon in particular, exposure to similar doses of prenatal cortisol as in our study has been shown to improve survival under benign thermal conditions (Capelle et al. 2016), as well as negatively impact survival when paired with elevated incubation temperatures (Warriner et al. 2020*a*). Despite these previous findings and in contrast to our predictions, neither exposure to exogenous prenatal cortisol or the interaction between cortisol exposure and temperature treatments impacted early survival in this study. As such, our results do not support prenatal stress signals acting as a potential mediator that improves offspring survival outcomes under elevated rearing temperatures. Our observed lack of a "rescue effect" for prenatal cortisol under chronically elevated thermal rearing conditions could be due to several factors, including that the prenatal cortisol signal's ability to modify the offspring's response to elevated temperatures might be overwhelmed by the negative effects of elevated temperature. The lack of an impact of cortisol exposure itself on survival is interesting, but in the current study may have resulted from a much larger impact of elevated T_w swamping out smaller effects of exposure to cortisol.

Implications for the management of Pacific salmonids

Many fish species are already being negatively affected by elevated water temperatures (Free et al. 2019) and wild fish populations are in a significant decline on a global scale (Dulvy et al. 2006; Pauly and Zeller 2016; Pacoureau et al. 2021), with climate change effects thought to be a contributing factor (Cheung et al. 2021). Salmonids, including Chinook salmon, are among the species that have seen numbers dwindle to the point of some populations being listed as endangered or threatened (Albaugh et al. 2011; Crozier 2016). Early developmental stages are increasingly exposed to fluctuating thermal conditions due to the greater daily and seasonal variation in temperature in river systems relative to larger bodies of water such as lakes or oceans (Webb and Nobilis 2007; Van Vliet et al. 2011). Both environmental variation (Melack et al. 1997; Carter 2005; Whitney et al. 2013) and maternal stress signals (Sheriff and Love 2013; Bateson et al. 2014) have been shown to significantly modulate offspring development. Our results highlighted a diversity of impacts from elevated water temperatures on transcriptional responses across a diversity of genes and confirmed negative impacts of these elevated temperatures on early egg survival. These alterations may be indicative of possible predictive–adaptive responses to important functional systems due to the temperature stressor itself, although more work linking responses to performance and fitness are still needed. Regardless, we found that early possibly preparatory maternal stress signals did not have the capacity to affect gene transcriptional profiles enough to enable early developmental stages to better respond to these temperature effects in Chinook salmon. This lack of significant alterations to functional groups or individual genes expression under elevated water temperatures could be an indicator that native and naturalized populations of Pacific salmonids may be unable to use additional plasticity to alter gene expression significantly, even when a possible maternal stress signal would indicate a need for such a response, or that the prenatal cortisol we chose was not at the appropriate signal strength necessary to elicit alterations in plasticity under elevated water temperature conditions. More work is needed to examine the possibility that later life history stages might have significantly different gene transcriptional responses to extreme environmental conditions (e.g., acute hyperthermal

fluctuations) when reared under these same conditions. Studies examining whether these differences in functional gene transcription persist into later life history stages need to be evaluated to determine whether these modulations to gene transcription are indeed possible examples of adaptive cellular response.

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Data availability

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Competing interests

The authors declare there are no competing interests.

Supplementary material

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