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Prenatal stress as a potential adaptive modulator of offspring survival, phenotype, and performance in response to elevated temperatures

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By

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DECLARATION OF CO-AUTHORSHIP

I hereby declare that this thesis incorporates material that is result of joint research, as follows: I am the sole author on Chapters 1 and 4 and the primary author on all other chapters (Chapter 2 and 3). Chapter 2 to 3 are co-authored with my co-supervisors Dr. Oliver Love and Dr. Christina Semeniuk and my collaborators Dr. Trevor Pitcher. In all cases, the key ideas, primary contributions, experimental designs, data analysis, interpretation, and writing were performed by the author, and the contribution of co-authors was primarily through the assistance with experimental design, data analysis, interpretation, logistical support, editing and funding. Chapter 3 is also co-authored with Dr. Daniel Heath who assisted with experimental design and interpretation.

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ABSTRACT

Climate change is increasing global water temperatures, and by altering temperatures, is subsequently impacting aquatic life, particularly ectothermic fish. When mothers encounter environmental stressors such as elevated temperatures during follicular recruitment (maternal stress), resultant offspring often have altered phenotypes. Recent studies suggest that this maternal stress signal may prepare offspring for a similarly stressful environment (environmental match). I applied the environmental match hypothesis to investigate whether a maternal stress signal can prepare offspring to cope in a stressful environment. Specifically, I exposed Lake Ontario Chinook salmon (*Oncorhynchus tshawytscha*) eggs to a biologically relevant maternal stress signal (1000ng/mL cortisol or control). We split and reared these dosed groups at temperatures indicative of current and future temperature conditions (+3°C). Then we investigated the interactive effects of thermal and maternal stress on offspring early survival, early morphology, and thermal tolerance (i.e., CTMax and energetic responses to temperature spikes). Overall I found that rearing temperature was an overriding modulator of offspring phenotype and performance. Offspring raised in elevated temperatures had lower early survival, were smaller and had a higher acute tolerance. Prenatal cortisol did not definitively modulate offspring phenotype or performance under elevated rearing temperatures. My thesis provides evidence for the role of predictive adaptive responses, as offspring who were reared in elevated temperatures had higher thermal tolerance at parr stage. Applying the environmental context of intergenerational plastic mechanism such as maternal stress remains imperative as the world continues to rapidly change under the effects of human-mediated climate change.

DEDICATION

To my Mum, my Dad, and my siblings Elisabeth and Natalie;

Who have always loved me and supported me.

To my Opa,

Who taught me the importance of family and nature.

And to my furballs Luna and Sapphira

Who bring me joy everyday.

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CHAPTER 1 — GENERAL INTRODUCTION

Environmental stress

Environments are dynamic, and experience normal fluctuations daily, seasonally, and annually, whether this is due to extreme weather events (e.g. floods, droughts), seasonal changes, or climatic fluctuations (e.g. El Nino). Organisms within these dynamic environments can respond to this environmental variation using phenotypic plasticity, behavioural flexibility, and contemporary evolution in order to persist (Fusco & Minelli, 2010; Piersma & Drent, 2003). These responses can lead to changes in organism phenotype (such as morphological, behavioural, or physiological), which in turn affect organism performance and ultimately, their fitness. Recently, humans have introduced a new category of environmental stressors we term as Human Induced Rapid Environmental Changes (HIREC), where changes in the environment are occurring faster than previously encountered. With this rapid rate of change, responses such as flexibility and plasticity within and across generations may become even more important for population persistence (Fox et al., 2019; Miner et al., 2005).

Climate change is a prevalent example of HIREC, leading to not only increased average temperatures across the globe (Stocker et al., 2013), but also contributing to extreme temperatures (Buckley & Huey, 2016), extreme weather events (Rahmstorf & Coumou, 2011), ocean acidification (Bates et al., 2008), and rising sea levels (Nicholls & Cazenave, 2010). Climate change is expected to both directly and indirectly reduce global biodiversity by species reduction and by altering community composition through changes in the ranges of warm adapted species (Parmesan & Yohe, 2003). Ectotherms are expected to be particularly susceptible to the increases in temperature since they are directly impacted by changes in their

environmental temperature (Rohr & Palmer, 2013). Higher temperatures under climate change may affect ectotherms sub-lethally by increasing their metabolism (Dillon, Wang, & Huey, 2010) and decreasing body size (Sheridan & Bickford, 2011).

Glucocorticoids as translators of environmental stress

Hormones are responsive to changes in the environment, and when individuals are exposed to both acute and chronic environmental stressors they may increase their glucocorticoid levels as a part of the stress response. Glucocorticoids (GCs) are steroid hormones involved in homeostatic energy management and in the acute stress response in vertebrates (hypothalamus-pituitary-interrenal (HPI) axis in fishes: Barton, 2002; Schreck & Tort, 2016; Wendelaar Bonga, 1997; hypothalamus-pituitary-adrenal (HPA) axis in birds, mammals, reptiles and amphibians: Moore & Jessop, 2003; Romero, 2004). Elevated GCs allow an organism to mobilize energy and elicit adaptive physiological and behavioural changes to flee or persist in the acute environmental changes (Wingfield, 2013). Once the stressor is removed, typically the circulating GC returns back to homeostatic levels. However, after prolonged (i.e., chronic) exposure to stressors, baseline GC levels may be elevated leading to effects such as slower growth (Sadoul & Vijayan, 2016), suppressed immunity (Maule et al., 1989), and delayed or altered reproduction (Schreck, Contreras-Sanchez, & Fitzpatrick, 2001).

Since climate change is expected to expose organisms to a host of new stressors, organism may respond to these environmental changes by increasing their GC levels. Higher temperatures may lead to higher energetic costs for some organisms—particularly ectotherms (Dillon et al., 2010; Somero, 2010), which then may be reflected in their baseline glucocorticoid levels. Additionally, climate change is also leading to greater environmental

extremes (e.g. droughts) (Fischer & Knutti, 2015), which can further exacerbate climatic effects on an organism's GCs. For example, marine iguanas (*Amblyrhynchus cristatus*) in an El Niño year (drought conditions) had higher corticosterone levels than a La Niña year that, in combination with body condition, predicted survival probabilities (Romero & Wikelski, 2001). Climate change may also alter the role of GCs in the stress response, as there may be an uncoupling of sensory cues of environmental stress and hormonal response due to the abruptness & frequency of environmental changes (Angelier & Wingfield, 2012). Thus, determining the role of elevated GCs in adjusting to new climatic conditions—such as their role in inducing phenotypic plasticity within and across generations—is important.

Organisms will often remain responsive to environmental stressors during reproduction with an elevation of GCs when they are faced with an acute or chronic stressor (Love, McGowan, & Sheriff, 2013). When females have elevated GCs during reproduction (i.e., maternal stress), GCs may be transferred to their offspring, either *in utero* (Matthews, 2002) or via transfer to the egg lipids in oviparous species (Sopinka et al., 2017). Maternal GCs may have important intergenerational effects, and experimental studies have reported that biologically relevant increases in egg cortisol levels induce a number of phenotypic alterations in offspring (Ensminger et al., 2018; Eriksen et al., 2006; Hayward & Wingfield, 2004). For example, experimentally increasing prenatal GCs (to mimic maternal stress) can result in smaller body size (Burton et al., 2011; Capelle et al., 2017; Love et al., 2005; but see Sopinka et al., 2016), lower growth rates (Hayward & Wingfield, 2004; Love et al., 2005; McCormick, 1998; Meylan & Clobert, 2005), modified baseline and stress response GC levels (Auperin & Geslin, 2008; Colson et al., 2015; Haussmann et al., 2012; Hayward & Wingfield, 2004; Nesan & Vijayan, 2016), and altered behavioural responses (Saino et al., 2005; Sopinka et al.,

2015). Thus increases in maternally transferred GCs provide a potential mechanism by which a maternal stress signal can pass from mother to offspring, resulting in altered offspring phenotype and performance (Love et al., 2013).

The primary glucocorticoid in fishes is cortisol (Barton, 2002; Wendelaar Bonga, 1997) and due to the lipophilic nature of cortisol, it can easily transfer from plasma in the mother to her eggs (Schreck et al., 2001; Stratholt, Donaldson, & Liley, 1997). Endogenous embryonically derived cortisol does not appear until shortly before or at hatching (Barry et al., 1995; Stouthart et al., 1998), thus only maternally-derived cortisol is present in the egg during early development (Nesan & Vijayan, 2016). GCs are thought to influence developmental pathways in offspring via glucocorticoid receptor signaling effects on transcription (Nesan & Vijayan, 2016; Pikulkaew et al., 2011), or via GC-mediated epigenetic changes (Ahmed et al., 2014; Li et al., 2010; Love et al., 2013). Eggs from stressed fish mothers have been shown to have increased egg cortisol concentrations (McCormick, 1998; Stratholt et al., 1997; but see Ghio et al., 2016), and subsequently, offspring exposed to elevated egg cortisol have altered traits and fitness (Sopinka et al., 2017). For example, Capelle et al (2017) found that egg cortisol-dosed offspring were smaller (at high dose) and had a higher survival (at a low dose). In order to manipulate cortisol levels in mothers and offspring, different direct and indirect techniques are employed to experimentally induce maternal stress: i) directly stressing the mother (e.g. chasing, air exposure); ii) increasing maternal GCs through injections, implants, or food infused with GCs; iii) injecting GCs into her gametes or embryos; or iv) bathing the embryos in a GC bath (Sopinka et al., 2017).

Maternal stress as an adaptive signal

Maternal effects are considerable drivers of offspring variation (Mousseau & Fox, 1998; Räsänen & Kruuk, 2007), thus it is important to determine whether maternal effects such as maternal stress may be contributing to variation in offspring performance and fitness (Sheriff & Love, 2013). The potential impacts of maternal stress on the developing fetus has long been recognized in biomedical research in humans (Viltart & Vanbesien-Mailliot, 2007). Indeed, the effects of maternal stress (e.g., malnourishment) during pregnancy in humans has been linked to traits such as decreased birth weight (Hobel & Culhane, 2018; Nkansah-Amankra et al., 2010). Early researchers proposed that this early life phenotype of a lower birth weight represented a 'thrifty phenotype' since a smaller body incurs less energetic cost and thus is adapted for nutritionally deficient environment (Bateson et al., 2004; Monaghan, 2008). This metabolic programming hypothesis was supported by evidence that low birth weight correlates with negative attributes (e.g., obesity, type II diabetes) when these offspring are provided with excess resources during early life (Harder et al., 2007; Yu et al., 2011). Gluckman et al. (2005) expanded this hypothesis within an adaptive framework by developing the "predictive adaptive responses" (PARs) hypothesis, which states that offspring will respond via developmental plasticity to environmental cues that will allow higher performance in the future environment. The PAR hypothesis provides a starting framework in which researchers are studying the effects of maternal stress across taxa (Breuner, 2008).

Maternal stress is a form of intergenerational plasticity that also has been shown to alter offspring morphology (e.g., smaller body size: McCormick, 1998; Meylan & Clobert, 2005), physiology (e.g., altered stress response: Redfern et al., 2017; Saino et al., 2005), and behaviour (e.g., aggression: Eriksen et al., 2006). Given the direction of these traits, the fitness

of these altered offspring traits has often been considered negative. However, these conclusions are often drawn without considering the life-history or environmental context of the organism (Love et al., 2013). From an evolutionary perspective, maternal stress may be acting as a predictive adaptive signal of expected life-history and environmental conditions to offspring (Sheriff et al., 2017), thus preparing offspring for future environments (e.g., environments such as lower maternal care: Love et al., 2005; higher predator density: Sheriff, Krebs, & Boonstra, 2009, 2010; and higher conspecific density: Dantzer et al., 2013). For example, when pregnant red squirrels (*Tamiasciurus hudsonicus*) were exposed to experimentally higher densities, they demonstrated elevated circulating plasma GCs and their offspring grew more quickly than control offspring (Dantzer et al., 2013). This increased rate of growth may allow red squirrel offspring to outcompete other juvenile squirrels for access to food or shelter.

Effects of thermal stress in ectotherms within and across generations

Ectotherms are particularly susceptible to the effects of climate change since their metabolism is dependent on ambient temperature and thus with increasing temperatures, their metabolism is also expected to increase (Dillon et al., 2010). Given this effect on metabolism, elevated temperatures can impact organism growth (Daufresne, Lengfellner, & Sommer, 2009), energetics (i.e., the HPI axis: (Quigley & Hinch, 2006; Relkin, 1989 but see Lankford, Adams, & Cech, 2003, blood metabolites: Gallant et al., 2017; Lankford et al., 2003), behaviour (Breau, Cunjak, & Peake, 2011; Crossin et al., 2008) and survival (Jeffries et al., 2012; Martins et al., 2011). Since early development is a sensitive period that can have long lasting effects on offspring phenotype (Monaghan, 2008), the effects of elevated temperatures have been rigorously studied in early development. The effect seen in offspring raised in

elevated temperatures include earlier development (Fuhrman et al., 2018; Whitney, Hinch, & Patterson, 2014), smaller bodies (Cingi, Keinänen, & Vuorinen, 2010; Sheridan & Bickford, 2011), increased metabolism (Clarke & Johnston, 1999; Enders & Boisclair, 2016), and altered behaviours (Abram et al., 2017; Kuehne, Olden, & Duda, 2012). These altered offspring phenotypes have subsequent effects on thermal performance (i.e., higher thermal tolerance: Bickford et al., 2010; Sandblom et al., 2016 but see Chen et al., 2013) and survival (Martins et al., 2012; Rohr & Palmer, 2013). Although the effects of increased temperatures in early development have been widely studied, the role of maternal stress (via GCs) in modulating offspring phenotype under increased thermal environments warrants further study.

The maternal thermal environment is also known to modulate offspring traits (via maternal effects) in fish (Burt, Hinch, & Patterson, 2011; Jonsson & Jonsson, 2016), and in reptiles (Paranjpe et al., 2013). When offspring are exposed to elevated temperatures, maternal effects still can play a role in how offspring respond to a warmer environment (Burt et al., 2011; Jonsson & Jonsson, 2014; Thorn et al., 2018). When studies examined the effects of elevated temperatures in both the parental and offspring environments in aquatic organisms (environmental matching via maternal & paternal effects) (Donelson et al., 2018), the resulting altered offspring traits were often considered advantageous (Burgess & Marshall, 2011; Donelson, Munday, & McCormick, 2012; Le Roy & Seebacher, 2018; Le Roy, Loughland, & Seebacher, 2017; Salinas & Munch, 2012; Shama et al., 2014). For example, when both the parental and offspring environments had elevated temperatures, offspring grew more quickly (Donelson et al., 2012; Shama et al., 2014), maintained higher metabolic scope (Le Roy et al., 2017), and higher swimming performance (Le Roy & Seebacher, 2018; Le Roy et al., 2017). Most studies found that when both the parental and offspring environments

experienced higher temperatures, offspring were larger (Donelson et al., 2012; Shama et al., 2014), except for one study which found that elevated-temperature-reared bryozoan (*Bugula neritina*) offspring were smaller, but also had higher dispersal capacity and metamorphic success (Burgess & Marshall, 2011). These studies highlight the potential of non-genetic maternal effects to induce rapid offspring phenotypic changes that increase performance in environments impacted by climate change (Salinas et al., 2013). Thus, examining the context of both parental and offspring environment is important to determine whether the effects of maternal stress represent an adaptive means of translating warming effects to offspring (Sheriff & Love, 2013).

Environmental Match Hypothesis

When integrating the life history and environmental context of maternal stress, recent studies suggest that maternal stress may act as an adaptive cue, allowing for offspring to prepare for a harsher environments (Sheriff et al., 2017). The environmental match hypothesis incorporates the environmental context into predictive adaptive responses (Love & Williams, 2008; Sheriff & Love, 2013), stating that if mothers and offspring encounter the same stressful environments (environmental match), a maternally-derived stress signal may prepare offspring for that stressful environment (Breuner, 2008). Through this maternal stress signal, offspring traits are altered, resulting in higher performance and fitness in the stressful environment compared to offspring not receiving this stress signal (Weber et al., 2018). One study examining the effects of prenatal stress in benign and stressful (low water conditions) postnatal environments found that low cortisol-dosed offspring had lower energetic demand, maintained a higher stress responsiveness, maintained their body size, and displayed refuge use (risk-averse behaviours) in low water conditions (Capelle, 2017). By maintaining their

stress responsiveness and being risk-averse in this stressful condition, these low cortisol-dosed offspring were able to respond to any additional stressors they encountered without incurring energetic costs (i.e. decrease in body size). Thus, offspring traits that would traditionally be considered to have negative impact on performance and fitness in a benign environment may in fact be more suitable (i.e., beneficial) in a stressful environment, reaffirming the importance of incorporating the environmental context when testing offspring performance. From an applied perspective, the environmental match hypothesis may demonstrate a method to which organisms can respond to environmental stressors induced by climate change (Sheriff & Love, 2013). Maternally-driven phenotypic plasticity has already been considered a potential mechanism for organismal responses to climate change (Meylan, Miles, & Clobert, 2012) and in theoretical models, maternal effects have been shown to increase phenotypic variance in rapidly changing environments (Kuijper & Hoyle, 2015). Thus, given the overlap between the maternal and offspring environment in many oviparous ectothermic species (i.e., fish), the effects of increasing temperatures caused by climate change on offspring may be mitigated through the signal of maternal environment.

Study system: Chinook salmon

In North America, Chinook salmon (*Oncorhynchus tshawytscha*) is a native species to the Northwest coast from Central California to Alaska (Healey, 1991). Like many other Pacific salmon, Chinook salmon populations are in decline, where six U.S. populations have been designated as threatened and one U.S. (Upper Columbia spring) and eight Canadian populations as endangered (Northwest Fisheries Science Center, 2015; COSEWIC, 2018; Ford et al., 2011). With populations already in decline, scientists and policy makers are concerned with how climate change will further affect these numbers (Crozier et al., 2008).

Higher temperatures (which are expected to occur under climate change) have been shown to result in lower survival in both laboratory (Whitney, Hinch, & Patterson, 2013) and wild (Crossin et al., 2008; Crozier et al., 2010) salmonid populations. Elevated temperatures are also expected to affect other traits, such as a higher rate of disease transmission (Ray, Holt, & Bartholomew, 2012), potential mismatch between juvenile emergence and food sources (Chittenden et al., 2010; Edwards & Richardson, 2004), and a host of phenotypic effects including impacts on growth, physiology (e.g., metabolism), and behaviour (e.g., activity and foraging: Enders & Boisclair, 2016; Kuehne et al., 2012; Martins et al., 2012). Temperature effects across generations have also been found in salmonids, such as changes in offspring phenotype (e.g., smaller egg sizes) when gravid female salmon migrated up warmer rivers (Jonsson & Jonsson, 2016).

Pacific salmon are an ideal candidate for testing the environmental match hypothesis to examine whether intergenerational effects may rescue populations from the effects of climate change for a number of reasons. First, there is a large concern about the survival of Pacific salmonid species under climate change (Crozier, 2015; Crozier et al., 2008; Kuehne et al., 2012). Second, Pacific salmon are semelparous, which means that females are under high pressure to maximize offspring survival. Thus, any offspring indicators of environmental quality should be under high selection (Kuijper & Hoyle, 2015). Third, salmon mothers and offspring share a spatial overlap of environments (i.e., the natal stream) that provides for signals from the mother during migration and spawning to be communicated to offspring about their future environmental quality. Finally, migration for these gravid female salmon is stressful causing elevations in their glucocorticoids (Baker & Vynne, 2014), lactate, and glucose (Mathes et al., 2010), while decreasing immune function (Dolan et al., 2016), and

depleting energy reserves (Hruska et al., 2010). Despite the toll that migration has on the females, studies have shown that female salmon remain sensitive to environmental stressors during migration, and may respond by elevating their GC levels even further (Cook et al., 2014). Importantly, females with elevated endogenous plasma GCs during this period may transfer these elevated GCs into eggs, generating further effects on offspring (Love et al., 2009). Indeed, physical stressors given to gravid female salmonids have been shown to increase female plasma GCs and GC levels in her eggs (Stratholt et al., 1997).

Maternal stress has been widely studied in salmonids, and there are established methods for experimentally increasing GCs in eggs (e.g., cortisol baths at fertilization, injections: Sopinka et al., 2017). However, the effects of cortisol elevation in eggs have been mainly studied in benign (i.e., control) environments. After exposure to elevated egg GCs, some studies have found that offspring are smaller (Burton et al., 2011; Capelle et al., 2017; Sopinka et al., 2016), are more dominant and bold (Sopinka et al., 2015), and have attenuated GC stress responses (Auperin & Geslin, 2008). However, there is limited information on the interactive role that prenatal GC exposure and offspring environment have on offspring phenotype, performance, and fitness. In one recent study, Capelle et al (2017) tested the effects of a prenatal cortisol dose (control, low, and high) within different post-natal environments (30-day high or low water treatment). The authors found that low cortisol-dosed offspring had lower energetic demand, higher GC responsiveness, and higher refuge use in a stressful (low water) conditions. During the riverine life stage, juvenile salmon must survive, grow, become smolts (prepare for saltwater entry), and migrate out to the ocean (or lake). There has been some evidence that delayed mortality (i.e., body condition in river stage) correlates with smolt to adult survival (Haeseker et al., 2012). Thus, maternally-derived

altercations to riverine juvenile phenotypes may have long lasting effects on individual survival, and furthermore may alter population dynamics.

Climate change in the Laurentian Great Lakes

In their native ranges, Chinook salmon are anadromous, migrating from the ocean to their natal freshwater streams in order to reproduce before dying. However, in their introduced range of the Laurentian Great Lakes, they are potamodromous and thus remain in fresh water their entire life (Parson, 1973). Chinook salmon were introduced to multiple Great Lakes through stocking efforts beginning in the 1960's (Parson, 1973). Although introduced, Chinook salmon face the same stressors as their native counterparts and may be useful models to study responses to warming fresh waters. The Laurentian Great Lakes are expected to undergo numerous human induced changes over the next century (Smith et al., 2015), with climate change being predicted to be one of the most impactful environmental stressors (Collingsworth et al., 2017). Historically, increases in surface water temperatures have been documented in the Great Lakes in the last 50 years: Lake Superior (+2.5°C), Lake Huron (+2.9°C) and Lake Ontario (+1.6°C) (Austin & Colman, 2007; Dobiesz & Lester, 2009). Several modelling studies predict higher monthly temperatures and higher maximum summer and lower minimum winter temperatures in the Great Lakes basin for the next 50 years (McDermid, Fera, & Hogg, 2015; Zhang et al., 2018, 2019). This is also reflected in tributaries and streams across the globe (Webb, 1996) and in North America (Isaak et al., 2012) which have been increasing in average temperatures, and are projected to continue increasing into the next century (Mantua, Tohver, & Hamlet, 2010; van Vliet et al., 2013). Since increasing temperatures have been shown to alter ecologically important fish habitats in

the Great Lakes, determining how fish may respond to these changes will be imperative to ascertain the effects on ecosystem health in the Great Lakes.

Thesis Objectives

My thesis aimed to test the environmental match hypothesis within a climate change scenario by exposing Chinook salmon eggs to maternal stress and raising the offspring under water temperatures predicted by climate change models. Specifically, I exposed eggs to control (0 $ng*mL^{-1}$) or cortisol-dosed (1000 $ng*mL^{-1}$) treatments after fertilization to mimic maternal stress, and raised the offspring in a current temperature regime (+0°C) based on river temperatures from their natal stream; **Figure 1.1A**) and an elevated temperature regime (+3°C) as predicted under climate change models (van Vliet et al., 2013; **Figure 1.1B**). By comparing phenotype, performance and fitness outcomes using a two by two experimental design (**Figure 1.2**), I assessed whether the interactive effects of prenatal stress and elevated water temperatures impact the ability of offspring to phenotypically match their expected future environment. Using an integrative approach, I measured survival, morphological, and physiological variation to determine offspring phenotype and performance. Overall, my goal was to determine whether maternal stress can act as a predictive signal of future environmental quality, enabling offspring to prepare for a stressful environment, in this case, elevated water temperature.

For all my thesis chapters, I used the manipulative experimental approach described in **Figure 1.2**. In Chapter 2, I assessed the effects of maternal stress (exogenous cortisol egg baths) and early rearing environment (current or elevated water temperature) on offspring early survival, size, and morphology. In Chapter 3, I examined the interaction of maternal stress and rearing temperatures on the acute heat tolerance of fry, through both behavioural-

(CTMax) and physiological (energetic metabolites) approaches following exposure to one or multiple water temperature spikes, respectively. By assessing the impacts on early life phenotype and performance of fish exposed to the interactive stressors of maternal stress and chronic elevated rearing temperatures, my thesis tests whether maternal stress can dampen the effects of within generational stress using an environmentally relevant context (Sheriff et al., 2017).

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Figures

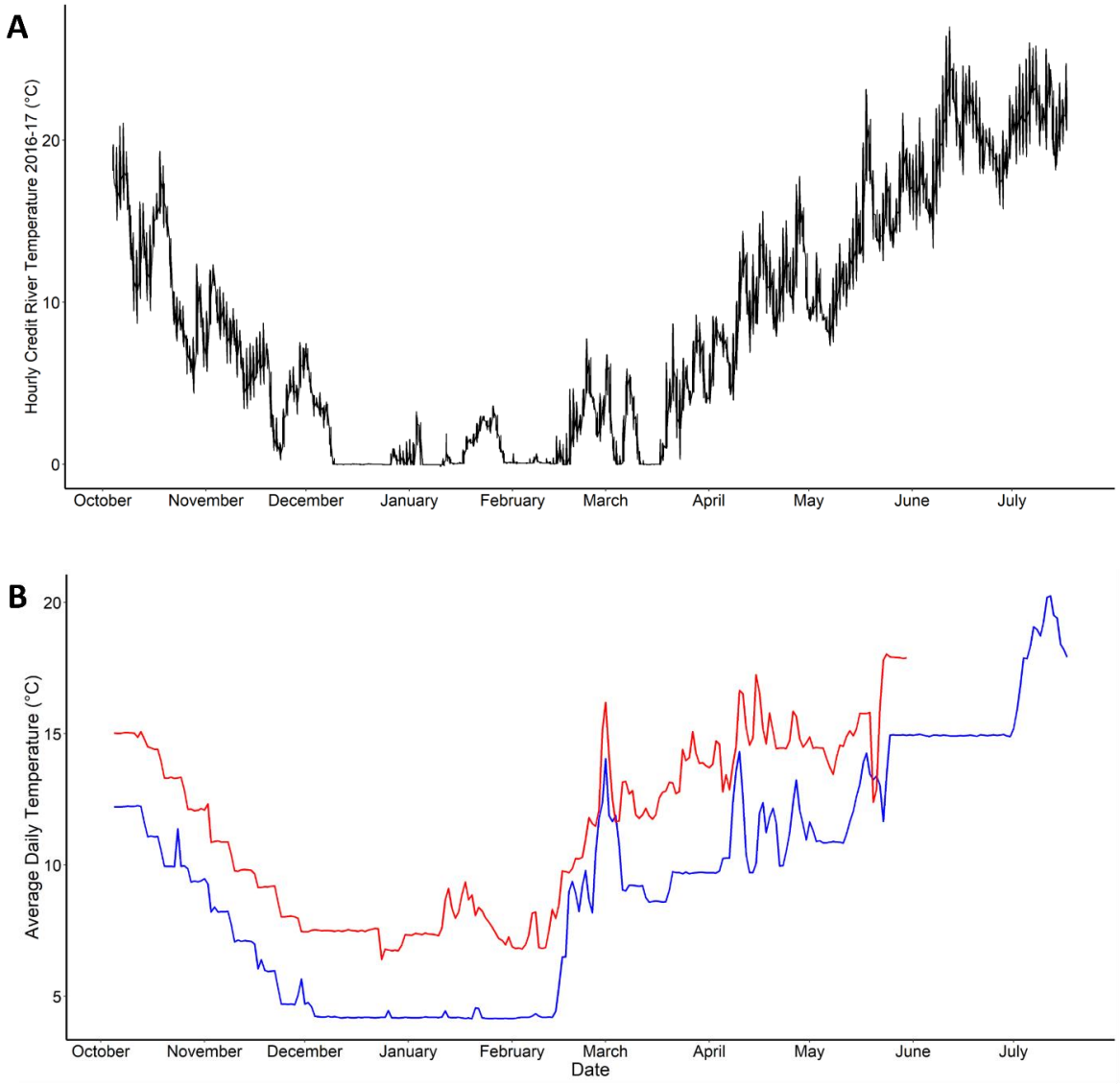

Figure 1.1: **A** Recorded hourly temperatures (HOBO®: Water temp pro v2) of the Credit River, Ontario, Canada (43°34'40.0" N 79°42'06.3" W) from Fall 2016 to Summer 2017. **B** Recorded average daily temperatures for each temperature regime in the rearing experimental design (HOBO®: Water temp pro v2). Current temperature regime is represented by the blue line while the elevated temperature regime is in red.

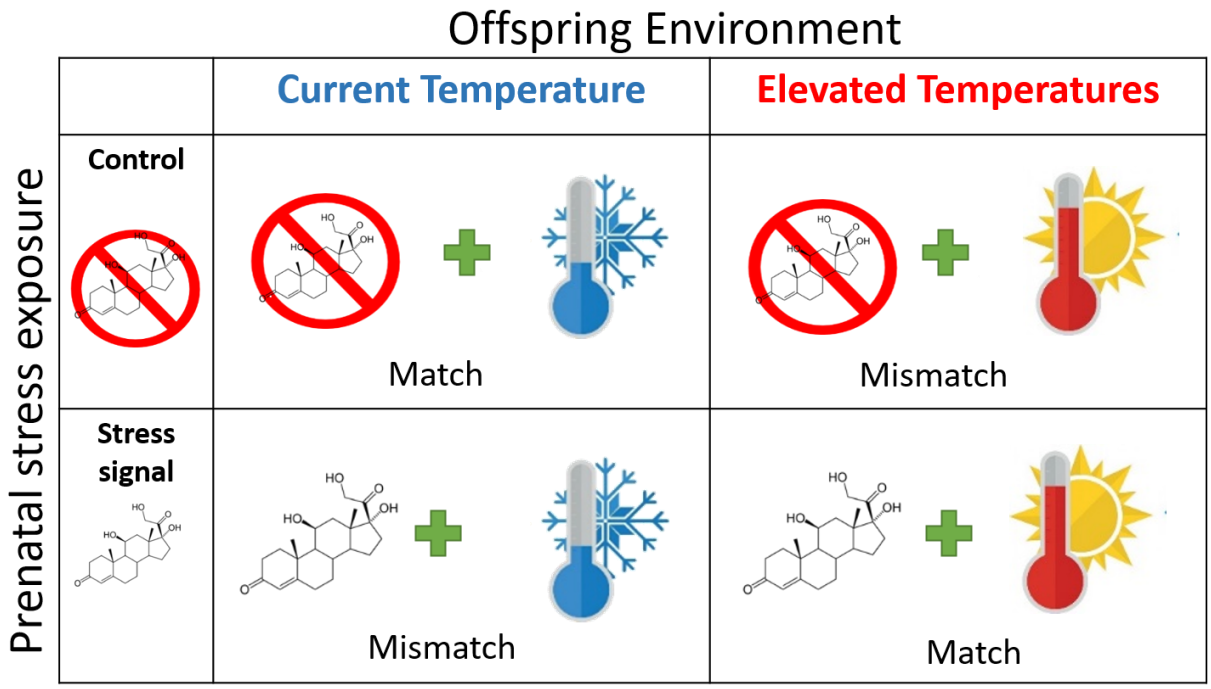

Figure 1.2: Graphical representation of experimental design to test the environmental match hypothesis within the interaction between thermal and maternal stress. When there is a match between the maternal (prenatal) environment and the offspring environment, offspring should have a higher relative performance. Thus, offspring that receive a prenatal cortisol signal should have higher performance than control offspring, within the elevated temperature conditions.

CHAPTER 2 — EXPOSURE TO MATERNAL STRESS DOES NOT RESCUE JUVENILE CHINOOK SALMON BODY SIZE, CONDITION OR SURVIVAL FROM THE EFFECTS OF ELEVATED WATER TEMPERATURES

Introduction

Climate change is altering habitats across the globe, introducing environmental stressors such as elevated mean temperatures (Stocker et al., 2013), increased frequency of extreme weather events (e.g., floods, droughts) (Easterling et al., 2000; Fischer & Knutti, 2015), and novel competitor- and predator interactions (e.g., via species range expansions towards the poles) (Parmesan & Yohe, 2003). An unique characteristic of climate change is the accelerated rate at which change occurs (Loarie et al., 2009), consequently affecting the capacity of organisms to respond adaptively (Palmer et al., 2017; Urban et al., 2016; Woodward et al., 2016). Organisms may respond to environmental stressors through phenotypic changes in their growth, morphology, reproduction, and survival (Barton, 2002), which can include rapid-acting mechanisms within and across generations such as phenotypic plasticity, phenotypically flexible responses, and contemporary evolution (Hendry, Farrugia, & Kinnison, 2008; Sih, Ferrari, & Harris, 2011). Climate change is expected to have particularly strong effects on aquatic systems, where altercations in the hydrological cycle (e.g., changes in precipitation) leading to more extremes in water availability (i.e., floods and droughts), increases in air temperature leading to increased water temperatures, and increased CO2 increasing ocean acidity (Bates et al., 2008), can have direct and indirect effects on aquatic organisms. Notably, increasing temperatures are expected to greatly—typically interpreted as negatively—impact ectothermic organisms such as invertebrates and fishes (Ficke, Myrick, & Hansen, 2007; Stoks, Geerts, & De Meester, 2014).

Indeed, warming waters affect offspring phenotypic traits such as smaller body size (Kuehne, Olden, & Duda, 2012; Whitney et al., 2014), faster growth (Beacham & Murray, 1990), and increased metabolism (Clarke & Johnston, 1999; Enders & Boisclair, 2016). In turn, higher temperatures affect offspring performance and life history traits such as lower thermal tolerance (Chen et al., 2013), earlier development (Beacham & Murray, 1990; Fuhrman et al., 2018), altered migration timing (Crozier, 2015) and modified reproduction (Pankhurst & Munday, 2011), ultimately resulting in changes to fitness (e.g., survival: Martins et al., 2012; Whitney, Hinch, & Patterson, 2013). Examining the underlying phenotypic plasticity mediating these effects is important to assess how we expect species to fare under future climate scenarios (Hoffmann & Sgró, 2011; Merilä & Hendry, 2014), yet we still know fairly little about the mechanisms of phenotypic plasticity (Monaghan, 2008), and whether temperature effects can be further altered by additional environmental modulators (i.e., maternal effects; Galloway, 2005).

Some of the most potent modulators of adaptive phenotypic responses in offspring include maternal effects (Green, 2008; Meylan, Miles, & Clobert, 2012) or epigenetic programming (Bonduriansky, Crean, & Day, 2012). Indeed, maternal effects have long been recognized for the role in the fine-scale tuning of adaptive responses to often larger environmental stressors (Räsänen & Kruuk, 2007). For example, maternal exposure to, or experience with, environmental stressors translates information to developing offspring about the relative quality of their future environment (Sheriff et al., 2017). Maternally derived hormones have recently been acknowledged as a possible mechanism by which information about environmental quality can be translated to offspring via the mother (Meylan et al., 2012). In particular, maternally-derived glucocorticoid (GC) hormones have been proposed as

preparative mediators of offspring phenotype and performance in relation to the predicted quality of the offspring's future environment (Love et al., 2005; Sheriff & Love, 2013). GCs are strong candidates for this mediatory effect since they are involved in energy management and the stress response in vertebrates (Barton 2002; Moore & Jessop, 2003; Romero, 2004). As such, environmental stressors can elevate baseline GCs of mothers (Love, McGowan, & Sheriff, 2013; Wendelaar Bonga, 1997), and maternally-derived GCs can be transferred to developing offspring *in utero* (Matthews, 2002) or via the lipid content of eggs in oviparous species (Love et al., 2009; Sopinka et al., 2017) to act as a reliable signal of current and potentially future environmental quality for offspring (Love, McGowan, & Sheriff, 2013). Recent studies suggest that when the maternal environment is indicative of the offspring environment (environmental match hypothesis: Sheriff and Love 2013; Sheriff et al. 2018), maternal stress may elicit predictive adaptive responses (PARs) in offspring (Bateson, Gluckman, & Hanson, 2014), generating offspring phenotypes that may be better prepared for harsher environments (Bian et al., 2015; P. M. Capelle et al., 2019; Gagliano & McCormick, 2009; Love et al., 2013; Love & Williams, 2008). From a global climate change perspective, where the projected increase in global mean surface temperatures expected over the fifteen years (0.3-0.7°C by 2035: Stocker et al., 2013), maternally-derived GCs may therefore be involved in modulating the responses of ectothermic offspring to elevated temperatures, thus better-preparing offspring for managing elevated temperatures experienced as developing offspring or indeed adults (Sopinka et al. 2017).

Here we apply the concept of environmental matching to examine whether exposure to maternal stress can buffer the negative phenotypic effects of developing within elevated water temperatures in Chinook salmon (*Oncorhynchus tshawytscha*). Pacific salmon are an ideal

study species for these questions since they are ectothermic and are sensitive to changes in environmental temperatures (McCullough et al., 2009); are susceptible to migration stress when they return to terminally spawn and therefore can increase their circulating plasma GCs (Cook et al., 2014; McConnachie et al., 2012); and mothers and offspring overlap spatially in their spawning and development environment respectively, meaning that an honest signal about environmental quality passed from mother to eggs may be valuable for salmon offspring. Importantly, climate change has been implicated for declines in Chinook salmon populations across the West Coast of North America, through direct and indirect effects of water temperature increases and drought (Isaak et al., 2012; Mantua, Tohver, & Hamlet, 2010). Determining whether offspring are better prepared for warmer waters after having received a hormonal maternal signal about increased environmental stress is an important component for quantifying the overall adaptive capacity of Chinook salmon to climate change. To investigate how the effects of elevated temperatures and maternal stress interact to affect Chinook offspring phenotype and performance, we exposed eggs to maternal stress (biologically relevant levels of exogenous cortisol) or a control solution immediately post fertilization, and then split these eggs within females and raised the offspring in one of two temperature regimes: current (0°C) and elevated (+3˚C as predicted in next century by current climate models; van Vliet et al., 2013). In accordance with previous research on temperature effects (e.g., Daufresne, Lengfellner, & Sommer, 2009; Whitney et al., 2014), we predicted that individuals raised in elevated temperatures (regardless of prenatal stress treatment) would have lower survival and body size than those raised in current temperatures. However, since preparatory responses following exposure to a maternal stress signal may dampen the effects of an environmental stressor (i.e., environmental matching hypothesis; Sheriff et al., 2017),

we predicted that being exposed to maternal stress would help to buffer these negative impacts, resulting in relative increases in survival and body size at emergence (fry stage) for fish raised under elevated water temperatures.

Methods

Fish Origins

Our study species was Chinook salmon (*Oncorhynchus tshawytscha*) from the Credit River (43°34'40.0"N 79°42'06.3"W), which drains into Lake Ontario, Canada. Chinook salmon were purposely introduced to Lake Ontario starting in 1967 and they continue to be stocked for recreational fishing purposes (OMNRF, 2014). Spawning in the Credit River population takes place in the early fall, where eggs incubate under gravel in these natal streams until hatching in February and emergence in March, with juveniles migrating out into the lake in summer (Johnson, 2014). One of the main challenges to aquatic life in the Laurentian Great Lakes is climate change (Collingsworth et al., 2017), with increases in average temperatures reported in the last 50 years (e.g., Lake Ontario (+1.6 °C) in summer average temperature; Austin & Colman, 2007; Dobiesz & Lester, 2009). Similar increases in temperatures in tributaries that flow into the Great Lakes are also occurring (Chu, 2015; van Vliet et al., 2013), and therefore investigating this population's responses to these environmental changes may provide information relevant to the future status of Great Lakes fisheries under climate change.

We collected eggs and milt from fifteen adult females and nine males on October 4th, 2016 from the Credit River. We measured female body mass (mean ±SE, range: 7.89 ±0.41, 5.5-11.7kg), fork length (85.6 ±0.89, 80.0-93.0cm) and ovarian mass (ovarian mass= pre-post stripping mass: 0.98kg, ±0.10, 0.55-2.00kg). Eggs and milt were collected by applying

pressure to the abdomen, and the gametes were transported on ice in coolers to the University of Windsor's Aquatic Facility. Wet mass per egg (drained of ovarian fluid) was measured (to the nearest 0.01g) in three replicates of ten eggs (mean per egg ±SE, range: 0.30±0.11, 0.25-0.40g). All work described here was approved and completed under University of Windsor Animal Use Project Proposals (AUPPs: # 14-25 & #15-15).

Egg cortisol exposure and incubation temperatures

At the Facility, we filled individual containers with 90g of eggs (~300 eggs) from each of the fifteen females and added ~0.5ml (4 drops) of pooled milt from the nine males. After gently swilling the egg-milt solution, we added 60mL of river water to activate the sperm (Lehnert et al., 2018). After 2 minutes (when sperm should be no longer motile) we added river water mixed to 1000ng*mL^{-1} concentration of cortisol (H4001, Sigma-Aldrich Canada Co; Capelle et al. 2016) dissolved in 90% alcohol (HPLC grade, Sigma-Aldrich Canada Co) or 0ng*mL^{-1} control solution (alcohol and water only) for a 2-hour incubation. These concentrations and exposure times were chosen as they have shown in previous published studies to result in biologically relevant elevations of cortisol in the eggs (Burton et al., 2011; Capelle et al., 2016; Sopinka et al., 2016). After the 2-hour exposure, we rinsed the eggs using dechlorinated water, and a subset of 3 were removed from each cortisol replicate to verify the effectiveness of the cortisol manipulation (see below). The remaining eggs were transferred to 4-in x 3-in cells within a vertical egg-incubation stack that followed one of two temperature treatments. Eggs were incubated either at ambient water temperatures indicative of the 'current' water temperature scenario or under the predicted 'future climate change' scenario (i.e., elevated by 3°C based on predicted water temperature increases - outlined below - van Vliet et al., 2013; **Figure 2.1**). The current temperature regime mirrored average water

temperature seasonal patterns (2010-2014) recorded by a weather station adjacent to the Credit River salmon spawning grounds as part of the Provincial Water Quality Monitoring Network (PWQMN: Ontario Ministry of Environment and Climate Change). For the 'future climate change' temperature regime, water temperatures were elevated by 3°C based on climate projections for the region (van Vliet et al., 2013). This experimental design resulted in four different treatment groups: current temperature—control, current temperature—cortisol-dosed, elevated temperature—control, and elevated temperature—cortisol-dosed. To control for maternal effects, each female's eggs were split across all 4 treatment combinations, where each treatment (maternal stress + temperature) had 2 replicate containers resulting in 8 containers per female. Eggs from each container were then split across 2 replicate cells within each incubation stack, resulting in 16 incubation cells per female. This design allows for the control of container- and position effects of rearing.

Fertilization success, morphology and survival

Offspring development and mortality were assessed every two days. We removed and stored dead eggs in Stockard's solution (5% formaldehyde, 4% glacial acetic acid, 6% glycerin, 85% water) to determine fertilization success and at which developmental stage death occurred. Due to the effects of water temperature on development in this ectothermic species, offspring raised in different temperature regimes reached development stages at different calendar days (**Figure 2.1**). We therefore equalized fish developmental stages based on Accumulated Thermal Units (ATUs) which is the sum of average daily temperatures, and has shown to be highly correlated to fish growth and development (Chezik, Lester, & Venturelli, 2014; Neuheimer & Taggart, 2007). Once offspring reached their emergence stage (complete absorbance of the yolk sac: 835/830 ATUs on Dec 23/Feb 16 for elevated and

current temperature, respectively), five offspring from each cell were haphazardly chosen and removed from the incubation stack, their body mass measured (±0.01g), and an image taken for morphological analysis using ImageJ (https://imagej.nih.gov/ij/). From these images we measured standard length (SL), forked tail length (FL), gape (GAPE: operculum flap to tip of nose), eye width (EYE), body depth 1 (BD1: perpendicular to standard length starting from dorsal fin) & body depth 2 (BD2: from dorsal fin to deepest part of the belly), caudal peduncle width (PED), and caudal fin width (FIN) (**Figure 2.2**).

Egg cortisol assay

To verify the success of the cortisol manipulation, we measured cortisol concentrations in both unfertilized and 2-hour post treatment eggs using the protocol in Capelle et al. (2016). Briefly, we sampled three eggs from each treatment (unfertilized, control- dosed and cortisol-dosed) for each of the 15 females (N= 45). We weighed and blended each female's set of eggs from a given treatment in 1.2mL assay buffer, and then extracted the cortisol using 3mL diethyl ether. We vortexed samples for 30 seconds and then spun them in a centrifuge at 3500 rpm for 5 mins. Samples were allowed to rest for 30 mins and then flash frozen at –80°C for 30 mins. We decanted the remaining liquids and allowed any remains to evaporate overnight. Samples were reconstituted using 1.2mL assay buffer and stored in –80°C freezer until use in assay. To determine cortisol concentrations, egg samples were run in triplicate using ELISA Cayman Cortisol kits in 1:57 dilution. Samples were read at 412nm on a plate reader. Intra- and inter-assay coefficients of variation were 8.1% and 20.4% respectively.

Statistical analysis

Statistical analyses were completed in R version 3.5.1 (R Core Team, 2018). Model assumptions were assessed by graphical inspection: residuals versus fitted values were plotted

to verify homogeneity, and we assessed the quantile-quantile plots of the residuals to verify normality. Data were transformed when assumptions were not met.

Egg cortisol

Post-treatment egg cortisol levels were normalized using Box-Cox power transformation (MASS package, λ=0.242: Venables & Ripley, 2003). The effect of cortisol treatment on post-treatment egg cortisol levels was analysed using LMM with maternal ID as a random factor.

Fertilization success and Survival

We used the buildBinary function in *fullfact* package (Houde & Pitcher, 2016) to create data sets where each individual was assigned 1 when fertilized and 0 when unfertilized for fertilization success or 1 when alive and 0 when dead for survival. Both models were tested using a binomial distribution GLMM with maternal ID and incubation-cell placement nested in incubation-tray identity in the stack as random factors. The fertilization success model tested the cortisol treatment and temperature as main effects and their respective interaction. The survival model included cortisol treatment, temperature, developmental stage and their two-way interactions: cortisol treatment x temperature interaction, cortisol treatment x development stage interaction and temperature x developmental stage interaction. Fixed effects were tested using likelihood ratios (LRT) by fitting full models for fertilization success and survival with maximum likelihood estimation (Zuur et al., 2009). If all interactions were not significant ($p>0.05$), then interactions were removed from model and main effects were tested sequentially and compared using LRT with maximum likelihood (ML) estimation. If a significant interaction was present ($p<0.05$), all interactions were kept in the model and refitted using restricted maximum likelihood estimation (REML), and pairwise comparisons

using Tukey's HSD in *emmeans* package (Lenth et al., 2018). If no significant interactions were present, but a main effect was, the model was refitted using REML and tested using Tukey's HSD pairwise comparisons. Effects of random variable maternal ID were tested using LRT.

Morphology

Each of the nine morphological measurements was incorporated into a Principle Components Analysis (PCA; see **Table 2.1** for means). We removed individuals with incomplete measurements due to image quality (n= 94), thus leaving 1006 individuals for analysis. Two components were significant under the Kaiser criterion (eigenvalue >1) with Varimax orthogonal rotation. We considered loadings above 0.55 to be significant—criteria were chosen to reduce significant loadings between components (**Table 2.2**). PC1 explained 32.7% of the variance in the model and had positive significant loadings for mass, SL, FL, GAPE, and EYE explaining structural size. PC2 explained 30.6% of the variance in the model and had positive significant loadings for mass, BD1, and BD2 describing body condition. Cortisol-dose and rearing-temperature effects on these two morphological components (i.e., body size and body condition) were determined using linear mixed models with maternal ID, tray placement, and cell placement in incubation stack as random factors. The full model included fixed effects of cortisol dose and rearing temperature, as well as their interaction. We tested the significance of the interaction with LRT using maximum likelihood fit. If the interaction was significant, the model was refitted with REML and we conducted post hoc analysis using Tukey's HSD (*emmeans*, Lenth et al., 2018). If the interaction was not significant ($p<0.05$), main effects were tested sequentially using LRT. After the final model

was established, we tested the effects of maternal ID in the model (included as a random factor) using LRT and report contributed variance.

Results

Egg cortisol

Cortisol concentrations were significantly higher in the cortisol treated eggs than the control eggs (LMM: t= 6.9, p<0.001, n_{cort}=15, $n_{control}$=15; **Figure 2.3**). The cortisol treatment mean (mean ±SD, range; 75.2 ± 42.4, 26.64—194.1 ng*g-1), was within one standard deviation of the unfertilized non-manipulated eggs (43.8 ±42.6, 1.89—151.5 ng*g-1) suggesting that the cortisol treatment was within a biologically relevant cortisol elevation, consistent with previous work on egg cortisol manipulations (Sopinka et al., 2016).

Fertilization success and survival

There was no interactive effect of temperature and cortisol exposure on fertilization success (LRT: χ^2= 0.094, p= 0.76; **Figure 2.4**). Upon removing the interaction from the model, here were no significant main effects of temperature (χ^2= 0.39, p= 0.53) and cortisol treatment (χ^2= 0.16, p= 0.69). Fertilization success was highly affected by maternal ID (variance contributed=47.7%, χ^2= 149.6, p<0.0001). There was no significant effect of temperature- and cortisol treatment interaction on survival (χ^2= 1.69, p= 0.19), meaning that within the offspring raised in elevated temperatures, cortisol-dosed offspring did not have a higher survival than control offspring. However, there was a significant temperature by stage interaction on survival (LRT: χ^2= 10.51, p= 0.0052; **Figure 2.5**). Eyed offspring raised in elevated temperatures and received cortisol dose had a lower survival rate than those incubated in current temperatures (Tukey's HSD: Current—cortisol-dosed & control vs Elevated—cortisol-dosed: p<0.001). At the hatch stage, offspring raised in elevated

temperatures had a lower survival rate than those raised in current temperatures (Tukey's HSD: all combinations of Current—cortisol-dosed & control vs Elevated—cortisol-dosed & control: $p<0.05$). At the fry stage, there were no significant differences between fish raised in elevated versus current temperatures (all combinations of Current—cortisol-dosed & control vs Elevated—cortisol-dosed & control: $p>0.10$). The cortisol treatment and stage interaction had a marginal effect on survival ($\chi^2=4.66$, $p= 0.097$), however the specifics of this effect were not detected in post hoc analysis. Maternal ID significantly impacted offspring survival (variation in model=65.4%, $\chi2=144.0$, $p<0.0001$).

Structural size and body condition

There was no temperature by cortisol exposure interaction on structural size (i.e., morphology PC1) of fish at the emergence (fry) stage ($\chi^2= 2.00$, $p=0.16$). After removing the interaction from the model, temperature had a significant effect on structural size ($\chi^2= 8.76$, $p=0.003$; **Figure 2.6A**), where fry raised in elevated temperatures were significantly smaller than those in current temperatures. Cortisol treatment marginally affected fry body size ($\chi^2= 3.72$, $p=0.054$), where cortisol-dosed fry were marginally larger than control-dosed. Maternal ID had a significant effect of structural size ($\chi^2= 83.3$, $p<0.0001$) and contributed 16.5% of the variance. There was no interaction between temperature and cortisol exposure on body condition ($\chi^2= 0.13$, $p= 0.72$), and following the removal of the interaction there were no temperature- or cortisol exposure effects on body condition (temperature: $\chi^2= 0.003$, $p=0.99$; cortisol exposure: $\chi^2= 0.33$, $p=0.57$; **Figure 2.6B**). However, maternal ID significantly contributed to body condition (variance contributed= 29.8%, $\chi^2= 136.3$, $p<0.0001$).

Discussion

It is already fairly well established that elevated temperatures during early development can have diverse 'negative' phenotypic, performance and fitness-related effects on aquatic organisms (see Introduction). Based on predictions from the environmental matching hypothesis, we aimed to test whether early developmental exposure to elevated egg cortisol buffers these temperature effects. Our results confirm the effects of warmer water temperature, but suggest that exposure to elevated cortisol does little to ameliorate these impacts.

Early survival in warming waters

We found that overall warmer temperature, but not early cortisol exposure, affected offspring survival as those raised in elevated temperatures had lower survival rate than those in current temperatures during the eyed and hatchling stages. Elevated incubation temperatures above species preferred temperature range result in lower survival in juvenile salmonids, regardless of whether the elevation follows a mean (stable) temperature increase (Murray & Mcphail, 1988; Tang, Bryant, & Brannon, 1987; Whitney et al., 2013) or an oscillating temperature regime (Geist et al., 2006; Taranger & Hansen, 1993). Since salmon are ectothermic, offspring are sensitive to changes in temperature, especially during early development (Beacham & Murray, 1990; Tang et al., 1987) when offspring respond strongly to environmental cues (Monaghan, 2008). This associated decline in embryo survival has been attributed to temperature-dependent increases in yolk coagulation (McCollough 1999) and increases in developmental deformities (Cingi, Keinänen, & Vuorinen, 2010; Fraser et al., 2015).

Contrary to our prediction, exposure to elevated cortisol (i.e., maternal stress) did not appear to dampen the negative effect of elevated incubation temperatures on offspring survival. Previous studies have found a huge diversity of effects of maternal stress on offspring survival in fish (Sopinka et al. 2017). In Atlantic salmon (*Salmo salar*) where mothers received cortisol injections and eggs were incubated under mild hyperthermia (+2°C), there were no effects on offspring survival (Eriksen et al., 2006, 2007). However, under benign conditions some studies have reported either lower (*Oncorhynchus mykiss*: Li et al., 2010) and even higher (*Oncorhynchus tshawytscha*: Capelle et al., 2016) early survival for offspring exposed to elevated egg cortisol, the majority of studies have reported no effect across a diversity of fish species (*Danio rerio*: Nesan & Vijayan, 2016; *Salmo trutta*: Sloman, 2010; *Oncorhynchus kisutch*, *Oncorhynchus keta*, *Oncorhynchus nerka*: Sopinka et al., 2016, 2015; *Oncorhynchus kisutch*: Stratholt, Donaldson, & Liley, 1997). Considering these contrasting results both within and across fish species, it may not be surprising that exposure to elevated egg cortisol was incapable of ameliorating survival impacts of elevated water temperatures. Instead, it is possible that prenatal exposure to cortisol may have downstream impacts on survival at later developmental stages through differences in performance (such as morphology, physiology and behaviour), especially when under the effects of a stressful environment. These effects largely remain to be studied in fish within this environmental context (although see Capelle 2017).

Survival from egg to exogenously feeding fry represents an important bottleneck that limits offspring migration success to the lake or ocean (Groot & Margolis, 1991). Thus, elevated temperatures in the riverine stage may have long lasting effects. In our study, effects of temperature on survival were significant during the very early (i.e., sensitive) stages of

development - the eyed and hatching stage. As such, if managers were only able to examine survival at the fry stage, temperature effects would not be detected, meaning that conservation measures could be delayed while populations are still declining. Therefore, quantifying relative survival across life stages to determine which are at higher risk of mortality due to stressors such as temperature is important, especially since many Chinook salmon populations across the Pacific coast are in decline (Kope et al., 2016).

Temperature and prenatal cortisol effects on fry structural size and body condition

Morphology and in particular, body size has an important impact on juvenile performance metrics such as foraging (Johnsson, 1993), or predator avoidance (Tucker, Hipfner, & Trudel, 2016), that ultimately contribute to variation in fitness. We found that fry raised in elevated temperatures were smaller than those raised under current water conditions, although we did not find any similar temperature effects on body condition. Previous work has shown that elevated water temperatures results in smaller body size in fish larva and fry (Beacham & Murray, 1990; Murray & Mcphail, 1988; Whitney et al., 2014). The higher metabolic costs of living in warmer waters is thought to drive the smaller body sizes (Kingsolver & Huey, 2008; Sheridan & Bickford, 2011), by allocating less energy for growth and non-maintenance activities to compensate for the thermal stress. Interestingly, biothermal modeling in ectotherms has predicted selection for smaller body size and maturation at a younger age (Daufresne et al., 2009). As such, smaller body size per se may not in itself be inherently negative, especially when body condition did not change, suggesting they are overall smaller, and not energetically limited. Additionally, it is important to emphasize that changes in phenotype do not necessarily translate into changes in performance, and most importantly, fitness.

We did not detect any significant effects of exposure to elevated prenatal cortisol on either structural size (PC1) or body condition (PC2) within the elevated rearing temperatures. Thus, our results do not support predictions from the environmental matching hypothesis that early developmental exposure to elevated cortisol better match offspring to warmer waters through an increase in body size or condition. As with our observed effects for survival, elevated temperatures may have an overwhelmingly greater impact as an environmental effect on growth and development compared to our modulatory maternal effect, prenatal egg cortisol. There was a marginal effect of prenatal cortisol on overall body condition regardless of temperature treatment, which suggest that prenatal cortisol may still enact phenotypic change. Previous work shows that prenatal cortisol often results in decreased body size in benign environments (Sopinka et al., 2017), and few studies have examined the interactive effects of prenatal cortisol and stressful environments on body size. Atlantic salmon fry whose mothers were exposed to cortisol close to spawning had smaller body length and body mass than control fry when incubated in (+2°C) elevated temperatures at fry stage (Eriksen et al., 2006). Likewise, Chinook salmon exposed to high dose egg cortisol (1000 $ng*ml^{-1}$) and kept in low water conditions were smaller and had lower growth (Capelle 2017). However, when low dose cortisol (300 $ng*ml^{-1}$) exposed Chinook were kept in same conditions (low water), they had higher freshwater growth, suggesting a match between prenatal signal and postnatal environment (Capelle 2017). This highlights the importance of following offspring phenotype throughout their life, as maternal stress may induce effects on phenotype and performance at later life stages. Importantly, it must be emphasized that despite having lower survival, offspring in elevated temperatures may benefit from a smaller body size via less caloric intake

needed (Gillooly, Brown, & West, 2001), or even better escape performance for smaller or lighter individuals (Chin et al., 2009).

Early life in stressful environment

Wild fish populations are globally in decline (Dulvy et al., 2006), and a number of salmonid species, including Chinook, are currently at population extinction risk, presumably due to the direct and indirect effects of climate change (Crozier, 2015). Early development in juvenile salmon occurs in a dynamic environment; streams and rivers can change considerably in temperatures both daily and seasonally (Caissie, 2006). Due to this fluctuating tendency and higher surface area to volume, streams and rivers are more likely to be affected by climate change through temperature increase and extreme changes in flow (Hill, Hawkins, & Jin, 2014; van Vliet, Franssen, et al., 2013). Since environmental variation during development plays a large role in generating variability in offspring phenotypes through developmental plasticity and flexibility, it is important to determine whether juvenile salmon have the capacity to respond to the rapid rate of climate change their environments are currently facing. In our study we examined the role of a stress-induced maternal effect, egg cortisol, as a relevant potential modulator of offspring phenotype and performance in response to elevated water temperatures induced by anthropogenic change. More broadly, we were also able to test predictions of the environmental matching framework to test the potential for maternal signals to modulate offspring responses adaptively to respond to stressful future environments. Although we did not find support that maternal stress led to altered offspring phenotypes or survival during early freshwater stages, it is still possible that exposure to maternal stress modulates phenotypes or performance at later important developmental stages. Regardless, interpreting the fitness impacts of maternal stress within an environmental context continues

to be highly important for determining how intergenerational maternal effects may assist in species responses to rapid changes in their environment.

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Tables

Table 2.1: Morphology traits (mean ± SE) of Chinook salmon fry across prenatal cortisol and temperature treatments.

Trait	Current Temperature		Elevated Temperature	
	Control	**Cortisol-dosed**	**Control**	**Cortisol-dosed**
Mass (g)	0.446 ± 0.003	0.443 ± 0.003	0.431 ± 0.004	0.434 ± 0.004
Standard Length (mm)	33.8 ± 0.07	33.7 ± 0.07	33.2 ± 0.07	33.3 ± 0.07
Fork Length (mm)	37.7 ± 0.08	37.6 ± 0.08	37.1 ± 0.08	37.1 ± 0.08
Gape (mm)	8.11 ± 0.02	8.12 ± 0.02	8.01 ± 0.02	8.05 ± 0.02
Eye Width (mm)	2.20 ± 0.01	2.21 ± 0.01	2.17 ± 0.01	2.19 ± 0.01
Body Depth 1 (mm)	6.26 ± 0.03	6.24 ± 0.03	6.18 ± 0.03	6.10 ± 0.04
Body Depth 2 (mm)	7.76 ± 0.04	7.74 ± 0.04	7.87 ± 0.05	7.79 ± 0.05
Caudal Peduncle (mm)	2.35 ± 0.01	2.33 ± 0.01	2.18 ± 0.01	2.21 ± 0.01
Caudal Fin (mm)	7.46 ± 0.04	7.39 ± 0.04	7.35 ± 0.04	7.40 ± 0.04
Number of samples	238	273	253	242

Table 2.2: Principal component loadings for the morphology PCA. PC1 and PC2 account for 32.7% and 30.6% of the variance in the model respectively. Loadings were considered significant (>0.55) are bolded.

Trait	**PC1** **Structural size**	**PC2** **Body condition**
Mass	0.55	**0.61**
Standard Length	**0.74**	0.49
Fork Length	**0.76**	0.51
Gape	**0.71**	0.22
Eye Width	**0.71**	-0.14
Body Depth 1	0.21	**0.87**
Body Depth 2	0	**0.86**
Caudal Peduncle	0.54	0.22
Caudal Fin	0.40	0.53
Percent Variance Explained	32.7%	30.6%
Eigenvalue	4.5	1.2

Figures

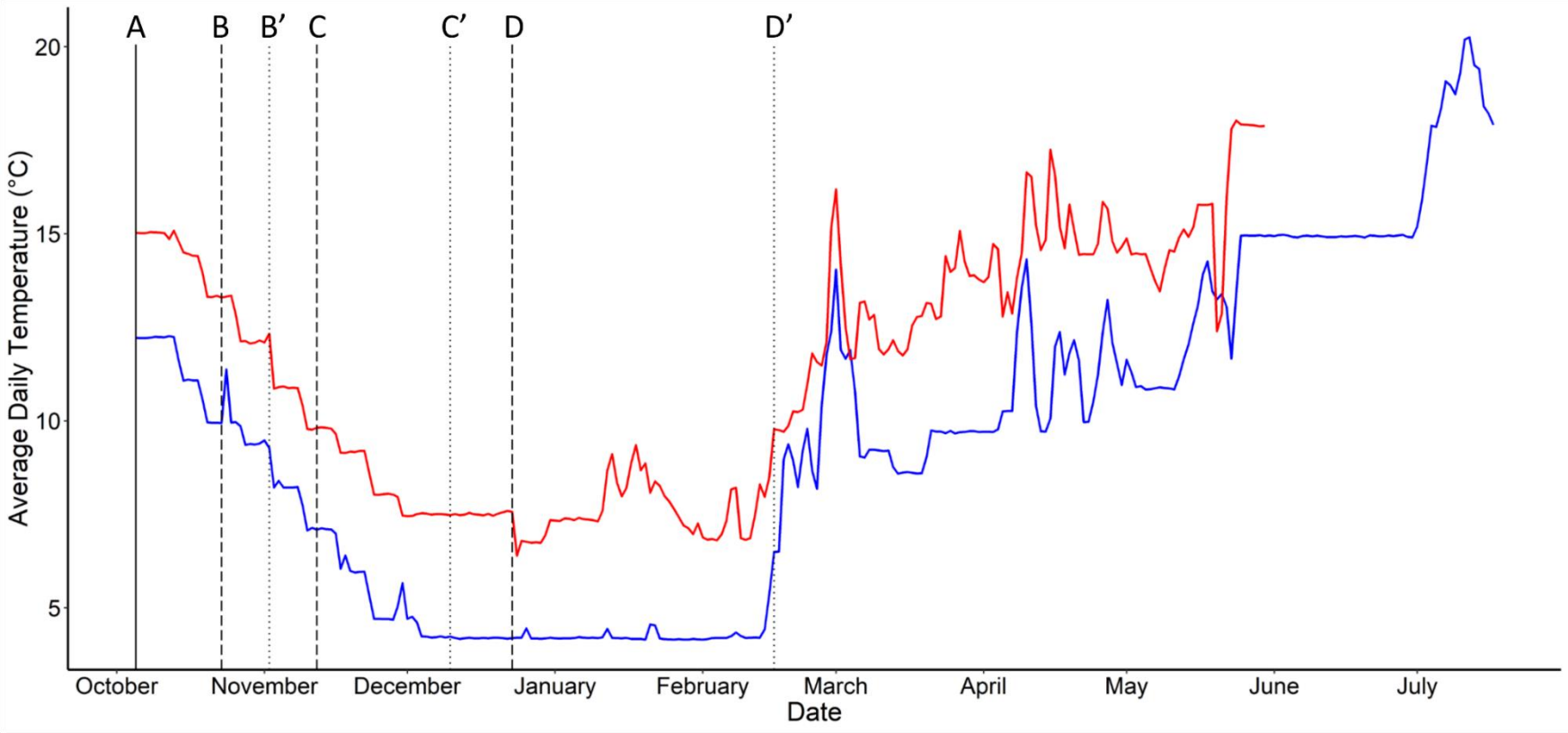

Figure 2.1: Recorded average daily temperature for each temperature regime in the rearing experimental design. Current temperature regime is represented by the blue line while the elevated temperature regime is in red. Vertical lines represent sampling timepoints: solid—both current and elevated groups; dashed—elevated, dotted—current. Timepoints are: **A**—Fertilization (Oct 4th), **B** and **B'**—Eyed (Oct 23rd/Nov 2nd), **C** and **C'**—Hatch (Nov 12th/Dec 10th), **D** and **D'**—Fry (Dec 23rd/Feb 16th).

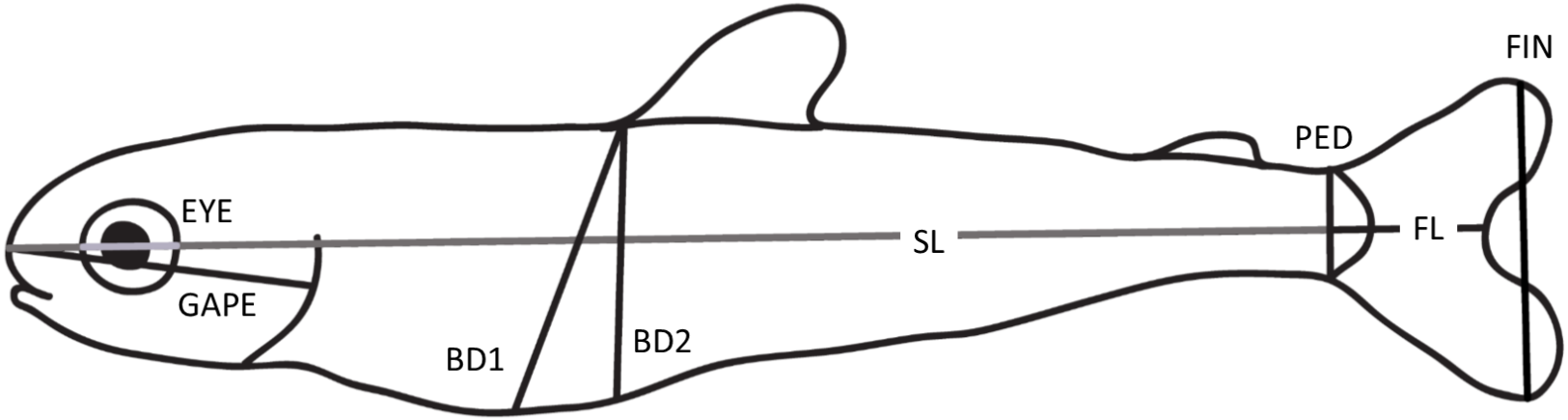

Figure 2.2: Morphological measurements of emerged juvenile Chinook salmon taken from photographs analysed using ImageJ. Measurements include standard length (SL: grey), forked length (FL: grey & black), gape (GAPE), eye width (EYE: light grey), body depths 1 & 2 (BD1 & BD2), caudal peduncle width (PED), and caudal fin width (FIN).

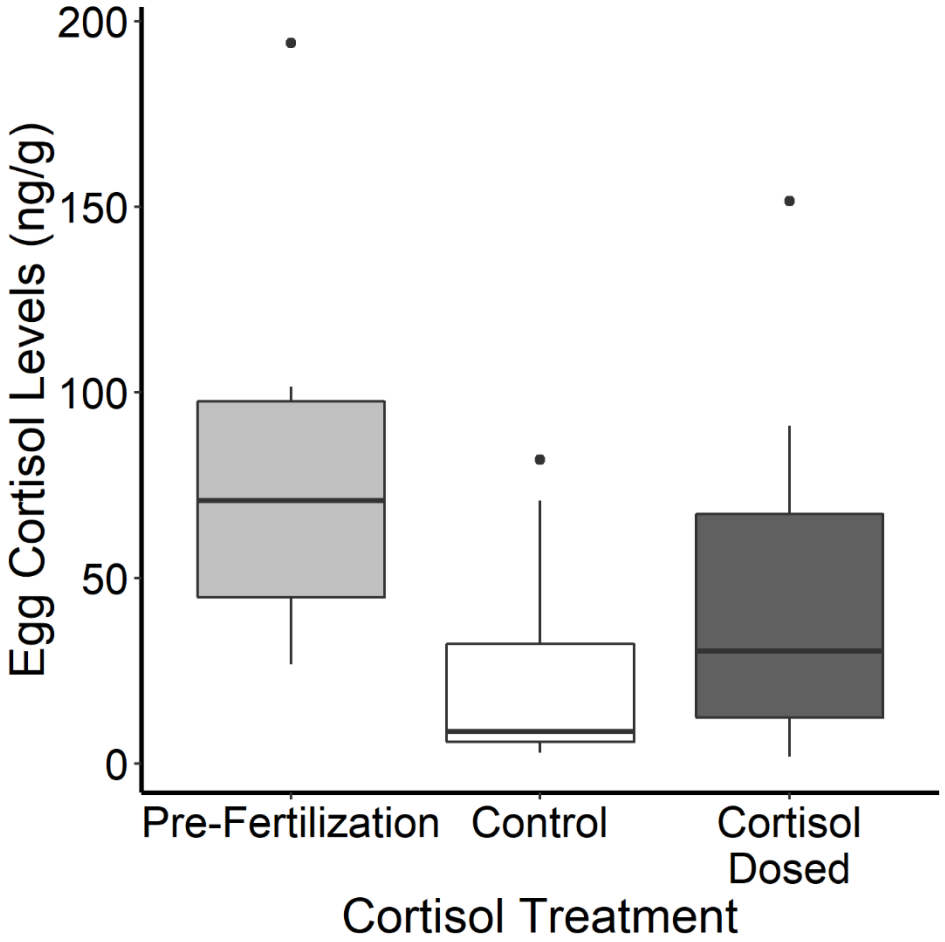

Figure 2.3: Cortisol concentrations in the pre-fertilized eggs and in manipulated eggs after the 2 hour cortisol treatment incubation: control 0ng*mL^{-1} or cortisol dosed 1000ng*mL^{-1}. Cortisol dosed eggs had significantly higher cortisol levels than control treated eggs.

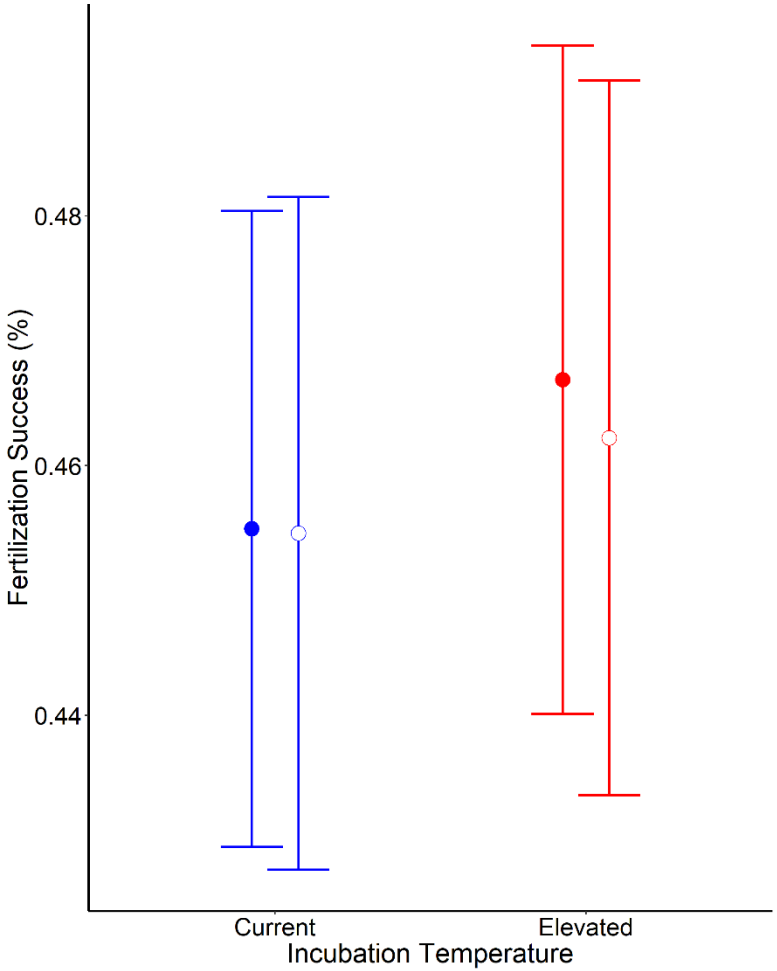

Figure 2.4: Percent fertilization success across the treatment groups. Open circles depict control and closed circle depict cortisol-dosed eggs. Temperature and cortisol dose treatment did not significantly influence fertilization success.

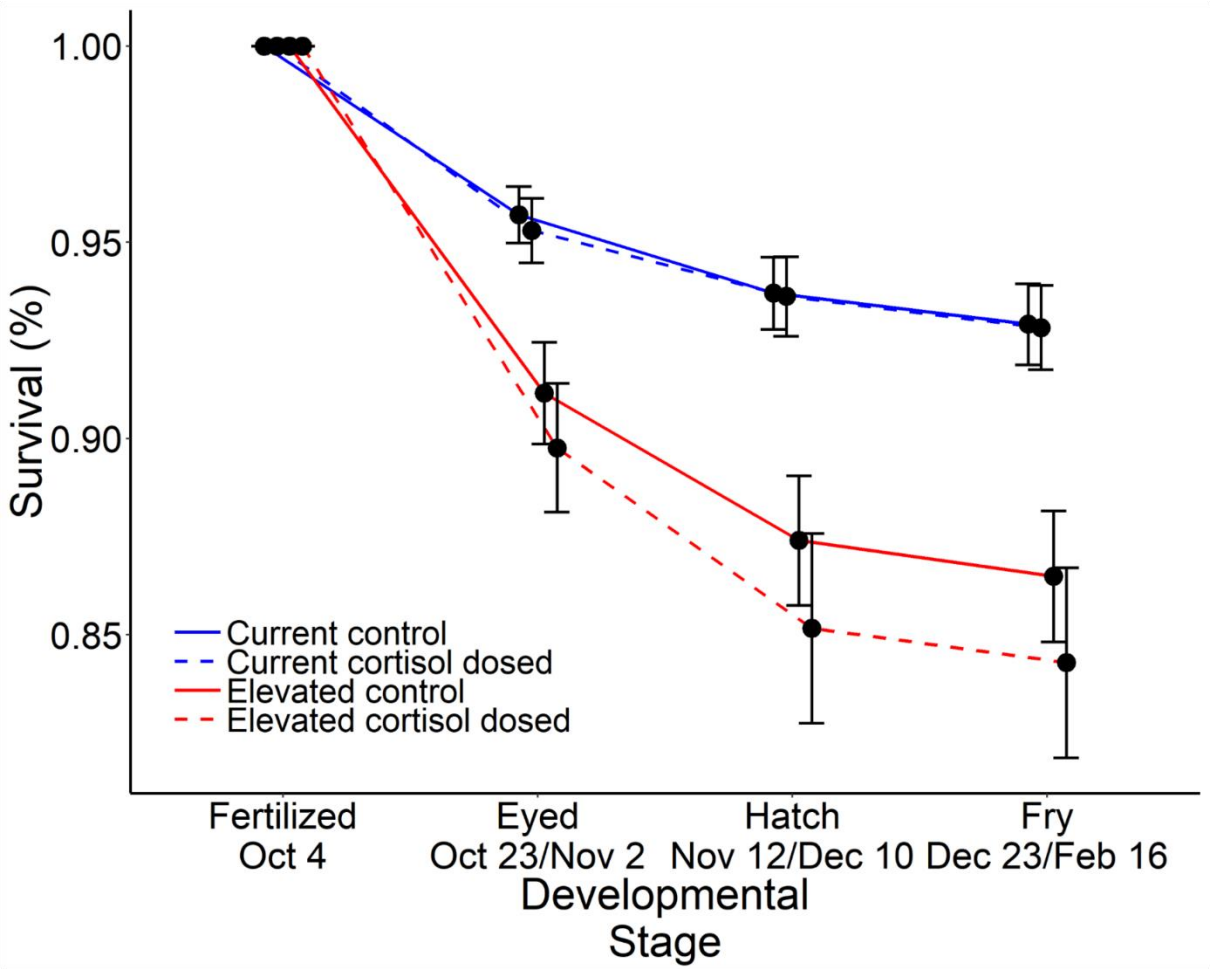

Figure 2.5: Percent survival across developmental stages. Blue lines represents offspring raised in current temperatures while red are those raised in elevated temperature. Solid lines depict control and dashed lines depict cortisol dosed offspring. Significant differences between elevated and current temperature fish were detected in the eyed and hatch stages only.

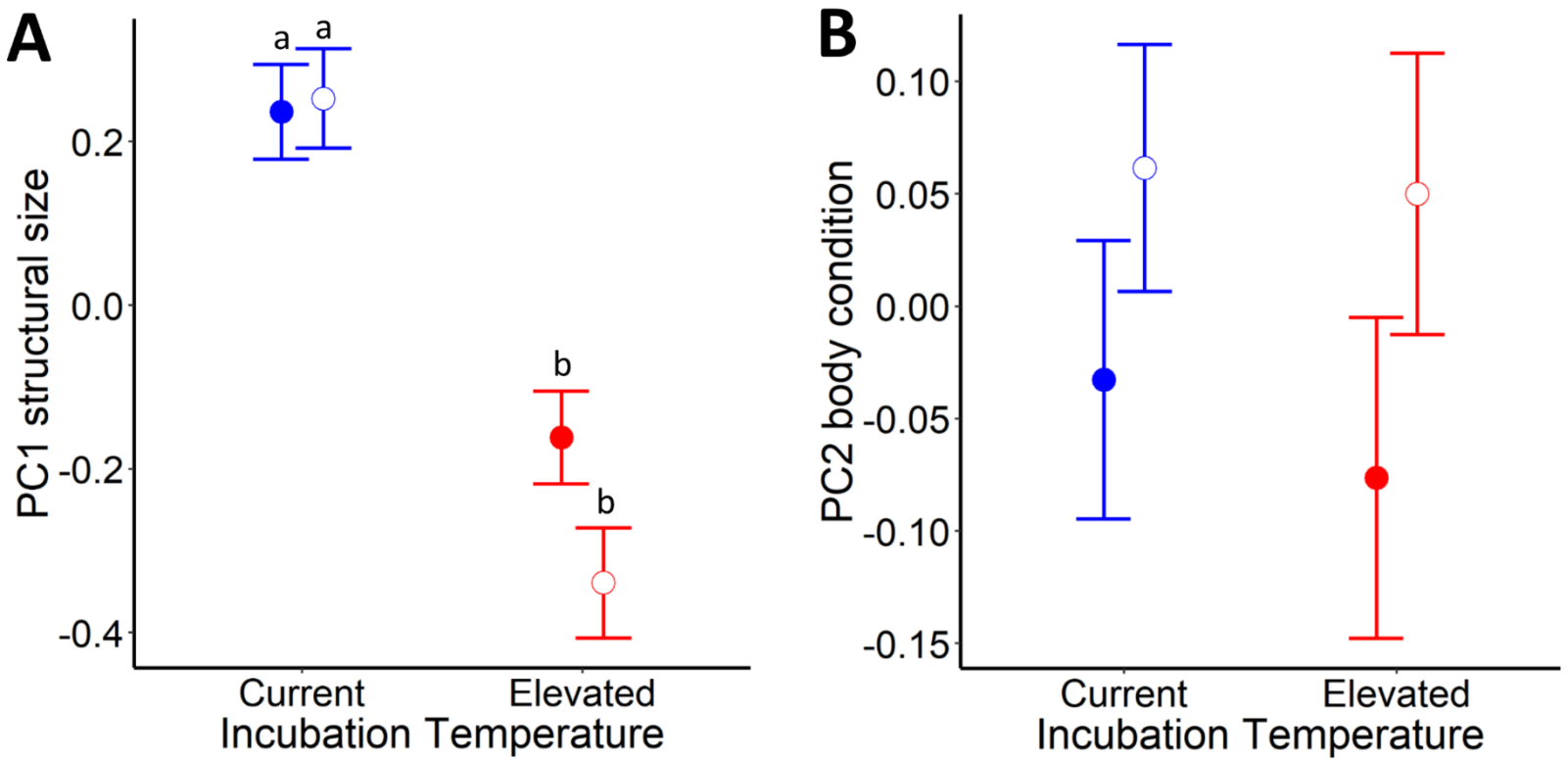

Figure 2.6: Morphology PCA scores for: **A** PC1—structural size and **B** PC2—body condition. Open circles depict control and closed circles depict cortisol-dosed offspring. A. Offspring raised in elevated temperatures were structurally smaller than those raised in current. B. No differences in body condition across the treatment groups.

CHAPTER 3 — COMBINED EFFECT OF ELEVATED REARING TEMPERATURES AND PRENATAL CORTISOL ON THERMAL TOLERANCE OF JUVENILE CHINOOK SALMON

Introduction

Climate change is a major contributor to rapid global changes, whether via increasing average temperatures (Solomon et al., 2012), or increasing frequency of extreme weather events (e.g., wildfires, droughts: Rahmstorf & Coumou, 2011). Ectotherms such as reptiles, amphibians and many fish species are potentially sensitive to increases in both average temperatures and increases in frequency of extreme weather events, which may have lasting effects within and across generations (Buckley & Huey, 2016; Deutsch et al., 2008; McCullough et al., 2009). To respond and persist within a rapidly changing world, species require mechanisms such as phenotypic plasticity, phenotypically flexible responses, and contemporary evolution which act within and across generations (Chevin, Lande, & Mace, 2010; Hendry, Farrugia, & Kinnison, 2008). These adaptive mechanisms enable organisms to optimize growth, morphology, and physiology in response to current (or expected) environmental conditions to ultimately maximize performance, reproduction, and survival (Fox et al., 2019; Seebacher, White, & Franklin, 2014). Indeed, ectotherms that develop in warmer temperatures have been shown to have altered phenotypes: smaller bodies (Cingi, Keinänen, & Vuorinen, 2010; Sheridan & Bickford, 2011; Whitney, Hinch, & Patterson, 2014), and increased metabolism (Clarke & Johnston, 1999; Enders & Boisclair, 2016). These responses have also been shown to impact performance (i.e., higher thermal tolerance: Bickford et al., 2010; Dillon, Wang, & Huey, 2010; Sandblom et al., 2016 but see Chen et al., 2013) and fitness (i.e., survival: Martins et al., 2012; Rohr & Palmer, 2013).

Non-genetic maternal effects such as variation in egg quality (Bernardo, 1996; Einum & Fleming, 1999; Sinervo, 1990), variation in parental behaviour (Champagne et al., 2003;

Koch & Meunier, 2014), and traits such as maternally-derived hormones (Dantzer et al., 2013; Ruuskanen, 2015) have long been recognized for their potential to shape offspring phenotype and performance in response to current or expected environmental quality (Green, 2008; Mousseau & Fox, 1998). When a mother is exposed to a stressful environment during gestation or follicular recruitment, she may mount a stress response by elevating her glucocorticoid (GC) levels (Schreck & Tort, 2016; Wingfield, 2013). Recent research examines the transfer of environmentally-elevated GCs from mother to developing offspring (i.e., maternal stress) as a modulator of offspring phenotype and performance (Love et al., 2005, 2009). Some researchers predict that GCs may act as an inducer of predictive adaptive responses in offspring faced with stressful future environments (Gluckman, Hanson, & Spencer, 2005; Sheriff & Love, 2013). These types of adaptive response mechanisms have already been highlighted as potential drivers of flexible responses to warming environments (Meylan, Miles, & Clobert, 2012), but the role of maternal GCs as a signal of warmer waters to offspring is not established. Exposure to increased maternal GCs has been shown to result in phenotypes that would be expected to have positive performance in warmer thermal environments (i.e., slower growth: Hayward & Wingfield, 2004, smaller body size: Burton et al., 2011; Love et al., 2005, and lower energetic demand: Capelle et al., 2019). In species where there is spatial or temporal overlap in the maternal and offspring environment (Sheriff & Love, 2013), these stress-induced maternal effects may be particularly relevant for preparing offspring for stressful future environments (Capelle et al. 2017; Sopinka et al. 2017). Although we already appreciate that elevated temperatures can lead to altered offspring phenotypes across generations (Burt, Hinch, & Patterson, 2011; Jonsson & Jonsson, 2016; Le Roy, Loughland, & Seebacher, 2017), it is unclear whether maternal stress signals (via GCs)

interact with developmental temperatures to even better prepare offspring for future increases in environmental temperatures, especially in at-risk species (Love et al., in review.; Sopinka et al., 2017).

Here we examine the interaction between exposure to maternal stress and rearing temperatures on the future thermal performance of juvenile Chinook salmon (*Oncorhynchus tshawytscha*). Aquatic systems are expected to be highly altered by climate change, not only by increasing average water temperatures, but by changing the hydrological cycle (e.g., increase in precipitation variation), by causing more extremes in water flow (e.g., droughts and floods), and by increasing daily temperature extremes (e.g., thermal spikes) (Mantua, Tohver, & Hamlet, 2010; Woodward et al., 2016). Exploring whether Pacific salmon possess stress-induced responses that may mitigate the effects of climate change is relevant from both a mechanistic and a conservation point of view. Mechanistically, Pacific salmon are ectothermic and thus are highly susceptible to thermal stressors (Kuehne, Olden, & Duda, 2012). Pacific salmon mount a stress response (i.e., elevated GCs) to additional environmental stressors during migration and spawning (Cook et al., 2014), and the maternal spawning and offspring development environments overlap, thus enabling maternal stress to act as a reliable signal of the offspring's future environment (Healey, 1991). From a conservation standpoint, increasing river temperatures and higher frequency of droughts are predicted to highly impact cold-water species such as North American Pacific salmon (Cunningham, Westley, & Adkison, 2018; McCullough et al., 2009); multiple Pacific salmon species are in (or soon expected to be in) decline due to thermal issues (Crozier et al., 2008; Ford et al., 2011); and multiple populations of Chinook salmon in particular are in rapid decline (COSEWIC, 2018).

To determine the effects of climate change on Pacific salmon, we applied the environmental matching paradigm (Sheriff and Love 2013), wherein we test the adaptive potential of altered offspring phenotypes (due to maternal stress) to cope with environmentally-relevant stressors such as increased temperatures under climate change (i.e., not simply within benign environments; Sheriff et al., 2017). Within this framework, we mimicked a maternal stress signal by exogenously elevating egg cortisol, and then raised resultant cortisol-dosed (and control) offspring under a current (+0°C) or an elevated temperature regime (+3°C) (**Figure 1.2**). At the parr life history stage we assessed the thermal performance of fish in two ways. First, we determined the CTMax of the fish, defined as the temperature at which fish lose equilibrium under steadily increasing water temperatures (Chen et al., 2013; McDonnell & Chapman, 2015). This is a standardized approach in the literature to approximate thermal tolerance (Becker & Genoway, 1979; Lutterschmidt & Hutchison, 1997). Second, we quantified the energetic coping ability of the fish following three days of environmentally relevant but challenging thermal spikes (+9 °C—one spike per day) (Corey et al., 2017; Gallant et al., 2017). Given previous work that found higher water temperatures led to higher thermal tolerance (Sandblom et al., 2016), we predicted offspring raised in elevated temperatures would have a higher thermal performance (i.e., higher CTMax, lower energetic cost) in both thermal performance experiments. We further predicted that based on the environmental matching hypothesis, fish exposed to a signal of maternal stress would have a greater thermal performance compared to control-dosed offspring facing the same thermal challenge.

Methods

Fish origins & husbandry

We caught 15 female and 9 male Chinook salmon from the Credit River, Ontario, Canada (43°34'40.0"N 79°42'06.3"W), stripped their gametes, and these were transferred to the University of Windsor on ice in coolers. We fertilized eggs from each female separately using pooled set of milt in 950mL containers. We activated the sperm by using 60mL of river water (Lehnert et al., 2018). Immediately following fertilization, river water mixed to 1000ng*mL^{-1} of cortisol (H4001, Sigma-Aldrich Canada Co) dissolved in 90% ethanol (HPLC grade, Sigma-Aldrich Canada Co) or 0 ng*mL^{-1} (alcohol and water only) was added to each container of eggs for our cortisol-dosed and control-dosed treatments respectively (8 containers per female: 4 cortisol-dosed & 4 control). The cortisol dose concentration was designed to increase egg cortisol levels within a biologically relevant range based on previous validations (Auperin & Geslin, 2008; P. M. Capelle et al., 2017; Sopinka et al., 2016). After a 2- hour cortisol treatment, eggs were rinsed using dechlorinated water, and each container of eggs were then further split into two cells (4-in x 3-in) within a vertical egg-incubation stack. In order to replicate our cortisol treatment, we placed 2 cortisol-dosed and 2 control treated containers from the same female in the same incubation stack (16 incubation cells per female). Eggs were then reared at either current (+0°C) or elevated (+3°C) temperature regime (one incubation stack per temperature treatment). The current temperature regime was chosen to mimic water temperatures recorded in the Credit River through the Provincial Water Quality Monitoring Network from 2010 to 2014 (PWQMN: Ontario Ministry of Environment and Climate Change), while elevated rearing temperatures were chosen to reflect the higher end of climate change models for river temperatures (van Vliet, Ludwig, & Kabat, 2013). This

resulted in 4 treatment combinations in a 2 x 2 design (**Figure 1.2**). Each female's eggs were split across all 4 groups to account for maternal effects. All work described here was approved and completed under University of Windsor Animal Use Project Proposals (AUPPs: # 14-25 & #15-15).

On Dec 23rd and Feb 16th for elevated- and current raised temperatures offspring respectively, button-up fry were transferred to recirculation-system 320L housing tanks (5 tanks per temperature treatment and a separate system for each temperature treatment). We separated offspring by mother and cortisol treatment using 10L perforated buckets placed within the holding tanks (6 buckets per tank). Buckets contained 100 offspring each (combined from replicate cortisol treatment containers and replicate incubation cells). During this period water changes occurred at least daily to maintain water quality. The fish were housed under red light conditions following a 12:12 hour light: dark cycle and were fed 3-4 times a day ad libitum. During this period, water temperature of the housing tanks continued to follow the current and elevated temperature seasonal regime (**Figure 3.1**). Due to mechanical error of an in-line chiller used to control the current-temperature treatment, housing temperatures were slowly raised with drop-in chillers to match that of the elevated-temperature treatment on March 3rd, 2017, to minimize stress. Since this overlap in temperature was for only 5 days until the chiller was repaired, and within the magnitude of temperature fluctuations found in riverine environments (Caissie, 2006), the effects of this period are expected to be minimal. Experiments were performed when fish were at similar accumulated thermal units (ATUs: **Table 3.1**), which has been shown to be highly correlated to fish development (Neuheimer & Taggart, 2007).

CTMax

We evaluated the acute thermal tolerance of the fish across maternal stress and rearing temperature groups by determining their Critical Thermal Maximum (CTMax). CTMax is defined as the temperature at which fish lose equilibrium under steadily increasing water temperature (Becker & Genoway, 1979; Lutterschmidt & Hutchison, 1997). These trials occurred on May 1st - 7th (elevated temperature) and on June 21st - 26th, 2017 (current temperature). Two experimental tanks were always run concurrently, and trials ran between 08:00H and 19:00H. Within each experimental tank, 4 individuals were each placed in separate tapered perforated circular buckets (top diameter 28 cm x bottom diameter 16.5cm x deep 28 cm), with individuals per trial consisting of the same maternal ID, cortisol- and rearing temperature treatment (n_{total}= 234). Two air stones were used per experimental tank to ensure dissolved oxygen levels remained high throughout the trials. These experimental tanks had the same water temperature as the housing tank at the start of the trial (±0.7°C, starting temperature range= 13.2-15.1 °C), which was controlled by an immersion circulating heater (SC100 Immersion Circulators: Thermo Fisher Scientific). At the end of the 1-hour acclimation period, we increased temperature (by ~0.2°C*min^{-1}, $\bar{x}$=0.20, range=0.13-0.35; similar to rate in Becker & Genoway, 1979) until the fish lost equilibrium. We measured temperature throughout the trial and dissolved oxygen pre- and post-trial in a sub-sample of experimental tanks ($n_{pre\text{-}trial}$ DO=40, $\bar{x}_{\text{ pre-trial}}$ DO=8.7, $n_{post\text{-}trial}$ DO=37, $\bar{x}_{\text{ post-trial}}$ DO=6.83; LabQuest 2, stainless steel temperature probe, optical DO probe: Vernier). Our temperature probe measured temperature to 0.1°C with ±0.2-0.5°C accuracy at 0-100°C respectively. The trials were filmed under red light using low-light sensitivity cameras for later video analysis. After each trial, we euthanized the 8 fish using clove oil and pithing, and measured their body

mass (to 0.01g). During video analysis, the experimenter—blind to maternal ID and cortisol treatment—recorded the time when the fish lost equilibrium for a minimum of 10 consecutive seconds, and at what temperature this occurred.

Energetic response to thermal spikes

As our second measure of thermal performance, we quantified the energetic (physiological) coping ability of fish in relation to the maternal stress and rearing temperature treatments following three days of thermal spikes (+ 9°C—one spike per day). The maximum temperature of our thermal spike was chosen from the results of our CTMax experiment (slightly lower so that temperature spike was challenging but not affecting locomotion). The rate of increase in our environmentally-relevant temperature spikes was chosen based on literature examining the effects of diel cycling and temperature spikes on fish energetics and metabolism (Corey et al., 2017; Gallant et al., 2017; Tunnah, Currie, & MacCormack, 2016). Many of these studies have based their ramping off river temperature data. After these experiments, we were able to confirm similar spikes in temperature in the natal stream through water temperature loggers deployed Oct 2016 to Oct 2017 (**Figure 3.2**). These trials occurred on May 25th -June 2nd and on July 12th -20th for the elevated and current treatments, respectively ($n_{\text{replicate trials}}$=4; **Figure 3.3**). On the evening prior to the first day of thermal spiking, 32 fish were transferred from their housing tanks into two experimental tanks. We conducted two different temperature cycle treatments: first, the spiked group, experienced three environmentally relevant thermal spikes over the 3 days, whereas the second, the constant group, was maintained at steady temperature (~18°C, to act as a control for potential transfer stress). In the spiked temperature treatment, thermal spikes were increased and decreased at a rate 1°C* h^{-1}, resulting in a + 9°C temperature spike in 18 hours. Within each

tank, 4 perforated buckets (top diameter 28cm x bottom diameter 16.5cm x deep 28cm) separating fish by treatment, each contained 3-4 randomly selected fish from the same temperature and cortisol treatment over the experimental period (n_{total}=117). On the night of the final day, buckets were covered with opaque lids to reduce disturbance during sampling planned for the following morning. At 07:00H of the 4th day, we removed the fish from their containers using a net, and collected their blood (within 3 minutes of first disturbance for each bucket) through a caudal puncture using 10μL heparinized microcapillary tubes. Each fish was then weighed (0.01g) and placed in RNA-Later for a future transcriptomics project (C. Finerty MSc). We transferred the blood into heparinized microcentrifuge tubes and placed these on ice, and we measured blood glucose and lactate concentrations on-site from whole blood using handheld meters within 8-13 minutes of whole blood being collected (Freestyle Insulinx: Abbott Diabetes; Lactate Plus: Nova Biochemical; Barkley et al., 2016; Beecham, Small, & Minchew, 2006; Wells & Pankhurst, 1999). Remaining blood was stored on ice until 1 hour later when it was centrifuged at 10000rpm for 12 mins, and the plasma was collected and stored at -80˚C for later cortisol analysis. We assayed the plasma of focal fish for baseline cortisol levels using non-extracted plasma and a previously-validated enzyme-linked immunosorbent assay (ELISA Cortisol Kit: Cayman Chemical; Capelle, 2017). We ran samples in triplicate at a 1:50 dilution. Assay plates were read at 412nm on a plate reader, and intra- and inter-assay variation were 2.8% and 17.5% respectively.

Statistical Analysis

Statistical analyses were completed in R version 3.5.1 (R Core Team, 2018). Model assumptions were assessed by graphical inspection: quantile-quantile plots of the residuals to verify normality, and residuals versus fitted values were plotted to verify homogeneity. Data

were transformed when assumptions were not met using a log transformation, or when needed, a Box-Cox power transformation (Osborne, 2010) in the *MASS* package (Venables & Ripley, 2003). Linear mixed models (LMM) were run in the *lme4* package (Bates et al., 2015).

After visually plotting CTMax scores, we detected one individual score to be 3 median absolute deviations (MAD) from the median (Med=28.8°C, MAD=0.48, datum=26.6) and thus it was identified to be an outlier and removed from the dataset (Leys et al., 2013). This individual was at the smaller body mass end of our range (mass=0.65g), and in field notes was recorded to be in poor body condition (i.e., frayed tail), which may have contributed to its earlier loss of equilibrium. For CTMax, we examined the interactive effects of rearing temperature and prenatal cortisol using a model that included the fixed effects of rearing temperature x cortisol treatment interaction, and their main effects (rearing temperature + cortisol treatment) and offspring mass. We included testing tank, testing bucket (nested within tank), start temperature, and maternal ID as random effects for this model.

The energetic response of offspring to temperature cycle (thermal spikes) was analysed using an LMM after transformation (plasma cortisol: Box-Cox, λ=0.323; glucose and lactate: log transformation) (Osborne, 2010). Since we a priori were interested in comparing energetic values within the same rearing temperature treatment, we separated the analyses into separate models for each rearing temperature. This decision also avoided the need to examine notoriously difficult to interpret a 3-way interaction between maternal stress treatment, water temperature treatment and the spike treatment. Within current and elevated temperature regimes, we tested models for response variables: cortisol, glucose, and lactate that included fixed effects of temperature cycle (constant or spiked), cortisol treatment, their interaction

(temperature cycle x cortisol treatment), and body mass (nested within temperature cycle). Models also included random effects of replicate round and bucket ID.

We analysed the interactions (CTMax: rearing temperature x cortisol treatment; thermal spikes: cortisol treatment x temperature cycle) in all models by fitting them with maximum likelihood (ML) estimations, and conducting a likelihood ratio test (LRT). If the interaction was significant, the model was refitted with restricted maximum likelihood estimation (REML), and we conducted post hoc analyses using false discovery rates (FDR, sharpened method) on pairwise comparison of interest (Pike, 2011; Verhoeven, Simonsen, & McIntyre, 2005). Using this FDR post hoc approach, we report q values, which are adjusted p values (Pike, 2011). If the interaction was determined non-significant ($p>0.05$), it was removed from the model, and main effects were tested using LRT with ML estimations. After the final model was established for CTMax, we tested the effects of maternal ID in the model (included as a random factor) using LRT. Maternal ID was not added to the statistical model of the energetic response of offspring to temperature cycle model since maternal ID was not tracked due to experimental constraints.

Results

CTMax

There was a marginally significant (at the 10% level) effect of rearing temperature by cortisol treatment interaction on CTMax (LMM, LRT: $\chi^2= 2.92$, $p= 0.087$; **Figure 3.4**, **Table 3.2**). CTMax was also influenced by rearing temperature, where fish that were raised in elevated temperatures had significantly higher CTMax than those raised in current temperatures ($\chi^2=77.9$, $p<0.001$). Cortisol dose alone did not significantly affect CTMax ($\chi^2= 0.13$, $p= 0.72$). Body mass had a marginally significant effect (at the 10% level) on CTMax

(χ^2= 2.78, p= 0.095), and thus was retained within the model. In the final model, maternal ID was a significant random effect for CTMax estimation (χ^2= 14.0, p<0.001).

Energetic response to thermal spikes

Plasma cortisol

Within the elevated temperature treatment, there was a significant cortisol treatment by temperature cycle interaction on plasma cortisol (LMM: χ^2=4.17, p= 0.041; **Figure 3.5A**, **Table 3.2**). Despite this overall effect, FDR post hoc analysis determined that there was only a marginally significant difference in plasma cortisol across temperature cycles for cortisol-dosed fish (q= 0.087), where spiked fish had lower plasma cortisol than constant temperature fish. Similarly, there was a marginally significant difference of cortisol-dosed and control-dosed offspring within the spiked-temperature treatment, where cortisol-dosed offspring had lower plasma cortisol than control offspring (q=0.087). However, there were no detectable differences across temperature cycles for the control-dosed offspring (q=0.16) nor between cortisol- and control-dosed offspring in the constant treatments (q=0.14). Within the current water temperature treatment, there was a marginally significant interaction (at 10% level) between cortisol and spike treatment (χ^2=3.66, p= 0.056), and no significant main effect of temperature cycles on plasma cortisol levels (χ^2=0.25, p= 0.62). However, cortisol treatment alone did significantly impact plasma cortisol (χ^2=7.59, p= 0.0059), where cortisol-dosed offspring had lower plasma cortisol levels than control-dosed. Body mass did not have a significant effect on plasma cortisol in either rearing temperatures (elevated: χ^2=0.92, p= 0.34; current: χ^2=0.3, p= 0.59).

Whole blood glucose

Within the elevated temperature treatment, there was no effect of cortisol and temperature cycle interaction (LMM: χ^2=0.15, p= 0.69; **Figure 3.5B**) or temperature cycle as a main effect on glucose (spike: χ^2=0.06, p= 0.80). Only cortisol treatment, as a main effect, significantly affected glucose (χ^2=5.03, p= 0.025), with cortisol-dosed offspring having higher glucose levels. Under the current temperature regime, glucose levels were not significantly impacted by cortisol and temperature cycle interaction (χ^2=0.37, p= 0.54), nor cortisol treatment as a main effect (χ^2=0.15, p= 0.70). However, temperature cycle as a main effect did significantly affect glucose (χ^2=4.14, p= 0.042), where spiked offspring had significantly lower glucose levels. Body mass did not have a significant effect on glucose in both rearing temperatures (elevated: χ^2=0.93, p= 0.34; current: χ^2=0.06, p= 0.81).

Whole blood lactate

Within the elevated temperature treatment, there was no cortisol by temperature cycle interaction on lactate concentrations (χ^2=0.94, p= 0.63; **Figure 3.5C**), nor a main effect of cortisol treatment (χ^2=2.48, p= 0.12). Temperature cycle had a marginally significant effect at the 10% level (χ^2=2.90, p= 0.09), as fish who underwent the temperature spikes had marginally higher average lactate levels. Overall lactate levels were higher in fish with a larger body mass regardless of treatment (χ^2=5.68, p= 0.017). Under the current rearing temperature, cortisol by temperature cycle interaction did not significantly affect lactate (χ^2=0.21, p= 0.65). Lactate also did not significantly differ across cortisol treatment (χ^2=1.02, p= 0.31), or temperature cycle (χ^2=0.86, p= 0.35), or mass (χ^2=1.57, p= 0.21).

Discussion

With climate change leading to increased water temperatures, we aimed to test whether maternal stress would mitigate the effects of elevated rearing temperatures on juvenile salmon. Under the environmental match hypothesis, we predicted that a prenatal cortisol dose would improve thermal performance when offspring were raised under elevated temperatures (environmental match). Similar to established work on acclimation temperature (McDonnell & Chapman, 2015), we demonstrated that elevated rearing temperatures can enhance offspring tolerance to rapid increases in temperature (i.e., CTMax). Contrary to our predictions, a prenatal cortisol signal did not noticeably modulate offspring thermal sensitivity (tolerance and energetic response) within each rearing temperature regime.

CTMax

Rearing temperature impacted offspring maximum thermal tolerance, but thermal tolerance was not further altered by exposure to egg cortisol: fish raised in elevated temperatures had a higher mean CTMax than fish raised in current temperatures. Previous work has shown that short-term acclimation to higher temperatures results in a higher CTMax (McDonnell & Chapman, 2015; Zhang & Kieffer, 2014). Long-term rearing (from early development) in elevated temperatures also led to increased CTMax (Del Rio et al., 2019; He et al., 2014; Muñoz et al., 2017; but see Chen et al., 2013). Thus, acclimation to higher temperatures may allow for organisms to persist within a warmer world under climate change. However, acclimation has a limited capacity to improve thermal tolerance, as evidence suggests that acclimation has diminishing returns on increasing upper thermal limits (Sandblom et al., 2016). Although elevated rearing temperatures improved offspring thermal tolerance, prenatal cortisol exposure did not further affect juvenile salmon CTMax. To our

knowledge, this is the first study to test the effects of prenatal stress on CTMax. Previous studies have proposed that intergenerational or transgenerational effects may be a mechanism for which fish may increase their thermal tolerance under climate change (Munday, 2014), although exposure to prenatal cortisol does not appear to be a significant maternal effect contributor to thermal tolerance and performance. Overall, we also found that CTMax was shaped by variation in juvenile body mass, where larger individuals had a higher CTMax. Mass has previously been shown to alter CTMax, with some studies reporting that larger fish (higher mass, length, or body condition) have higher CTMax (Chen et al., 2013; Gallant et al., 2017), although others have found the opposite, with larger fish having lower CTMax (Di Santo & Lobel, 2017; Messmer et al., 2017). Although not the main purpose behind our study, we did find that maternal identity was highly influential on offspring CTMax. Maternal effects are thought to play a significant role in determining how organisms respond to elevated temperatures (Burt et al., 2011). Maternal identity has been shown to account for a significant degree of intra-population differences in offspring traits (Aykanat, Bryden & Heath, 2012; Feiner, Malinich, & Höök, 2017), and our study provides further evidence that incorporating the role of maternal identity when investigating the effects of environmental stressors on offspring phenotypes is imperative.

Energetic coping to thermal spikes

Despite the significant interaction between egg cortisol treatment and temperature cycle (spiked or constant) on baseline plasma cortisol for offspring raised in elevated temperatures, we did not detect any significant post-hoc differences ($\alpha = 0.05$) across treatments. However, there were some marginally significant post hoc outcomes which suggest that cortisol-dosed offspring may have lower plasma cortisol when undergoing

stressful temperature spikes compared to both cortisol-dosed in constant conditions and control dosed in spiked conditions. This lower plasma cortisol in spiked-temperature treatment may reflect a lower demand for energy in the cortisol-dosed offspring while simultaneously coping with the temperature spikes. Within the cortisol-dosed group, we found a large degree of variation in baseline plasma cortisol under constant conditions (as seen in replicate rounds in **Figure 3.6**), with less apparent variation under the spiked conditions. Among fish exposed to the temperature spikes, control-dosed offspring had similarly greater variation in plasma cortisol. Overall, our study suggests that prenatal cortisol exposure does not impact the plasma cortisol response when fish were raised in elevated temperatures. By rearing these fish under elevated temperatures, the offspring may be more prepared for temperature spikes, which is why we may not have seen an impact of temperature spike on plasma cortisol. However, under our benign current temperature treatment, cortisol-dosed offspring had lower plasma cortisol regardless of whether the offspring had undergone the thermal spike treatment (**Figure 3.5A**). According to the environmental match hypothesis, the cortisol-dosed offspring should be mismatched to the current reared environment, and yet they had significantly lower baseline plasma cortisol than control fish. Previous work showed that cortisol-dosed offspring had a lower plasma cortisol in a benign semi-natural postnatal environment (compared to low water conditions; Capelle, 2017) and prenatal egg cortisol has been shown to alter metabolism (Sloman, 2010). However, in our current study the average plasma cortisol level for each cortisol and temperature cycle treatment was >25ng/ml, except for cortisol-dosed fish in the constant group reared at current temperatures (24.4ng/mL). Compared to previous studies in Chinook salmon (Dender et al., 2018; Capelle, 2017), the combined average for cortisol-dosed offspring (33.9ng/mL) is not abnormally low, and thus these lower plasma cortisol levels are

most likely not maladaptive. This suggests that the lower plasma cortisol responses we report may reflect a lower energetic demand in benign conditions compared to control offspring, which in turn may have long-term phenotypic consequences such as lower growth.

Among offspring reared in the elevated temperatures, cortisol-dosed fish had higher glucose than controls during the temperature-spike experiments. It is somewhat difficult to determine the origin of this response since having a higher glucose level may be indicative of glucose mobilization or higher food intake (since fish were fed throughout the experiment) (Polakof et al., 2012). Previous studies have found that fish cease feeding in warmer temperatures (Breau, Cunjak, & Peake, 2011), and thus continued feeding may indicate that cortisol-dosed fish are retaining the ability to maintain homeostasis after thermal stress. Regardless, the temperature cycle treatment itself did not impact glucose levels, suggesting that living in chronically warmer waters may allow fish to be recover faster (i.e., via habituation) to temperature spikes and maintain a higher blood glucose levels, which is furthermore enhanced in cortisol-dosed offspring. In support of this, Barton et al. (1987) found that rainbow trout (*Oncorhynchus mykiss*) exposed to 10 weeks of daily handling stress habituated to additional handling and did not increase their glucose levels as a result. Similarly, a study comparing energetic responses to exposure between one and multiple temperature spikes found that after multiple thermal spikes, fish were able to maintain glucose levels by inducing anabolic metabolism and replenishing glycogen reserves (Callaghan et al., 2016). Since elevated-temperature-reared fish were chronically exposed to increased temperatures, they may be using an anabolic phenotype to respond to temperature spikes, potentially allowing for the maintenance of higher glucose levels. Alternatively, after being reared under current temperatures, fish undergoing temperature spikes had lower glucose

levels, regardless of egg cortisol treatment. This suggests that fish undergoing the temperature spikes may have incurred an energetic cost, thus reducing their reserves and glucose homeostatic concentrations when raised in a benign environment.

Across both rearing temperatures, neither cortisol treatment nor temperature cycle influenced offspring plasma lactate, although among fish reared in elevated temperatures, plasma lactate was higher in fish with a larger body size. Since we ensured that oxygen levels remained high during the temperature spikes (via air stones), anaerobic metabolism may not have been necessary for the fish to persist in the higher temperatures, which may be why we did not detect differences across temperature cycle treatments. Furthermore, since lactate is a by-product of anaerobic metabolism in muscle tissues (Dando, 1969), larger fish having higher lactate levels may be due to a higher proportion of muscle tissue available.

Potential for maternal stress to adjust thermal performance

Overall, we did not find that prenatal cortisol improved thermal tolerance when fish were raised under elevated water temperatures. Contrary to predictions of the environmental match hypothesis, egg cortisol treatment did not affect CTMax nor did egg cortisol interact with rearing temperature to clearly modulate the energetic response to temperature spikes. Prenatal cortisol did lead to increased blood glucose in elevated-temperature-reared offspring, in theory allowing these offspring to have energy more readily available to cope with additional thermal stressors in elevated temperatures. Although prenatal stress has been shown to improve energetic responses to stressful postnatal environments (e.g., low water conditions: Capelle 2017), and has shown to alter a host of offspring traits (Burton et al., 2011; Sloman, 2010; Sopinka et al., 2017), our study suggests that maternally derived GCs do not noticeably improve thermal tolerance under elevated water temperature conditions in fish. Thus, maternal

GCs may not be able to adjust thermal tolerances via environmental matching, even in combination with elevated rearing temperatures. Nonetheless, there is some evidence in other studies for environmental matching to improve thermal tolerance when both parents and offspring are exposed to elevated temperatures (Le Roy & Seebacher, 2018; Le Roy et al., 2017; Sandblom et al., 2016). For example, Donelson et al (2012) found that offspring reared under elevated water temperatures whose parents were also raised in elevated temperatures were able to maintain their aerobic scope (an indicator of ability to complete aerobic activities). Other pathways for maternal effects to impact thermal responses (i.e., epigenetics and maternal provisioning) may allow for a mechanism by which environmental matching can improve offspring performance in warming waters (Munday, 2014). Thus, further testing the role of intergenerational stress within the environmental context of climate change remains imperative to determine which mechanisms will enable organisms to rapidly respond to global warming.

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Tables

Table 3.1: The starting dates for the CTMax and thermal spike experiments with the respective accumulated thermal units (ATUs).

	CTMax		Thermal Spike	
	Starting Date	Accumulated thermal units (ATUs)	Starting Date	Accumulated thermal units (ATUs)
Current	21-26 June 2017	2284-2359	12-15 July 2017	2637-2694
Elevated	1-7 May 2017	2251-2337	25-28 June 2017	2611-2665

Table 3.2: Thermal tolerance performance and phenotype metrics (mean ±SE) of juvenile chinook salmon.

		Temp Cycle	Current Cortisol-dosed	N	Current Control	N	Elevated Cortisol-dosed	N	Elevated Control	N
CTMax	CTMax (°C)	--	28.4 ±0.09	59	28.3 ±0.07	57	28.9 ±0.03	59	28.9 ±0.03	58
	Mass (g)	--	1.77 ±0.14	59	1.74 ±0.13	57	2.20 ±0.15	59	2.14 ±0.14	58
Temperature Cycle	Cortisol ($ng*mL^{-1}$)	*Constant*	24.4 ±5.8	15	55.8 ±9.8	14	58.1 ±12.4	14	30.3 ±6.0	15
		Spike	43.3 ±9.8	15	42.5 ±5.9	15	21.8 ±3.4	15	50.2 ±8.2	14
	Glucose ($mmol*l^{-1}$)	*Constant*	2.29 ±0.08	15	2.39 ±0.11	14	2.53 ±0.15	14	2.16 ±0.09	15
		Spike	2.17 ±0.09	15	2.16 ±0.09	15	2.31 ±0.05	15	2.32 ±0.09	14
	Lactate ($mmol*l^{-1}$)	*Constant*	2.03 ±0.08	15	2.29 ±0.18	14	2.10 ±0.18	14	1.73 ±0.12	15
		Spike	2.30 ±0.18	15	2.48 ±0.27	15	2.23 ±0.17	15	2.00 ±0.21	14
	Mass (g)	*Constant*	4.45 ±0.53	15	4.40 ±0.40	14	5.94 ±0.72	14	4.62 ±0.60	15
		Spike	4.00 ±0.34	15	4.48 ±0.32	15	4.64 ±0.62	15	3.57 ±0.55	14

Figures

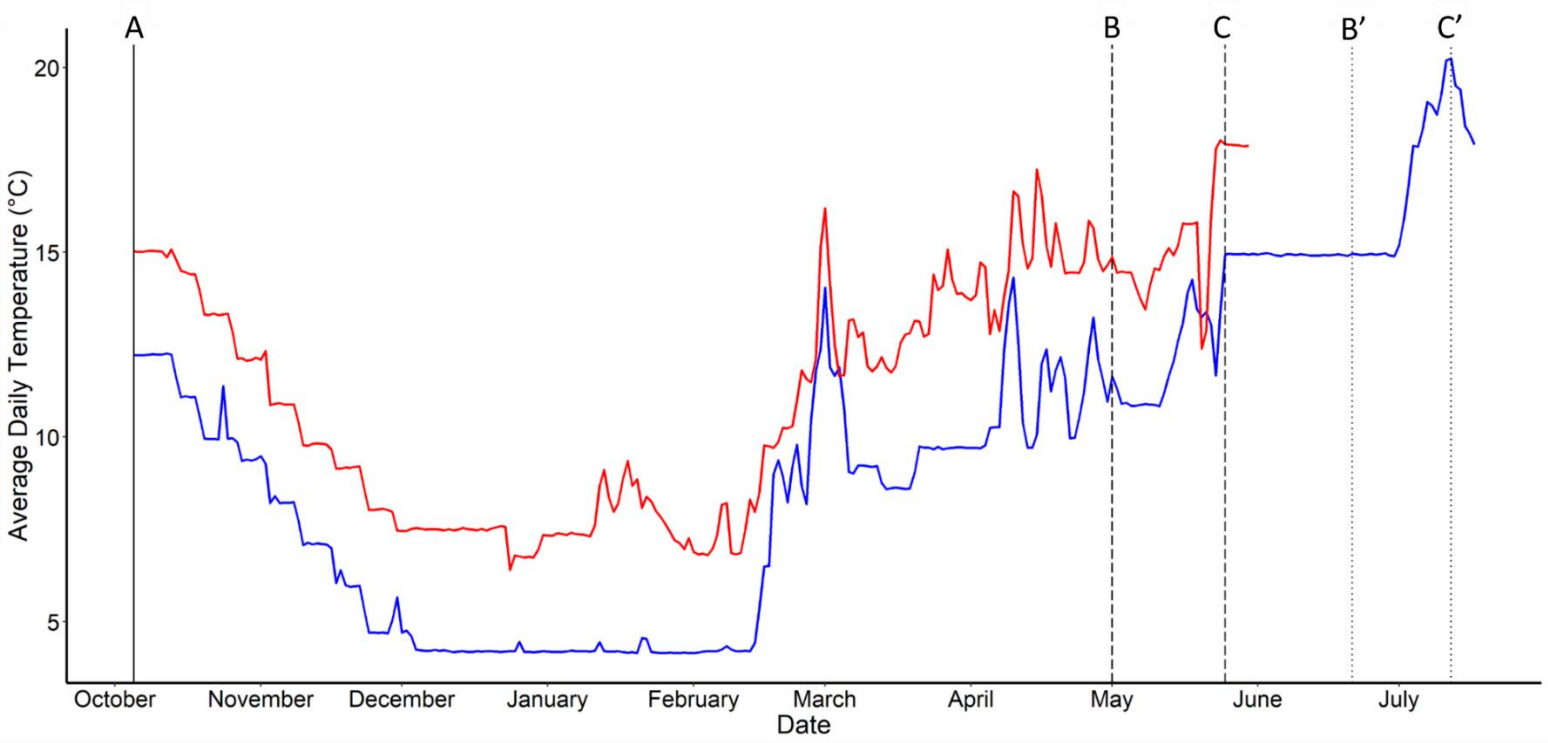

Figure 3.1: Recorded average daily temperature for each temperature regime in the rearing experimental design. Current temperature regime is represented by the blue line while the elevated temperature regime is in red. Vertical lines represent sampling timepoints: solid—both current and elevated groups; dashed—elevated, dotted—current. Timepoints are: **A**—Fertilization (Oct 4th), **B** & **B**'—CTMax start date, and **C** & **C'**—Thermal spike start date.

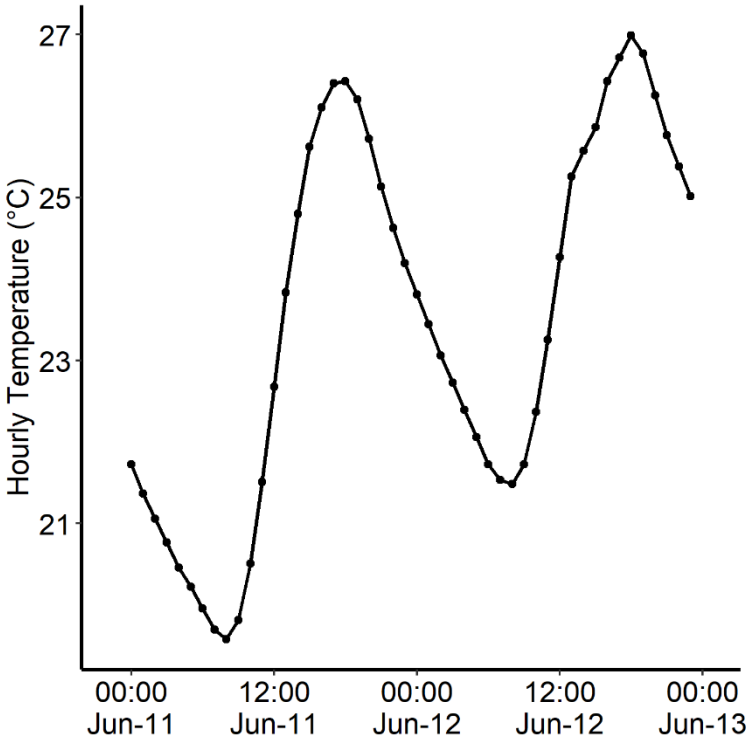

Figure 3.2: River temperatures from natal stream (Credit River: 43°34'40.0"N 79°42'06.3"W) during June 11th-13th 2017 (HOBO®: Water temp pro v2). Highest temperature (27.0°C) reached on June 12th, 2016 at 6pm.

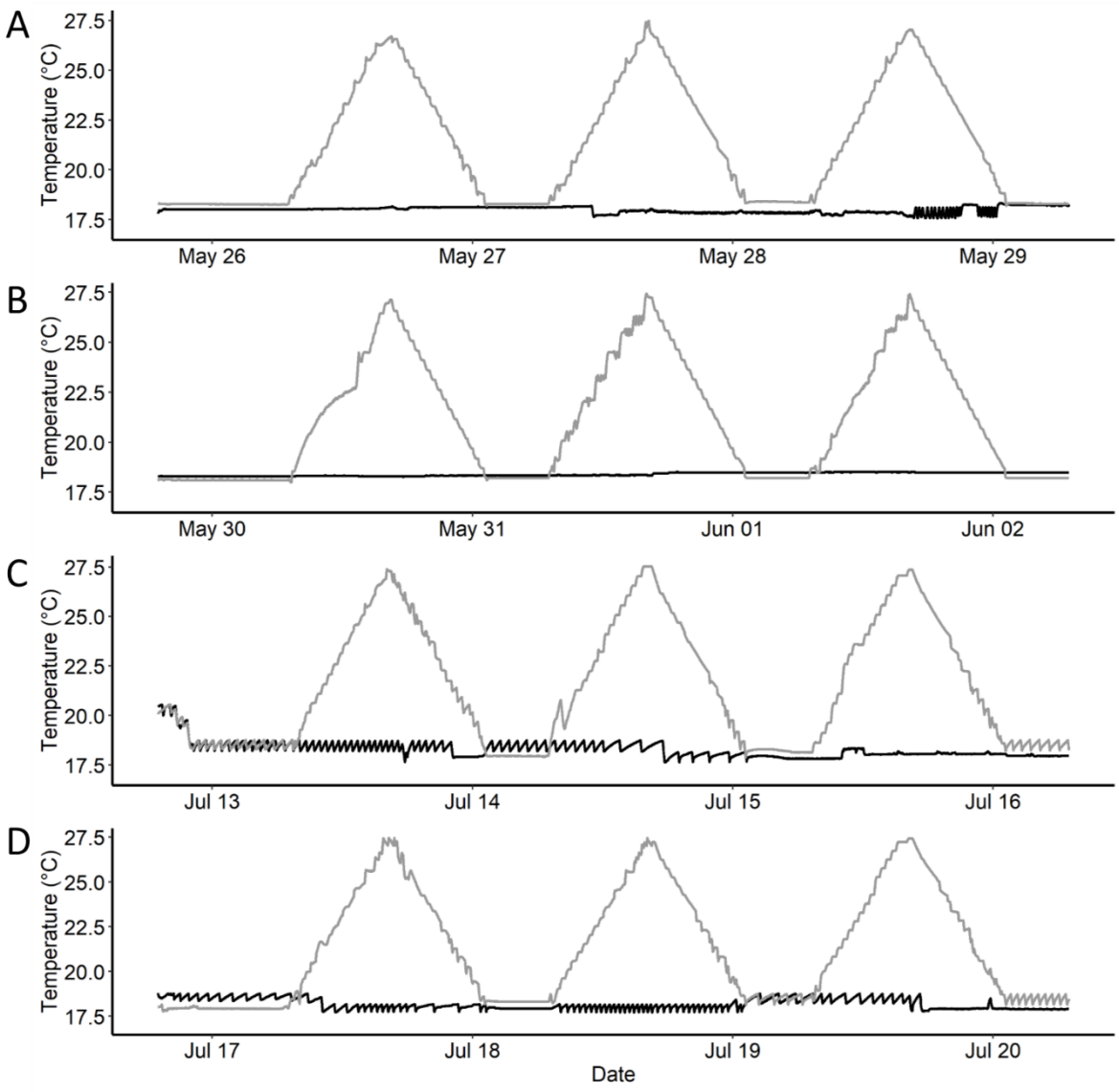

Figure 3.3: Temperature recording from temperature loggers (HOBO®: Water temp pro v2) that measured water temperature once every minute. Black lines represent the constant temperature groups (~18°C) and the grey line represents the spike groups (+9°C per day). **A-D—**represent Round 1 to 4 respectively.

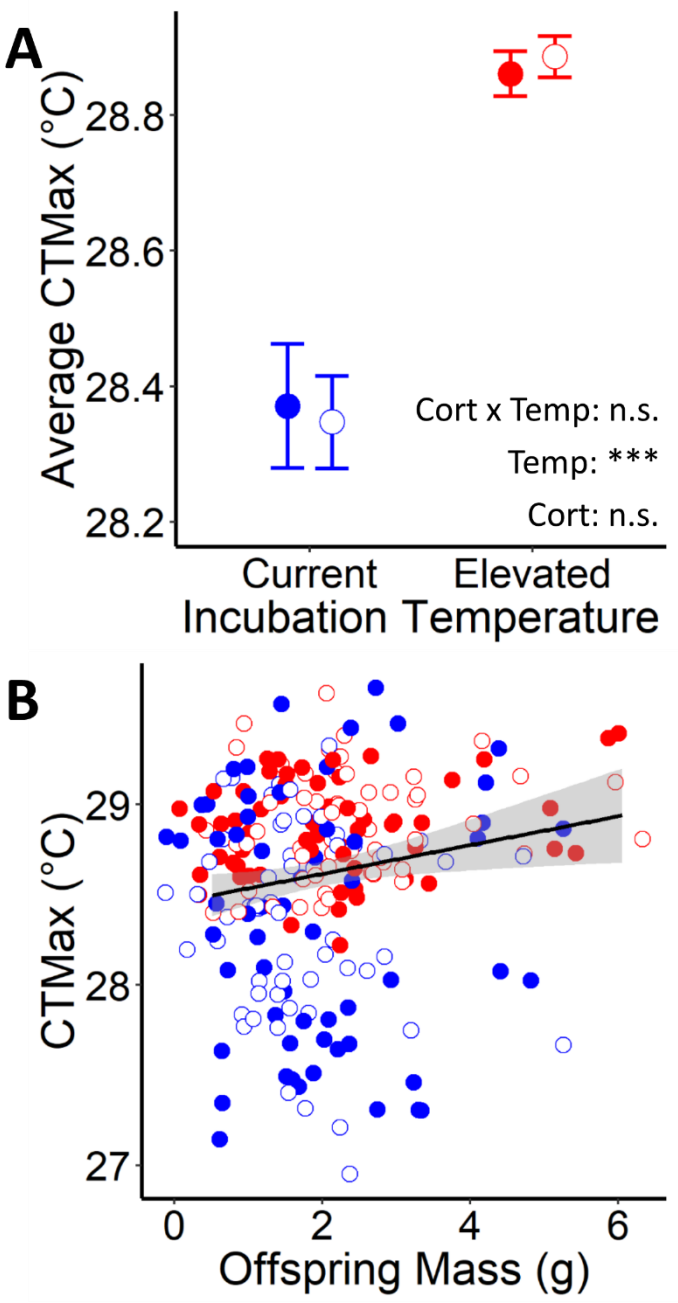

Figure 3.4: **A**—Effects of rearing temperature and prenatal cortisol on average CTMax. **B**—Relationship between CTMax and offspring body mass. Open circles depict control and closed circles depict cortisol-dosed. Blue points are fish raised in current temperature regime, while red points depicts those raised in elevated temperatures. n.s. and *** represent p values that were >0.05 and <0.001, respectively.

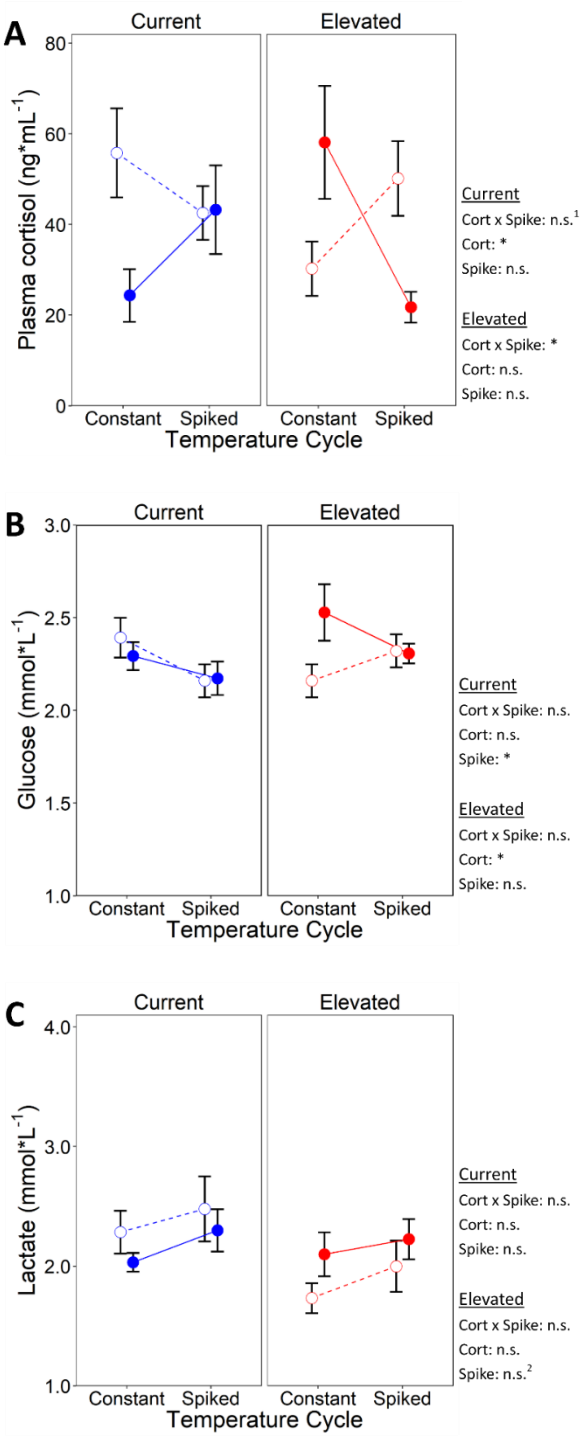

Figure 3.5: Effects of cortisol treatment and temperature cycle on **A**—plasma glucose, **B**—whole blood glucose, and **C**—whole blood lactate. Open circles depict control and closed circles depict cortisol-dosed. Blue points are fish raised in current temperature regime, while red points depicts those raised in elevated temperatures. n.s. and * represent a p value of >0.05 and <0.05, respectively. n.s.[1] and n.s.[2] represents a marginal significance of p=0.056 and p=0.090 respectively.

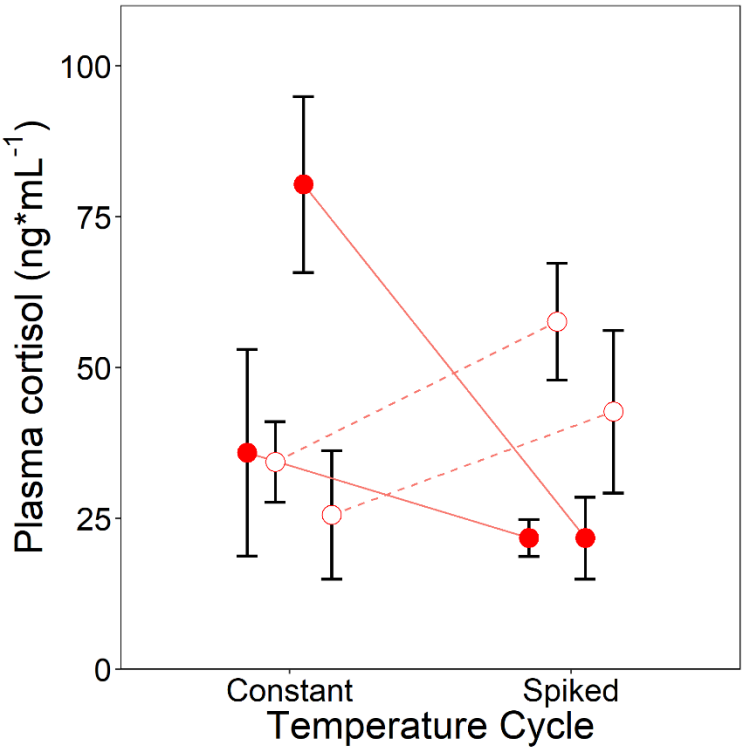

Figure 3.6: Plasma cortisol levels plotted by replicate (rounds) from offspring raised in elevated temperatures. Open circles depict control and closed circles depict cortisol-dosed.

CHAPTER 4 — GENERAL DISCUSSION

Applying the environmental match hypothesis to climate change

Since climate change is altering environments rapidly, organisms may be unable to persist. Thus, any mechanisms that allow for organisms to adapt to these environmental changes should be further explored. Mechanisms such as phenotypic flexibility, developmental plasticity, contemporary evolution, and intergenerational plasticity enable organisms to persist in the face of environmental stressors (Fusco & Minelli, 2010; Piersma & Drent, 2003; Stockwell, Hendry, & Kinnison, 2003). For example, through intergenerational plasticity, the environment experienced by the parents can induce phenotypic changes in their offspring, with subsequent (and sometimes positive) effects on offspring performance and fitness (Mousseau & Fox, 1998). Maternal stress is an inducer of intergenerational plasticity that occurs when stress that mothers encounter is passed onto offspring during gestation or follicular recruitment, resulting in altered offspring phenotypes (Sheriff & Love, 2013). Glucocorticoids (GCs) are a common mediator of environmental quality between the maternal and offspring environments, and maternally-derived GCs have been shown to induce a number of phenotypic changes in offspring across a variety of taxa (Love et al., 2005; Sopinka et al., 2016). According to the environmental match hypothesis, maternal stress may act as a predictive signal to offspring when the maternal environment is indicative of the environment offspring are expected to encounter, in turn, better preparing them for future stressors (Sheriff & Love, 2013). Recent studies that have considered the effects of maternal stress within the context of environmental or life-history variation have provided support for the adaptive role of maternally-derived GCs where offspring exposed to maternal stress have higher

performance when raised in similarly stressful environments (e.g., Capelle, 2017; Dantzer et al., 2013; Love et al., 2005).

The overall aim of my thesis was to test the environmental match hypothesis by examining whether prenatal cortisol (1000ng*mL^{-1} cortisol-dosed compared to control 0ng*mL^{-1}) could dampen the effect of elevated rearing temperatures (elevated +3°C compared to current 0°C). Taken together, my thesis suggests that thermal stress is an overriding modulator of offspring phenotype, and that prenatal stress had no or minimal effects on offspring phenotype when fish are reared under elevated water temperatures. In Chapter 2, rearing offspring in elevated water temperatures resulted in lower survival and smaller offspring, with no effects of prenatal cortisol on survival, offspring size, nor body condition. In Chapter 3, offspring reared under elevated water temperatures had higher thermal performance (higher CTMax), with no additional effects of prenatal cortisol. When examining the effects of temperature spikes on the energetic responses of offspring, I found some effects of prenatal cortisol exposure in response to the temperature spikes. The interaction of prenatal cortisol and temperature spike treatment significantly impacted offspring plasma cortisol, however this interaction only had marginally significant post-hoc differences. Cortisol-dosed offspring who were raised in elevated temperatures and exposed to temperature spikes had marginally lower plasma cortisol than cortisol-dosed kept at constant temperature conditions, and control-dosed offspring exposed to temperature spikes. Moreover, prenatal cortisol did affect plasma and blood glucose regardless of temperature spike treatment. Cortisol-dosed offspring raised in current temperatures had lower plasma cortisol and cortisol-dosed offspring raised in elevated temperatures had higher blood glucose than control-dosed offspring. Together, these results suggest that exposure to prenatal cortisol modulated baseline

energetics of offspring, but did not assist with responses to additional thermal stressors (such as temperature spikes). To place my results into context, I explore the factors contributing to offspring phenotypes that in turn, determine their ability to cope with climate change (with an emphasis on increasing temperatures). I examine how environmental factors affect thermal tolerance, how egg cortisol can act as an adaptive maternal signal, how alternative maternal effects can shape offspring responses, and finally, how environmental matching may induce higher offspring performance in a warming world. I then discuss implication for fisheries managers and future avenues of research.

Quantifying the effects of environmental factors on thermal tolerance

Recent research focused on plastic responses to changing environmental conditions has concentrated on climate change (Merilä & Hendry, 2014). As climate change is expected to increase average temperatures, investigating the role of plasticity on altering the thermal tolerance of organisms facing climate change is becoming increasingly important. Studies have shown that thermal tolerance in fish can be shifted up or down through several environmental factors such acclimation temperature (McDonnell & Chapman, 2015) and oxygen levels (Del Rio et al., 2019). Thus, increases in thermal tolerance may allow aquatic organisms to persist in warmer waters. Upper thermal tolerance is determined by two main mechanisms: oxygen (i.e., a mismatch between oxygen demand & supply: oxygen- and capacity-limited hypothesis) and subcellular (i.e., enzyme denaturation) limitations (Gangloff & Telemeco, 2018; Pörtner & Knust, 2007). As a result of physiological limits, the degree to which thermal tolerances can shift upwards is restricted. For instance, my thesis revealed that rearing juvenile salmon in elevated temperatures (+ 3°C) led to increase in CTMax, but that this increase in average thermal tolerance was only ~0.5°C. Studies have suggested that the

upper thermal tolerance is less flexible (Sandblom et al., 2016), and my thesis has confirmed that the effects of rearing temperatures has diminishing returns. Indeed, if this ~0.5°C increase in thermal tolerance has lasting effects on fish performance and fitness, further studies are needed where fish raised in elevated temperatures are tested *in situ* or tracked in the wild.

When organisms reach their CTMax, they lose their ability to respond to their environment and in fish this is commonly the loss of equilibrium (Lutterschmidt & Hutchison, 1997). In the wild, CTMax is the temperature at which organisms are unable to escape the heat, forage, or escape predation, resulting in fitness consequences. However, there are limitations to using CTMax as the only form of determining thermal tolerance since organisms may incur negative effects at temperatures lower than CTMax. Energetic demands for maintenance during thermal stress may lead to reduced energy available for growth, foraging, and reproduction (Neuheimer et al., 2011). A proposed metric for quantifying this effect is the agitation temperature which is the temperature at which organism display escape behaviours and indicates when fish may behaviourally respond to increasing temperatures (McDonnell & Chapman, 2015). However, agitation temperature is not suitable metric for all species because not all species will display obvious increases in activity or escape behaviour within a laboratory setting (i.e., if the species already has an active baseline behaviour). Since thermal tolerance can also alter physiological processes, measuring changes in energetic and stress indicators in the tissues (such as blood, liver, and gills) after exposure to thermal stress may indicate thermal tolerance (Callaghan et al., 2016; Corey et al., 2017). Through my temperature spike experiment, I was able to detect the effects of prenatal cortisol on offspring tolerance that I was not able to detect through CTMax. However, interpretation of these energetics is often difficult since both increases and decreases can be argued to be beneficial

responses (Romero, 2004). Thus, quantifying additional traits alongside these markers, such as how DNA transcription may be altered by thermal stress—especially changes in the transcription of metabolism, growth, and stress genes—provides for a more holistic approach to determine when waters are too warm for a given species.

Egg cortisol as a maternal stress signal

Maternal exposure to stressors has been shown to result in higher maternal GCs and in turn higher egg GC levels in fish (Stratholt, Donaldson, & Liley, 1997 but see Ghio et al., 2016; Sopinka et al., 2014). Furthermore, experimentally increasing maternal plasma GC levels (via injections, implants, etc.) often results in increased egg GC levels (Eriksen et al., 2006; Gagliano & McCormick, 2009; Hayward & Wingfield, 2004). Increased egg GC levels has been shown to result in altered offspring phenotypes (see Chapter 1 for extensive review). However, there remains vast variation in egg cortisol within and across fish species, which may have consequences on how egg cortisol ultimately shapes offspring phenotypes. In my thesis, within the unfertilized and non-manipulated eggs, there was still significant variation (range= 1.89-151.5 ng*g-1). One study examining hatchery-produced Chinook salmon found lower levels of egg cortisol (range=5.02-27.21 ng*g-1), but this may be due to the fact that the population lived in relatively stress-free hatchery conditions, whereas my dams were from a wild Credit River population. Similarly, chickens (*Gallus gallus domesticus*) raised on farms have lower levels and lower inter-individual variation in baseline glucocorticoid levels than other avian species (Cockrem, 2013; Henriksen, Groothuis, & Rettenbacher, 2011). Sopinka et al. (2017) reported differences across wild populations of Chinook salmon on the North American West Coast, and attributed the differences to migration length (with higher egg cortisol found in the population with the longer migration). The Credit River population has a

relevantly short migration (~14km) compared to many other Chinook populations (Thorn et al., 2018), perhaps indicating that the higher and more variable egg cortisol levels could be due to other factors than migration distance. First, female salmon may be experiencing more environmental stressors than other populations prior to and during migration including increased temperatures, reduced water quality, or habitat degradation, due to the high anthropogenic impacts documented in the Great Lakes (Smith et al., 2015). Second, there may be a genetic basis for the differences, since Great Lake populations may be experiencing a founder effect given that Great Lakes Chinook salmon originated from a single population from Green River, Washington (Parson, 1973; Weeder, Marshall, & Epifanio, 2005). By establishing the extent of natural egg cortisol variation within and across fish populations, as well as the environmental and genetic factors that help to contribute to this variation, researchers can begin to establish whether variation in maternally-derived cortisol may be selected for in relation to variation in environmental stressors (Love & Williams, 2008).

Some experiments investigating the effects of cortisol at multiple doses have found that offspring phenotypes are dependent on the cortisol dose given to the offspring (Capelle, 2017; Eriksen et al., 2006; Li et al., 2010). For instance, Li et al (2010) found that offspring exposed to the low egg cortisol bath ($100ng*ml^{-1}$) had higher growth than both control and high dosed ($1000ng*ml^{-1}$) offspring. Similar patterns emerged in studies that examined offspring performance in low water (drought stimulated) conditions, where low-dosed ($300ng*ml^{-1}$) offspring had the highest performance (i.e., lower energetic demand, maintenance of a higher stress response, maintenance of body size, higher refuge use) compared to high-dosed offspring ($1000ng*ml^{-1}$) which had lower growth, higher energetic demand and decreased stress responsiveness (Capelle, 2017). In my thesis, prenatal cortisol

dose had few effects on offspring phenotype raised under elevated temperatures (except for significant interaction in plasma cortisol across cortisol treatment and temperature cycle and the higher blood glucose concentration in cortisol-dosed offspring). Although the concentrations chosen for my thesis were based on previous literature on prenatal cortisol, the dose may have not been the appropriate one expected to match offspring phenotype to +3°C elevated temperatures.

Maternal effects beyond maternally-derived cortisol

Parental effects beyond maternally-derived cortisol can greatly influence how offspring are expected to react to both prenatal cortisol and their natal environment (Munday, 2014). In respect to the role that intergenerational plasticity has to play in responding to climate change, maternal effects have been found to greatly influence offspring response to elevated temperatures during early development (Burt, Hinch, & Patterson, 2011). By using a cortisol bath to mimic maternal stress, my thesis was able to isolate the role of prenatal cortisol on offspring phenotypes from the genetic and non-genetic influences of each mother. Since each mother may also influence how offspring responded to prenatal cortisol and elevated rearing temperatures, maternal identity was included as a random effect (with the exception of the temperature spike experiment). Overall, I found a highly significant impact of maternal identity on early life morphology, survival, and thermal tolerance, further suggesting the importance of individual maternal effects when assessing the impacts of maternal- and thermal stress. These additional maternal effects could be translated to offspring through maternal provisioning (e.g., egg size and composition: Heath, Fox, & Heath, 2016; Thorn & Morbey, 2018; Thorn et al., 2018), or non-genetic inheritance (i.e., epigenetic inheritance: Bonduriansky, Crean, & Day, 2012; Day & Bonduriansky, 2011; Jablonka & Raz, 2009).

Thus, when a mother is stressed, her pre-existing condition and/or stress phenotype prior to stressor exposure may have additional influences on how offspring respond to elevated temperatures (Munday, 2014). Furthermore, mothers can use maternal effects to match offspring phenotypes to future environments (environmental matching; Sheriff & Love, 2013).

An alternative intergenerational response to environmental stressors is bet-hedging, which occurs when a mother induces a greater variation in offspring traits in order to increase chances that some of her offspring will survive (Herman et al., 2014; O'Dea et al., 2016). Environmental matching is advantageous when offspring environment is predicted by the maternal environment whereas bet-hedging should be selected for when future environmental conditions are inconsistent (Shama, 2015). Egg GCs has been proposed as a potential bet-hedging strategy in birds (Love et al., 2005), but there is limited evidence for hormone-mediated bet-hedging. Some evidence of bet-hedging has been found on egg size in fish (Morrongiello et al., 2012), but no studies to my knowledge have found evidence for egg cortisol as a mechanism of bet-hedging. In my thesis, during my temperature spike experiment, there was great variation in plasma cortisol levels in offspring reared in elevated temperatures and were kept at constant temperatures, which may be suggestive of a bet-hedging strategy. However, since the cortisol-dosed offspring raised in elevated temperatures had reduced variation in the temperature spike condition, bet-hedging may not be the underlying strategy of maternally-derived cortisol. Maintaining a higher variation in constant conditions may allow offspring to allocate energy and increase foraging in benign conditions to maximize growth, but under stressful conditions, lowering energy demand in order to maintain homeostasis may be beneficial.

Evidence for environmental matching under elevated temperatures

Although my thesis provided only partial evidence for the role of prenatal cortisol as a maternal signal of offspring environmental quality, there have been studies supporting the role of adaptive parental (maternal & paternal) effects when parents and offspring share similar environments (i.e., environmental match; Donelson et al., 2018). For instance, when both parents and offspring are reared under the same elevated temperatures, results suggest that the altered offspring traits provide for higher performance in warmer waters (Donelson, Munday, & McCormick, 2012; Le Roy & Seebacher, 2018; Le Roy, Loughland, & Seebacher, 2017; Salinas & Munch, 2012; Shama et al., 2014). In addition, offspring reared in elevated temperatures whose parents were also reared in warmer waters often grew more quickly (Donelson et al., 2012; Shama et al., 2014), maintained higher metabolic scope (Le Roy et al., 2017), and had higher swimming performance (Le Roy & Seebacher, 2018; Le Roy et al., 2017). Together, these traits may provide offspring with higher performance in warmer waters. For instance, by maintaining a higher swimming performance, fish may be able to escape additional stressors such as increased predation attempts, and maintain higher foraging rates even within these warmer waters (Plaut, 2001). While these experiments did not explicitly test for the role of maternal stress under the environmental match hypothesis, they nonetheless provide evidence that when the stressful parental environment matches the severity of the offspring environment, these offspring may incur a performance advantage. Since these effects were seen within one or two generations, it is unlikely that these effects are due to contemporary evolution, but rather through intergenerational or transgenerational plasticity that may be translated through nutritional provisioning, hormones, and epigenetics (Jonsson & Jonsson, 2016; Sheriff & Love, 2013). Thus, even though my prenatal cortisol

treatment did not clearly induce environmental matching between parental and offspring environment, there is evidence that environmental matching may still play a key role in the rapid responses of organisms to environmental change.

Implications for fisheries managers

Many Pacific salmon populations are threatened or in decline (COSEWIC, 2018), hence increased water temperatures (as expected under climate change) may further exacerbate the decline of these populations (Martins et al., 2012). Coupled with increases in temperature, there may be a higher rate of mortality (Crossin et al., 2008; Crozier et al., 2010), increases in parasite and disease load (Ray, Holt, & Bartholomew, 2012), declines in food availability (Chittenden et al., 2010), increases in metabolism (Enders & Boisclair, 2016), and changes in behaviour (e.g., fleeing high temperatures: Breau, Cunjak, & Peake, 2011, fleeing predation: Marine & Cech, 2004). Thus, hatchery programs should ideally target individual wild female salmon that have been able to persist and reach the spawning ground despite elevated temperatures for stock propagation. If programs are using captive adults to propagate hatchery stock, managers could acclimate adult salmon to a higher temperature to utilize environmental matching between the maternal and offspring environment which may better prepare offspring for increased water temperatures. Managers may also raise juveniles at higher temperatures, although higher temperatures can result in higher mortality in some early life stages (as my thesis found in early development), the surviving offspring are expected to have higher thermal tolerances, and ultimately may have a higher fitness. Additionally, managers may use prenatal cortisol dose as a signal of elevated temperatures by using a dose curve to determine which cortisol concentration results in the highest offspring performance. Overall, incorporating the effects of intergenerational effects and whether juveniles are

matched to the river they will be released in may enhance hatchery efforts to restore Pacific salmon populations.

Future Directions

My thesis tested the environmental match hypothesis by examining the interaction of prenatal cortisol and elevated temperatures, and found limited effects of prenatal cortisol on early life development and thermal tolerance. However, my study highlights the importance of developmental plasticity, as elevated rearing temperatures increased offspring acute thermal tolerance. My thesis provides the basis for many future studies examining the interaction between maternal- and thermal stress. First, experiments should assess alternatives to CTMax to describe thermal tolerance by including metrics such as behavioural responses (e.g., agitation temperatures) and transcriptional responses to heat stress. Second, studies should quantify natural variation in egg cortisol levels within and across populations to determine the relationship between maternal stressor severity and egg cortisol concentrations. Additionally, more work is required investigating egg cortisol levels after experimentally exposing mothers to stressors, which includes establishing what type of stressors may lead to the transfer of maternal cortisol, the duration of the stressor, and the role of multiple stressors in modulating maternal signals to offspring. Third, studies could examine how offspring respond to experimentally elevated cortisol-dose in relation to their natural egg cortisol levels (i.e., does individuals who already had high cortisol levels incur benefits from additional cortisol, or does additional cortisol only benefit individuals who already had low cortisol levels). Fourth, more experimental work is required to determine what additional intergenerational mechanisms (beyond maternally-derived cortisol) could be translating maternal stress to offspring to allow for environmental matching (e.g., nutritional provisioning and non-genetic

inheritance). Fifth, studies should integrate the role of individual maternal effects when examining intergenerational effects on a population level. For example, Feiner et al. (2017) suggested that individual maternal effects can account for a large component of intra-population variation when examining offspring traits, and thus should be incorporated into analyses when examining environmental effects on offspring phenotypes (e.g., in the form of female condition, maternal plasma cortisol, unfertilized egg cortisol, nutritional quantity and composition in eggs, etc.). Lastly, more experimental studies are needed that test the environmental match hypothesis using ecologically relevant stressors (including human-induced rapid environmental changes). Within this context, researchers should begin examining the effects of multiple stressors under the environmental match hypothesis since organisms rarely experience stressors in isolation.

Conclusion

Maternal stress may play an adaptive role in preparing offspring for a stressful environment, especially for a world impacted by human-mediated climate change. My thesis tested the adaptiveness of maternal stress by using an environmentally relevant two by two design to test the interaction between prenatal cortisol and rearing temperature on offspring phenotype, performance and survival within the environmental match hypothesis framework. Although there were limited effects of prenatal cortisol on offspring phenotypes, my study highlights the significant role that thermal stress has to play in modulating offspring phenotype and performance. Fish and fisheries may be able to acclimate to warmer waters through developmental plasticity. This project highlights the importance of testing offspring performance and fitness within environmentally relevant contexts, and especially the importance of environmental effects on early-life development, survival and performance.

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