Oecologia (2014) 175:493–500
DOI 10.1007/s00442-014-2915-9

BEHAVIORAL ECOLOGY - ORIGINAL RESEARCH

Mother knows best, even when stressed? Effects of maternal exposure to a stressor on offspring performance at different life stages in a wild semelparous fish

N. M. Sopinka · S. G. Hinch · C. T. Middleton · J. A. Hills · D. A. Patterson

Received: 9 October 2013 / Accepted: 25 February 2014 / Published online: 12 March 2014

Abstract The environment mothers are exposed to has resonating effects on offspring performance. In iteroparous species, maternal exposure to stressors generally results in offspring ill-equipped for survival. Still, opportunities for future fecundity can offset low quality offspring. Little is known, however, as to how intergenerational effects of stress manifest in semelparous species with only a single breeding episode. Such mothers would suffer a total loss of fitness if offspring cannot survive past multiple life stages. We evaluated whether chronic exposure of female sockeye salmon (*Oncorhynchus nerka*) to a chase stressor impaired offspring performance traits. Egg size and early offspring survival were not influenced by maternal exposure to the repeated acute stressor. Later in development, fry reared from stressed mothers swam for shorter periods of time but possessed a superior capacity to re-initiate bouts of burst swimming. In contrast to iteroparous species, the mechanisms driving the observed effects do not appear to be related to cortisol, as egg hormone concentrations did not vary between stressed and undisturbed mothers. Sockeye salmon appear to possess buffering strategies that protect offspring from deleterious effects of maternal stress that would otherwise compromise progeny during highly vulnerable stages of development. Whether stressed sockeye salmon mothers endow offspring with traits that are matched or mismatched for survival in the unpredictable environment they encountered is discussed. This study highlights the importance of examining intergenerational effects among species-specific reproductive strategies, and across offspring life history to fully determine the scope of impact of maternal stress.

Keywords Burst swimming · Egg cortisol · Intergenerational effects · Sockeye salmon · *Oncorhynchus nerka*

Communicated by Marc Mangel.

N. M. Sopinka (✉) · S. G. Hinch · C. T. Middleton
Pacific Salmon Ecology and Conservation Laboratory, Department of Forest and Conservation Sciences, University of British Columbia, 2424 Main Mall, Vancouver, BC V6T 1Z4, Canada
e-mail: natsopinka@gmail.com

J. A. Hills · D. A. Patterson
Fisheries and Oceans Canada, Science Branch, Pacific Region, Cooperative Resource Management Institute, School of Resource and Environmental Management, Simon Fraser University, Burnaby, BC V5A 1S6, Canada

Introduction

With a growing multitude and intensity of anthropogenic and environmental stressors comes a need to understand interactions between ecological stressors, animal physiology, and behaviour. Existing knowledge on how stress modifies traits within and across generations is primarily derived from biomedical research on domesticated species (Romero 2004), and, to date, key predictions [e.g., elevated glucocorticoid (GC) levels] are only modestly corroborated by trends emerging from wild populations (Dickens and Romero 2013). Free-living populations exposed to ecological disturbances do demonstrate endocrine changes that can be implicated in the reprogramming of offspring performance (Love et al. 2013; Sheriff and Love 2013). Such intergenerational effects are heavily attributed to altered maternal investment into offspring, as maternal provisioning (e.g., nutrients, hormones, parental care) typically exceeds the genetic investment of fathers (Mousseau and Fox 1998). Indeed, maternal stress influences various

offspring parameters in vertebrates with multiple reproductive opportunities (e.g., birds; Henriksen et al. 2011), allowing adjustment of current reproductive investment when faced with unfavourable breeding conditions (Love and Williams 2008). Theoretical and experimental evidence suggests that the resonating evolutionary consequences of transformed developmental trajectories can potentially shape how individuals will cope with future environmental change (Räsänen and Kruuk 2007; Badyaev and Uller 2009; Love et al. 2013). Currently lacking is how intergenerational effects of exogenous maternal stress manifest in semelparous species where there is no opportunity to postpone reproduction when under stressful conditions.

The striking contrasts between semelparous and iteroparous reproductive life histories are apt to influence maternal effects of stress (Heath and Blouw 1998). With only a single chance to maximize fitness, semelparous mothers must arm offspring with resources that will ensure adequate growth and survival, especially in the absence of or limited parental care. Arguably, due to immense selection pressure, semelparous species should be evolved to persist through ecological stressors without compromising reproduction (Wingfield and Sapolsky 2003). However, recent studies show that, under simulated conditions of ecological stress, senescing animals can mount a glucocorticoid stress response (Cook et al. 2011; Donaldson 2012) and reproduction can be impaired (e.g., incomplete egg release, McConnachie et al. 2012; depressed reproductive hormone concentrations, Jeffries et al. 2012). Furthermore, Heath and Blouw (1998) showed that maternal effects (e.g., body size affecting egg size) are stronger in semelparous fish than they are in iteroparous species. This relationship, coupled with potential reproductive sensitivity to stressors, suggests that offspring of semelparous mothers may be particularly responsive to maternal effects of stress. Generally, it is accepted that maternal effects of stress are predominantly deleterious. Evidence is now growing that stressed mothers may confer adaptive information to their offspring (Love et al. 2013), and that maternal effects initially described as negative may actually increase fitness of both offspring and mother when "matched" to stressful environmental conditions (Sheriff and Love 2013). Disentangling these contrasting (but not necessarily mutually exclusive; see Coslovsky and Richner 2011) types of maternal effects is especially pertinent given the recent declines of semelparous populations of Pacific sockeye salmon (*Oncorhynchus nerka*; Cohen 2012), a commercially, culturally, and ecologically significant species that is exposed to a gauntlet of ecological stressors during reproductive maturation.

In contrast to other vertebrate classes, the mechanisms and specific outcomes of maternal effects of stress are largely unknown in wild fishes. Cortisol, the primary GC in fish, is the most widely accepted maternally-derived candidate driving observed 'downstream' effects of maternal stress (Schreck et al. 2001), yet the developmental role of egg cortisol is still unclear (Mommsen et al. 1999). Furthermore, beyond egg size and offspring survival data (Campbell et al. 1994; Stratholt et al. 1997), the effects of maternal exposure to a stressor on offspring performance traits represent an ecological black box (but see McGhee et al. 2012; Roche et al. 2012). Proxies for imposing maternal stress via exogenous cortisol injection (Eriksen et al. 2011) and immersion of eggs in cortisol at fertilization (Sloman 2010; Burton et al. 2011) are logistically less challenging than chronically exposing adults to stressors. Such experimental designs have yielded intriguing behavioural carry-over effects of maternal stress, though this and the aforementioned work remain limited to domesticated and/or iteroparous fish. Extension of those research findings to wild fish should be cautioned given the notable differences in behaviour and reproductive maturation between hatchery and wild fish (Johnsson 1997). Nevertheless, a foundation exists for exploring how adult exposure to stressors affects subsequent generations in wild populations of fish.

Semelparous populations of Pacific salmon (*Oncorhynchus* spp.) that migrate through and spawn in the Fraser River watershed in British Columbia (Canada) can be used to understand the latent consequences of maternal stress for offspring. After spending 1–3 years in the Pacific Ocean, adult salmon migrate to natal freshwater spawning grounds on a fixed energy budget due to cessation of feeding, in tandem with senescence and deterioration of major physiological processes (Groot and Margolis 1991). Returning salmon encounter a myriad of aquatic stressors: pollution, hydroelectric dams, warming water temperatures, predators, and the fishing nets/hooks of recreational, commercial, and indigenous fishers. The cumulative effects of endogenous stress and exogenous stressors are thought to be driving the population declines of sockeye salmon (*O. nerka*; Cohen 2012), but there has been minimal investigation into how intergenerational effects of maternal experience may be implicated in changes in population dynamics (but see Braun et al. 2013). With only a single breeding episode before death to produce the next generation of spawners, and brief parental care to mediate negative effects of maternally derived handicaps (e.g., elevated egg GC levels; Love and Williams 2008), how maternal stress translates to offspring phenotypes could considerably influence a female's lifetime fitness. Specifically, if offspring performance traits, such as burst swimming ability, are compromised, this could have detrimental effects at various stages of development. Fry migrating to nursery areas where they will feed and grow for 1–2 years (Groot and Margolis 1991) need adequate burst swim performance to successfully avoid predators (Taylor and McPhail 1985).

We sought to quantify the intergenerational effects of maternal stress in wild (i.e., lived from egg to adult in the wild and originate from parents produced in the wild) semelparous sockeye salmon. We predicted that mothers repeatedly exposed to an acute chase stressor would have smaller eggs with higher concentrations of cortisol. Changes in egg composition were predicted to reduce embryo survival and fry burst swimming performance.

Materials and methods

Adult exposure and offspring rearing

Wild adult sockeye salmon were collected in fall 2011 during their up-river migration along a main tributary of the Fraser River (British Columbia, Canada), the Harrison River (49°17′5N, 121°54′27W), approximately 6 weeks prior to historical peak spawning (mid-November). Fish were transported 60 km (~1 h) to the Department of Fisheries and Oceans (DFO) Cultus Lake Salmon Research Laboratory (Cultus Lake, British Columbia, Canada) and held in ten large, circular tanks (3 m diameter, 2 m deep, 10,000 L capacity). Water temperature in tanks decreased from 12.6 to 7.6 °C, reflective of changes in water temperature experienced in the Harrison River. Each tank held 26–30 fish (14–26 females and 4–12 males). Tanks were divided into two treatment groups: (1) control fish that were not disturbed throughout the duration of experimental holding (five tanks), and (2) stressed fish that were chased (five tanks), but not trapped, with a net for 3 min, two times a day to mimic the repetitive nature of encounters with physical stressors (e.g., fisheries net, predator) experienced by fish migrating in the wild. Similar methods whereby experimenters manually chased wild Pacific salmon for 3 min showed that heart rate, plasma lactate, and plasma cortisol levels are significantly increased (Donaldson 2012), indicating individuals mount a stress response when exposed to such a stressor. Fish were chased at randomized times between 0900 and 1700 hours to reduce habituation. Fish were terminally sampled after 37 ($n = 22$ females; $n = 22$ males) and 42 ($n = 13$ females; $n = 13$ males) days of chasing, as timing of ripeness was staggered in time. The proportion of ripe females was, however, equal among control (46 %) and stressed (45 %) tanks at the termination of study, and similar numbers of fish were removed and sampled from each tank. Sampled fish were sacrificed using cerebral concussion, and total mass and fork length were measured to the nearest 0.01 kg and 0.1 cm, respectively. Gametes were extracted and total ovary mass was recorded to the nearest g for females. Mass per egg was assessed by weighing (to the nearest 0.0001 g) three replicates of ten eggs from each female wet and after being dried in an oven for 48 h at 60 °C. Three unfertilized, wet eggs from all but one female (control) were also frozen in liquid nitrogen and transferred to −80 °C for cortisol analysis.

Gametes were fertilized at the University of British Columbia (UBC) following methods described in Whitney et al. (2013). Eggs from 18 control and 17 stressed females were fertilized with sperm from control males to create full sibling crosses. Control males were paired once with a control female and once with a stressed female (one male was paired only once, with a control female). Each full-sibling cross was replicated three times. Full-sibling crosses were used as the focus of the study was to determine differences at the level of maternal stressor treatment. Consequently, replication between but not within mothers does not exclude the influence of parental effects. Following fertilization, eggs still separated by family and replicate were incubated in Heath trays with mean (±SE) water temperature of 8.4 ± 0.6 °C. Developing embryos were monitored daily and dead eggs/embryos were removed from Heath trays and stored in Stockard's solution [5 % formaldehyde, 4 % glacial acetic acid, 6 % glycerin, 85 % water] to evaluate fertilization success. When fry reached emergence (complete absorption of the yolk sac) approximately 5 months from fertilization, up to five fish from each replicate of each family were sacrificed with a lethal dose of tricaine methanesulphonate (MS-222; Sigma; http://www.sigmaaldrich.com), blotted dry, and body mass (to the nearest 0.001 g) and fork length (to the nearest 0.1 cm) were measured for each fish. Replicates with <35 % survival were not sampled to ensure adequate numbers of fish for swim trials. Separated by maternal treatment (families pooled), emerged fry were then transferred to a single 1,000-L flow-through rectangular pond until swim trials. Water supply to the ponds came from local municipal reservoirs and flowed continually with minimal flows. Photoperiod was adjusted throughout rearing to mimic natural photoperiod at latitude 49°18′N. Water temperature in ponds ranged from 6 to 11 °C throughout rearing due to natural changes in municipal water. Fish were fed powdered fishmeal (EWOS Canada; http://www.ewos.com) ad libitum twice daily until 24 h before burst swimming trials. Fish were monitored daily and all mortalities were recorded. These methods have been used previously to raise Pacific salmon in the laboratory (Burt et al. 2012).

Egg cortisol assay

Cortisol concentrations were determined using enzyme immunoassay (EIA; Neogen, http://www.neogen.com). Briefly, three unfertilized eggs from each female used for fertilizations ($n = 34$, eggs were not obtained from one

control female) were homogenized in 1,200 μL of assay buffer. Diethyl ether (3 mL) was added to the homogenate. Samples were vortexed, centrifuged for 5 min at 10,000*g*, flash frozen at −80 °C, and the liquid phase poured off and evaporated under nitrogen. The residue was reconstituted in 1,200 μL of assay buffer and heated for 10 min at 65 °C. An aliquot of 250 μL was removed and frozen at −80 °C for later use on the EIA plate. Samples were run in duplicate on three assay plates with intra- and inter-assay coefficients of variation 4.5 and 4.9 %, respectively.

Fry burst swimming performance

At approximately 1 month post-emergence, control ($n = 100$) and stressed ($n = 100$) fry were swum in a fixed speed test to assess burst swim performance. Following Sopinka et al. (2013), without exposure to air, a single fish was randomly selected from its holding pond and dropped into a sectioned area (30 cm length × 6.9 cm width × 4 cm depth) of an open-top rectangular swim flume (230 cm length × 17 cm width × 4 cm depth). To maintain laminar flow of water entering the sectioned area, a plastic honeycomb was positioned at the front of the flume. The front 20 cm and back 10 cm of the sectioned area were covered and illuminated with lights, respectively, to encourage fish to swim forward in the flume. Water speed (cm s^{-1}) was calculated as water speed = discharge (L^3 s^{-1})/cross-sectional area (cm^2). Discharge was quantified by recording (in s) how long a 10-L receptacle took to fill completely. Water speed was kept constant at 25 cm s^{-1} (which equated to 9 L_T s^{-1}) throughout the duration of swim trials. Water temperature in the flume during swim trials was 8.0 °C. All swim trials were recorded at 60 frames per second using a digital camera (Canon EOS Rebel T3i; http://www.canon.com). A trial ended when a fish at the back of the sectioned area failed to initiate swimming after being probed three times with a blunt instrument. On average, fry swam for 62 s (range 8–302 s). Fish were then removed from the flume and sacrificed with a lethal dose of MS-222, blotted dry, and body (to the nearest 0.001 g) and fork length (to the nearest 0.1 cm) were measured.

Burst swimming duration and burst swimming rate were quantified from videos viewed in Quicktime Pro (http://www.apple.com). For fish that burst swam for one continuous bout, the length of that bout was defined as total burst swimming duration. For fish that reinitiated swimming after falling back out of the covered area, the length of all the bouts of burst swimming were summed to quantify total burst swimming duration. Burst swimming rate was calculated by summing the number of times (10 s^{-1}) fish re-entered the covered area at the front of the flume after falling back out of said covered area.

Data analyses

Statistical analyses were based on treatment level means. When data could not be transformed ($\log_{10}$, logit) to meet assumptions of normality or equal variance, non-parametric tests were employed. Body condition was calculated using the formula: (body mass/FL^{-3}) × 100 %. To assess differences between control and stressed mothers, *t* tests (female body condition, egg cortisol, offspring survival to 1-month post-emergence, offspring body condition at 1-month post-emergence), a general linear model (GLM) with maternal treatment (control or stressed) as a fixed effect and female body mass as a covariate [female ovary mass, egg mass (wet, dry)], and a GLM with maternal treatment as a fixed effect and family ID as a random effect (fertilization success, offspring survival to emergence, offspring body condition at emergence) were used. Pearson correlations were used to determine relationships between egg cortisol and fertilization success, offspring survival to emergence ,and emergent offspring body condition. A Chi-squared test was used to evaluate differences in the proportion of non-swimming fry due to maternal exposure to a stressor. Body mass (Pearson correlation, $r^2 = 0.21$, $n = 169$, $P < 0.0001$) and fork length ($r^2 = 0.13$, $n = 169$, $P < 0.0001$) positively correlated with burst swimming duration. Differences in swimming duration between offspring from control and stressed mothers were thus examined using a GLM, with fry body mass or length as a covariate and maternal treatment as a fixed effect. Maternal treatment differences in burst swimming rate were determined with a Wilcoxon signed-rank test. Non-significant interactions ($P > 0.05$) were removed from all models. Post hoc differences ($P < 0.05$) were determined using Tukey's HSD test.

Results

Maternal exposure to the repeated stressor did not affect female body condition, egg mass (dry and wet), egg cortisol, or fertilization success (Table 1). Chased mothers did, however, have reduced ovary mass (Table 1). Across multiple stages of development, no differences in survival or body condition were detected between offspring from control and stressed mothers (Table 1). Offspring survival to emergence was, however, more variable for stressed mothers (Levene's test, $F = 4.69$, $P = 0.04$; coefficient of variation, control mothers = 0.36, stressed mothers = 0.58). Across females (control and stressed), egg cortisol levels did not correlate with fertilization success (Pearson correlation, adjusted $r^2 = -0.03$, $n = 34$, $P = 0.70$) or offspring survival to emergence (adjusted $r^2 = -0.03$, $n = 34$, $P = 0.79$). There was a tendency for emergent offspring

Table 1 Effects of maternal exposure to the chase stressor on reproductive, gametic and offspring traits of Pacific sockeye salmon (*Oncorhynchus nerka*)

Trait	Maternal treatment	*n*	Mean ± SE (range)	*t*/*F*, *P* value
Body condition	Control	18	1.12 ± 0.02 (1.01–1.32)	$t = -0.20$ $P = 0.84$
	Stressed	17	1.11 ± 0.02 (0.98–1.31)	
Ovary mass (kg)	Control	18	0.554 ± 0.022 (0.387–0.719)	$F_{1,32} = 9.00$ $P = 0.01$
	Stressed	17	0.503 ± 0.021 (0.295–0.620)	
Wet egg mass (g)	Control	18	0.155 ± 0.004 (0.119–0.190)	$F_{1,32} = 0.05$ $P = 0.82$
	Stressed	17	0.154 ± 0.004 (0.130–0.194)	
Dry egg mass (g)	Control	18	0.063 ± 0.002 (0.050–0.072)	$F_{1,32} = 0.64$ $P = 0.43$
	Stressed	17	0.060 ± 0.007 (0.051–0.075)	
Egg cortisol (ng/g)	Control	17	11.59 ± 1.74 (3.21–28.26)	$t = 0.55$ $P = 0.58$
	Stressed	17	13.03 ± 1.99 (4.12–32.78)	
Fertilization success (%)	Control	18	82.30 ± 4.30 (28.44–98.62)	$F_{1,33} = 0.76$ $P = 0.39$
	Stressed	17	86.94 ± 3.04 (56.05–98.30)	
Survival to emergence (%)	Control	18	76.19 ± 6.53 (8.74–97.50)	$F_{1,33} = 0.58$ $P = 0.45$
	Stressed	17	67.52 ± 9.46 (1.60–98.73)	
Survival 1-month post-emergence (%)	Control	18	85.61 ± 4.65 (28.77–99.90)	$t = -0.75$ $P = 0.33$
	Stressed	17	92.53 ± 2.02 (69.05–99.90)	
Fry body condition (at emergence)	Control	15	8.14 ± 0.18 (6.96–9.64)	$F_{1,25} = 0.15$ $P = 0.71$
	Stressed	12	8.24 ± 0.18 (7.57–9.26)	
Fry body condition (1-month post-emergence)	Control	100	6.81 ± 0.07 (5.34–8.64)	$t = -0.53$ $P = 0.60$
	Stressed	100	6.87 ± 0.08 (5.20–10.13)	

body condition to decrease with increased egg cortisol (adjusted $r^2 = 0.14$, $n = 26$ $P = 0.06$).

Similar proportions of fry from control (13 %) and stressed (18 %) mothers failed to burst swim in the flume (Chi-squared test, $\chi^2 = 0.96$, $n = 200$, $P = 0.33$). Burst swimming performance was related to maternal treatment. Controlling for body mass (GLM, maternal treatment: $F_{1,166} = 32.53$, $P < 0.0001$; body mass: $F_{1,166} = 43.04$, $P < 0.0001$), and length (maternal treatment: $F_{1,166} = 24.88$, $P < 0.0001$; body length: $F_{1,166} = 17.34$, $P < 0.0001$), fry reared from stressed mothers burst swam for shorter periods of time compared to fry reared from control mothers (Fig. 1a). When burst swimming rate was quantified, the relationship between maternal treatment and swim performance reversed; fry reared from stressed mothers had higher burst swimming rates than fry reared from control mothers (Wilcoxon signed-rank test, $Z = 5.29$, $n = 169$, $P < 0.0001$; Fig. 1b). Following Sopinka et al. (2013), as burst swimming rate increased, total burst swimming duration decreased (Spearman's rank correlation, control: $\rho = -0.52$, $n = 87$, $P < 0.0001$; stressed: $\rho = -0.50$, $n = 82$, $P < 0.0001$).

Discussion

We have shown for the first time in a wild semelparous vertebrate that mothers repeatedly exposed to a chase stressor do not incur significant impairment to reproductive and early offspring parameters (gametic composition, embryo survival). However, burst swimming performance of emergent fry was influenced by maternal exposure to the stressor. Gametic/offspring buffering (Schreck et al. 2001) and maternal match/mismatch (reviewed in Breuner 2008) are discussed here. Furthermore, a lack of variation in egg cortisol between stressed and control mothers prompts

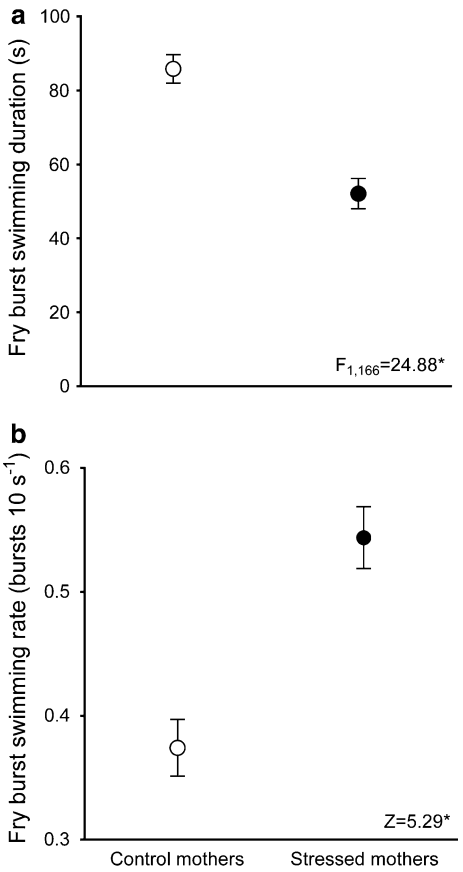

Fig. 1 **a** Mean (±SE) length-adjusted burst swimming duration (in s) of Pacific sockeye salmon (*Oncorhynchus nerka*) fry reared from control (*empty circles*, $n = 87$) and stressed (*filled circles*, $n = 82$) mothers. Untransformed data presented for illustrative purposes only. ANCOVA statistics presented at $^*P < 0.0001$ used $\log_{10}$-transformed data. **b** Mean burst swimming rate (±SE) for fry from control and stressed mothers. Burst swimming rate was calculated by summing the number of times (10 s^{-1}) fry re-entered the shaded area at the front of the flume after falling back out of the shaded area. Wilcoxon signed-rank test statistics are presented at $^*P < 0.0001$

discussion of alternative mechanisms driving the observed behavioural differences in offspring.

Egg size (Brooks et al. 1997) and cortisol (Mingist et al. 2007) can influence offspring survival and hence the capacity for mothers to effectively mediate these effects is advantageous. Such a system would be especially advantageous for sockeye salmon given that in the wild, typical egg–fry survival is already very low (7 %, Bradford 1995). In this study, a "progeny-protecting system" (Schreck et al. 2001) may account for the observed resilience to increases in egg cortisol and reductions to egg size and offspring survival in sockeye salmon chronically exposed to an acute stressor. Though overall ovary mass was reduced in stressed females, absence of reduced egg size and increased egg cortisol could be the result of maternal modulation of egg lipo-protein (e.g., vitellogenin) and hormone content. It is unclear, however, when this potential modulation would have occurred (onset of stressor treatment versus final stages of ovulation). In mammals, hormonal modulation of offspring is accomplished via the enzyme 11β-hydroxysteroid dehydrogenase (11β-HSD) which converts cortisol to its inactive form cortisone (Benediktsson et al. 1997). Rainbow trout (*Oncorhynchus mykiss*) ovarian follicles, ovulated eggs, and fertilized embryos show evidence of converting cortisol to cortisone, which suggests the presence of similar protective enzymes in fishes (Li et al. 2012). We speculate that, in semelparous, senescing animals, 11β-HSD levels may increase in tandem with GCs due to degeneration of tissues that regulate the hypothalamic–pituitary–interrenal (HPI) axis (Maldonado et al. 2002). In combination with stress response attenuation as maturation progresses (Wingfield and Sapolsky 2003; Cook et al. 2011), it is possible that semelparous mothers possess superior mechanisms to reduce offspring exposure to elevated levels of GCs.

Maternal match/mismatch is receiving increasing attention (Breuner 2008; Love and Williams 2008; Love et al. 2013; Sheriff and Love 2013). We hypothesize that chased mothers perceived the daily chase stressor as a chronic, unpredictable, large surface-attacking predator (i.e., bear) and/or enclosing seine/gill fishing net (both tangible threats to migrating Pacific salmon that can elicit a burst swimming response). The riverine environment shared by mothers and later by their offspring would be characterized as unpredictable and having a high predation risk. Accordingly, mothers produced offspring with phenotypes (increased burst swimming rates) that could be beneficial if fry also encountered a world with chronic, unpredictable stressors that required individuals to repeatedly swim away to ensure survival; consistent with maternal match predictions. Although fry may not encounter the same types of stressors as migrating adults, higher burst swimming rates can still be advantageous when escaping life-stage appropriate predators (e.g., great blue heron *Ardea herodias*; larger/older *Oncorhynchus* spp.). Furthermore, swim capacity traits manifesting in young fry can correlate with adult swim requirements in sockeye salmon (Sopinka et al. 2013). Mothers that complete a longer and more arduous migration produce offspring with higher burst swimming rates, suggesting that mothers may prime offspring with swimming abilities detectable early in development that also attribute to successful completion of a more challenging migration later in life (Sopinka et al. 2013). Alternatively, from a mismatch perspective, increased burst swimming rates may be a hyperactive response and exhaust energy stores more rapidly, and reduced burst swimming durations result in faster and/or inevitable capture by a predator/fishing net. Energetically and behaviourally inefficient predator escape would lead to the conclusion that

maternal stress conferred unfavorable traits to offspring. Interestingly, locomotory differences occurring in the absence of differences in egg cortisol clearly indicate that intergenerational effects can become manifest via mechanisms other than cortisol.

Hormone- and epigenetic-mediated maternal effects have received considerable attention across taxa. In fish, cortisol is the predominant hormone manipulated by researchers to mimic maternal stress and evoke differences in offspring performance. One of the primary predictions made was that repeated exposure to an acute stressor would increase maternal deposition of cortisol into eggs. Comparable egg cortisol levels between stressed and control mothers did not support this prediction nor previous findings with hatchery coho salmon (*O. kisutch*.; Stratholt et al. 1997). Other egg hormones (sex and thyroid hormones) not measured may have varied and contributed to differences detected later in offspring development. Other egg components that could elicit latent behavioural effects are those related to the genetic contribution of mothers to their offspring. In non-migrating fish (that do not move through as many diverse environments throughout life), genetic influences may be under greater selection pressure and maternal-matching is apt to be a more reliable strategy to confer adaptive information to offspring, as the environment of mother and offspring is considerably more similar. In migrating sockeye salmon, a mother's genetic makeup affects fry burst swim performance (Burt et al. 2012; Sopinka et al. 2013) and cellular machinery supporting burst swimming varies at the family and population level (Patterson et al. 2004). It is possible that changes to gene expression associated with maternal stress (e.g., Jeffries et al. 2012) could interact with inherent maternal effects, consequently altering the phenotype of offspring that would emerge under undisturbed conditions. Exposure to a temperature stressor during embryonic development modifies parental effects on emergent sockeye salmon burst swimming performance (Burt et al. 2012). Due to logistical constraints, we were unable to comprehensively control for parental effects within treatment groups. If the mechanisms driving stress-induced epigenetic changes are heritable, stressed mothers could reprogram offspring to express traits necessary to survive a similarly stressful environment (i.e., context-specific maternal effect; Badyaev and Uller 2009). Further research quantifying the heritability of performance traits between ecologically stressed and undisturbed Pacific salmon will help to quantify and qualify the impact of maternal effects and enrich current population modelling.

This study stresses the importance of examining different metrics of offspring performance at different life stages. Becoming more evident is that predicting the latent effects of ecological stressors in wild populations is not as straightforward as previously thought (Dickens and Romero 2013). Free-living animals are now exposed to cumulative stressors across all stages of life, and stress-coping mechanisms vary with species-specific reproductive strategies. Approaching maternal effects in a holistic manner that encompasses the ecology, endocrinology, and behaviour of a species throughout its life history will provide a more complete picture of how the environment affects more than one generation at a time (Sheriff and Love 2013).

Acknowledgments All research conformed to protocols approved by the University of British Columbia Committee on Animal Care (#A11 0215) and met the Canadian Council for Animal Care guidelines. We thank members of UBC's Pacific Salmon Ecology and Conservation Laboratory, Chehalis First Nation, DFO Cultus Lake Salmon Research Laboratory, DFO Environmental Watch, and undergraduate volunteers for fish collection and offspring rearing, and G. Raby, two anonymous reviewers and Marc Mangel for helpful manuscript comments. S.G.H. is funded by a Natural Sciences and Engineering Research Council (NSERC) Discovery and Strategic grant, and the Ocean Tracking Network. N.M.S. was funded by an NSERC graduate scholarship, and C.T.M. was funded by an NSERC undergraduate student research award (USRA).

References

Badyaev AV, Uller T (2009) Parental effects in ecology and evolution: mechanisms, processes and implications. Philos Trans R Soc Lond B 364:1169–1177

Benediktsson R, Calder AA, Edwards CRW, Seckl JR (1997) Placental 11β-hydroxysteroid dehydrogenase: a key regulator of fetal glucocorticoid exposure. Clin Endocrinol 46:161–166

Bradford MJ (1995) Comparative review of Pacific salmon survival rates. Can J Fish Aquat Sci 52:1327–1338

Braun DC, Patterson DA, Reynolds JD (2013) Maternal and environmental influences on egg size and juvenile life-history traits in Pacific salmon. Ecol Evol 3:1727–1740

Breuner C (2008) Maternal stress, glucocorticoids, and the maternal/fetal match hypothesis. Horm Behav 54:485–487

Brooks S, Tyler CR, Sumpter JP (1997) Egg quality in fish: what makes a good egg? Rev Fish Biol Fish 7:387–416

Burt JM, Hinch SG, Patterson DA (2012) Developmental temperature stress and parental identity shape offspring burst swimming performance in sockeye salmon (*Oncorhynchus nerka*). Ecol Freshw Fish 21:176–188

Burton T, Hoogenboom MO, Armstrong JD, Groothuis TGG, Metcalfe NB (2011) Egg hormones in a highly fecund vertebrate: do they influence offspring social structure in competitive conditions? Funct Ecol 25:1379–1388

Campbell PM, Pottinger TG, Sumpter JP (1994) Preliminary evidence that chronic confinement stress reduces the quality of gametes produced by brown and rainbow trout. Aquaculture 120:151–169

Cohen BI (2012) Commission of Inquiry into the Decline of Sockeye Salmon in the Fraser River. Public Works and Government Services Canada, Ottawa. Cat. No.: CP32-93/2012E−1 (v. 1)

Cook KV, McConnachie SH, Gilmour KM, Hinch SG, Cooke SJ (2011) Fitness and behavioral correlates of pre-stress and stress-induced plasma cortisol titers in pink salmon (*Oncorhynchus gorbuscha*) upon arrival at spawning grounds. Horm Behav 60:489–497

Coslovsky M, Richner H (2011) Predation risk affects offspring growth via maternal effects. Funct Ecol 25:878–888

Dickens MJ, Romero LM (2013) A consensus endocrine profile for chronically stressed wild animals does not exist. Gen Comp Endocrinol 191:177–189

Donaldson MR (2012) Understanding the consequences of fisheries-related stressors on adult migrating Pacific salmon. PhD dissertation, University of British Columbia, Vancouver

Eriksen MS, Færevik G, Kittilsen S, McCormick MI, Damsgård B, Braithwaite VA, Braastad BO, Bakken M (2011) Stressed mothers-trouble offspring: a study of behavioural maternal effects in farmed *Salmo salar*. J Fish Biol 79:575–586

Groot C, Margolis L (1991) Pacific salmon life histories. University of British Columbia Press, Vancouver

Heath DD, Blouw DM (1998) Are maternal effects in fish adaptive or merely physiological side effects? In: Mousseau TA, Fox CW (eds) Maternal effects as adaptations. Oxford University Press, New York, pp 178–201

Henriksen R, Rettenbacher S, Groothuis TGG (2011) Prenatal stress in birds: pathways, effects, function and perspectives. Neurosci Biobehav Rev 35:1484–1501

Jeffries KM, Hinch SG, Sierocinski T, Clark TD, Eliason EJ, Donaldson MR, Li S, Pavlidis P, Miller KM (2012) Consequences of high temperatures and premature mortality on the transcriptome and blood physiology of wild adult sockeye salmon (*Oncorhynchus nerka*). Ecol Evol 2:1747–1764

Johnsson B (1997) A review of ecological and behavioural interactions between cultured and wild Atlantic salmon. ICES J Mar Sci 54:1031–1039

Li M, Christie HL, Leatherland JF (2012) The in vitro metabolism of cortisol by ovarian follicles of rainbow trout (*Oncorhynchus mykiss*): comparisons with ovulated oocytes and pre-hatch embryos. Reproduction 144:713–722

Love OP, Williams TD (2008) The adaptive value of stress-induced phenotypes in the wild: effects of maternally-derived corticosterone on sex allocation, cost of reproduction and maternal fitness. Am Nat 172:E135–E149

Love OP, McGowan OP, Sheriff MJ (2013) Maternal adversity and ecological stressors in natural populations: the role of stress axis programming in individuals, with implications for populations and communities. Funct Ecol 27:81–92

Maldonado TA, Jones RE, Norris DO (2002) Timing of neurodegeneration of beta-amyloid (AB) peptide deposition in the brain of aging kokanee salmon. Dev Neurobiol 53:21–35

McConnachie SH, Cook KV, Patterson DA, Gilmour KM, Hinch SG, Farrell AP, Cooke SJ (2012) Consequences of acute stress and cortisol manipulation on the physiology, behavior, and reproductive outcome of female Pacific salmon on spawning grounds. Horm Behav 62:67–76

McGhee KE, Pintor LM, Suhr EL, Bell AM (2012) Maternal exposure to predation risk decreases offspring antipredator behaviour and survival in threespined stickleback. Funct Ecol 26:932–940

Mingist M, Kitani T, Koide N, Ueda H (2007) Relationship between eyed-egg percentage and levels of cortisol and thyroid hormone in masu salmon *Oncorhynchus masou*. J Fish Biol 70:1045–1056

Mommsen TP, Vijayan MM, Moon TW (1999) Cortisol in teleosts: dynamics, mechanisms of action, and metabolic regulation. Rev Fish Biol Fish 9:211–268

Mousseau TA, Fox CW (1998) The adaptive significance of maternal effects. Trends Ecol Evol 13:403–407

Patterson DA, Guderley H, Bouchard P, Macdonald JS, Farrell AP (2004) Maternal influence and population differences inactivities of mitochondrial and glycolytic enzymes in emergent sockeye salmon (*Oncorhynchus nerka*) fry. Can J Fish Aquat Sci 61:1225–1234

Räsänen K, Kruuk LEB (2007) Maternal effects and evolution at ecological time-scales. Funct Ecol 21:408–421

Roche DP, McGhee KE, Bell AM (2012) Maternal predator-exposure has lifelong consequences for offspring learning in threespined sticklebacks. Biol Lett 8:932–935

Romero LM (2004) Physiological stress in ecology: lessons from biomedical research. Trends Ecol Evol 19:249–255

Schreck CB, Contreras-Sanchez W, Fitzpatrick MS (2001) Effects of stress on fish reproduction, gamete quality, and progeny. Aquaculture 197:3–24

Sheriff MJ, Love OP (2013) Determining the adaptive potential of maternal stress. Ecol Lett 16:271–280

Sloman KA (2010) Exposure of ova to cortisol pre-fertilisation affects subsequent behaviour and physiology of brown trout. Horm Behav 58:433–439

Sopinka NM, Hinch SG, Lotto AG, Whitney CK, Patterson DA (2013) Does among-population variation in burst swim performance of sockeye salmon *Oncorhynchus nerka* fry reflect early life migrations? J Fish Biol 83:1416–1424

Stratholt ML, Donaldson EM, Liley NR (1997) Stress induced elevation of plasma cortisol in adult female coho salmon (*Oncorhynchus kisutch*), is reflected in egg cortisol contest, but does not appear to affect early development. Aquaculture 158:141–153

Taylor EB, McPhail JD (1985) Burst swimming and size-related predation of newly emerged coho salmon *Oncorhynchus kisutch*. Trans Am Fish Soc 114:546–551

Whitney CK, Hinch SG, Patterson DA (2013) Provenance matters: thermal reaction norms for embryo survival among sockeye salmon *Oncorhynchus nerka* populations. J Fish Biol 82:1159–1176

Wingfield JC, Sapolsky RM (2003) Reproduction and resistance to stress: when and how. J Neuroendocrinol 15:711–724