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Synergistic effects of parental and embryonic exposure to predation risk on prey offspring size at emergence

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Abstract. Cues signaling predation risk can strongly influence prey phenotypes both within and between generations. Parental and embryonic effects have been shown to operate independently in response to predation risk, but how they interact to shape offspring life history traits remains largely unknown. Here, we conducted experiments to examine the synergistic impacts of parental and embryonic experiences with predation risk on offspring size at emergence in the snail, *Nucella lapillus,* which is an ecologically important intermediate consumer on rocky intertidal shores. We found that when embryos were exposed to predation risk, the offspring of risk-experienced parents emerged larger than those of parents that had no risk experience. This response was not the result of increased development time, greater resource availability, or fewer emerging offspring, but may have occurred because both parental and embryonic experiences with risk increased growth efficiency, perhaps by reducing embryonic respiration rates under risk. Our results highlight the potential for organisms to be influenced by a complex history of environmental signals with important consequences for individual fitness and predator–prey interactions.

Key words: Carcinus maenas; *developmental effects;* Nucella lapillus; *parental effects; physiology; transgenerational effects.*

Introduction

Prey often respond to the fear of being eaten (i.e., predation risk) by changing morphological and behavioral traits (Lima and Dill 1990), which can strongly influence individual performance and fitness across all stages of life history (Werner and Gilliam 1984, Ludwig and Rowe 1990). For example, the use of refuge habitats in response to predation risk (Sih 1980, Mangel and Clark 1986) can limit foraging opportunities that curtail prey growth (Werner et al. 1983, Donelan et al. 2017) and reproduction (Fraser and Gilliam 1992). Prey are also sensitive to predation risk as embryos, as evidenced by induced hatching to evade egg predation (Warkentin 1995, Chivers et al. 2001) or prolonged development to reduce the risk of predation after emergence (Sih and Moore 1993, Stoks et al. 2006), which can impact offspring size, developmental state, and survival at emergence (Petranka et al. 1987, Warkentin 1995). In addition, embryonic plasticity can influence prey throughout ontogeny and into adulthood (Peckarsky et al. 2001, Vonesh and Bolker 2005). Hence, while antipredator strategies available to prey during embryonic stages may be more limited compared to those at other stages of ontogeny, they can nevertheless have cascading impacts on prey fitness that may ultimately affect the dynamics of prey populations and natural communities.

Importantly, parental experience with predation risk may also exert a strong influence on the responses of their offspring to risk. Offspring of risk-experienced parents can exhibit enhanced growth (Donelan and Trussell 2015, Beaty et al. 2016) and altered survival rates (Storm and Lima 2010, McGhee et al. 2012) in the presence of risk compared to offspring of risk-naïve parents, while also producing induced defenses (Agrawal et al. 1999, Beaty et al. 2016) or modifying their behavior or foraging effort (Shine and Downes 1999, Donelan and Trussell 2015). In addition, parental experience with predation risk can affect the response of offspring to risk both early (Coslovsky and Richner 2011) and late in life (McGhee et al. 2012, Donelan and Trussell 2015), and have important consequences for the demography of prey populations (Sheriff et al. 2010). While there is great potential for risk effects to operate transgenerationally, our understanding of the prevalence of such parental effects remains limited.

In many systems, parental and embryonic risk experiences may act synergistically to impact offspring traits, particularly when developing embryos have limited dispersal. In such cases, offspring may rely upon signals that they receive from their parents about their potential risk environment as well as those they experience while developing as embryos. Indeed, the degree to which these environmental cues are complementary may influence the likelihood of parental effects expression (Mousseau and Fox 1998, Uller 2008, Burgess and Marshall 2014). There

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is also evidence that parental and embryonic effects can independently impact the same offspring traits (e.g., size at emergence [Warkentin 1995, Bestion et al. 2014], time to maturity [Tollrian 1995, Orizaola and Braña 2005]). Hence, while both parental and embryonic experiences can independently shape the response of prey offspring to predation risk, their combined effects on offspring performance are poorly understood. Indeed, to our knowledge, their interaction remains unexplored.

The temporal proximity of parental and embryonic exposure to risk may enhance their interactive effects on early life history traits of prey. Offspring size at emergence is often an important life history trait because of its influence on foraging and scope for growth (Sinervo 1990, Stearns 1992), which can dictate future offspring fitness (Metcalfe and Monaghan 2001). Larger offspring often (though not always; Kaplan 1992) have higher survivorship than smaller offspring (Moran and Emlet 2001, Marshall et al. 2003), particularly in stressful environments (Reznick and Yang 1993, Warkentin 1995). As adults, prey grow less in the presence of risk because shifts in behavior (i.e., increased refuge use) limit foraging opportunities (Kats and Dill 1998, Lima 1998) and because heightened metabolic demands associated with risk-induced physiological stress can reduce growth efficiency (Hawlena and Schmitz 2010). For example, exposure to risk can increase prey respiration rates (Slos and Stoks 2008, Hawlena and Schmitz 2010), thereby limiting growth via decreased assimilation efficiency (McPeek et al. 2001, Trussell et al. 2006). Embryonic experience with predation risk may elicit similar physiological effects in offspring and further limit their size at emergence. However, because many prey also produce morphological defenses in response to risk (Harvell 1990), embryos may respond similarly to increase their overall size (e.g., via larger shells [Trussell et al. 2003], taller helmets [Tollrian 1995]), thereby reducing their vulnerability to predators. These morphological changes may also have cascading effects on prey performance through changes in individual metabolism (Czarnołęski et al. 2008).

Parental experience with risk may operate via similar mechanisms to either reinforce or weaken embryonic effects. Given the well-established trade-off between offspring size and number (Smith and Fretwell 1974, Stearns 1992), parents exposed to predation risk may produce larger offspring, which should be favored in stressful, risky environments (see above), at the cost of producing fewer offspring. Alternatively, parental experience with risk may shape embryo physiology (Giesing et al. 2010) or the production of morphological defenses (Agrawal et al. 1999) to allow their offspring to achieve a larger size as embryos. Parents may also, however, allocate fewer overall resources to reproduction in the presence of risk (Bernardo 1996), thereby limiting their offspring's capacity for embryonic growth. Because parental and embryonic effects can have complex and independent effects, it is difficult to predict how their interaction may shape offspring traits at emergence.

The effects of predation risk from the predatory green crab, *Carcinus maenas*, operate strongly both within and between generations of the carnivorous snail *Nucella lapillus,* an important consumer on rocky intertidal shores. Exposure to *Carcinus* risk cues negatively affects *Nucella* foraging, growth, and growth efficiency (Trussell et al. 2006, Matassa et al. 2016), but parental experience with *Carcinus* can weaken these effects in *Nucella* offspring such that they forage and grow more in the presence of risk than offspring of risk-naïve parents (Donelan and Trussell 2015). Female *Nucella* lay egg capsules containing multiple offspring that emerge as crawl-away, juvenile snails. Thus, this system is well suited to explore the synergistic effects of parental and embryonic exposure to predation risk because offspring develop in spatial proximity to their parents and are therefore likely to face similar risk conditions.

We conducted a series of laboratory experiments to examine whether parental experience with risk, embryonic experience with risk, and duration of embryonic experience interact to influence the size of *Nucella* offspring at emergence. We also explored the potential mechanisms that may drive differences in offspring size. We found that offspring of risk-experienced parents emerged larger than offspring of risk-naïve parents, but only if they experienced risk as embryos. We suggest that these patterns occurred because of changes in embryonic respiration rate; under predation risk, the offspring of risk-experienced parents had lower embryonic respiration rates, which may promote larger size via increased energetic efficiency. Our results provide new insight into the interactive effects of parental and embryonic experiences with predation risk, enhance our understanding of how predators drive prey performance in natural systems, and suggest how parental and embryonic effects may operate in response to other environmental cues.

Materials and Methods

Parental and embryonic exposure to risk

We conducted several fully factorial laboratory experiments to examine the effects of (1) parental experience with predation risk (presence, absence), (2) embryonic experience with predation risk (presence, absence), and (3) duration of embryonic experience (short, long) on offspring size at emergence and its potential underlying mechanisms using the carnivorous snail *Nucella lapillus* (hereafter *Nucella*) as the prey and the green crab *Carcinus maenas* (hereafter *Carcinus*) as the predator. Experiments were performed during the spring and summer of 2015 and 2016 at the Marine Science Center in Nahant, Massachusetts, USA. In both years, adult *Nucella* (>20 mm shell length; Etter 1989) were collected before the onset of mating from an exposed shore in Nahant in early February. *Nucella* were returned to the lab, sexed, and held in separate containers by sex until the experiments began. Because females can store sperm for up to three months

(Crothers 1985), sexes were kept separately before the summer to ensure that females did not use stored sperm to produce egg capsules during the experiments.

Experiments began in mid-May (in 2015) and early June (in 2016), using male and female adult *Nucella* paired at random to create 50 and 30 parental pairs, respectively. Our experimental design required that the risk environment of the developing embryos be separate from the risk environment of their parents. Hence, it was necessary to prevent female *Nucella* from depositing egg capsules while exposed to green crab risk cues. We therefore manipulated parental experience with predation risk by placing parent *Nucella* in a week-long cycle consisting of two stages: (1) three days in the presence/absence of risk, where the male and female in a given pair were individually housed in small containers that, along with a risk manipulation chamber, were submerged in a larger bucket followed by (2) four days in mating chambers in the absence of risk, where the male and female were placed together in the same container for mating and egg capsule deposition.

To manipulate parental experience with risk, we placed one male and one female adult *Nucella* separately in adjacent perforated containers (8 × 10 cm, diameter × height), each with a food supply of six blue mussels (*Mytilus edulis*, shell length 13.8 ± 1.4 mm [mean ± SD]), that sat downstream of another perforated, "risk" chamber (11.5 × 10 cm). These three containers (male + female + risk chambers) were placed together in a larger plastic bucket (24 × 24 cm, diameter × height) that received an independent supply of flowing seawater. We manipulated predation risk by placing one adult male green crab (carapace width 73.7 ± 2.2 mm [mean ± SD]) into a risk chamber with two *Nucella* for food (presence of risk) or placing two *Nucella* alone (absence of risk). One-half of the parent pairs were exposed to the presence of risk and one-half were exposed to the absence of risk. While the food snails provided to crabs may also release cues that induce responses in the experimental parent snails, it is important to note that *Nucella* react strongly to crab risk cues alone (e.g., Trussell et al. 2006).

During the mating stage, we transferred the male and female from a given pair from their individual risk treatment containers and placed them together in a perforated mating chamber (11.5 × 10 cm, diameter × height) that was submerged in a larger plastic bucket (24 × 24 cm) that received an independent supply of flowing seawater. All mating events took place in the absence of risk. After four days in the mating chamber, the male and female were returned to their respective, individual containers in their original risk-manipulation bucket and resupplied with six blue mussels. We repeated this cycle each week for 12 and 5 weeks in 2015 and 2016, respectively.

After each mating period was complete (i.e., once per week), we inspected mating chambers for the presence of new egg capsules. Newly laid clutches were removed, divided approximately in half, and placed into two separate, mesh-lined tea infusers (Upton Tea Imports, Holliston, Massachusetts, USA). To manipulate embryonic experience with risk, we placed one tea infuser from each clutch in the presence of risk and one in the absence of risk. Individual tea infusers were placed into a plastic bucket (17 × 14 cm) that also contained a downstream, perforated "risk" chamber (10 × 10 × 7 cm, length × width × height) and received its own supply of seawater. Predation risk was manipulated as described above for parents. All egg capsules remained in the presence and absence of risk for one week. After one week, we manipulated the duration of embryonic experience by removing one-half of the egg capsules from *each* tea infuser (those in both the presence and absence of embryonic risk) and placing them into a new tea infuser that was individually submerged in a plastic jar (8 × 10 cm) that received risk-free flowing seawater. Egg capsules that were moved to risk-free conditions after one week were in the "short" duration of embryonic experience treatment level. The other egg capsules remained in their original risk conditions for five more weeks (six weeks in total) and thus represent the "long" duration of embryonic experience treatment level. After six weeks, we moved egg capsules in the long duration of embryonic experience treatment level to new tea infusers and placed them in risk-free plastic jars as described above. This experiment is fully factorial with eight total treatment combinations (2 × 2 × 2) because both levels of the duration of embryonic experience treatment (short/long) were manipulated for both levels of the embryonic experience with risk treatment (presence/absence). Egg capsules in the absence of embryonic risk were never exposed to risk, but were moved into new tea infusers at the same time as those in the presence of embryonic risk to control for procedural effects.

Beginning six weeks after egg capsule deposition, we checked all tea infusers every two to three days for the emergence of new *Nucella* offspring. We measured each newly emerged offspring for maximum shell length (mm) using an AZ100 Nikon stereomicroscope and NIS Elements Basic Research microscope imaging software (v. 4.30, Nikon Corporation, Tokyo, Japan). In 2016, we also measured offspring maximum shell width (mm) and, for a subset of egg capsules, maximum egg capsule length (mm) and width (mm). To determine the influence of emergence time and number on offspring size, we also monitored the number of days that elapsed between egg capsule deposition and offspring emergence and counted the total number of offspring that emerged from each egg capsule in tea infusers that contained a single egg capsule (n = 107 in 2015, n = 75 in 2016). In total, we measured 1,462 offspring from 167 egg capsules laid by 21 parental pairs for shell length at emergence in 2015 and 872 offspring from 73 egg capsules laid by 24 parental pairs for shell length and width at emergence in 2016. The same proportion of parental pairs produced egg capsules in the presence and absence of risk in both experimental years (2015, P = 0.77; 2016, P = 0.36). Offspring from 12–17 replicates per treatment combination were used in the analyses of shell length, number emerging, and number of days until emergence.

Experiments to explore the mechanistic basis of offspring size patterns

We examined other potential factors that may impact offspring size at emergence in 2016: (1) parental provisioning to embryos, (2) allocation of energy to induced morphological defense (shell mass) by embryos, and (3) embryonic respiration rates in response to predation risk. Differential parental provisioning frequently affects offspring size in other systems (Bernardo 1996), and may operate in this system because predation risk effects on *Nucella* foraging and tissue growth (Trussell et al. 2006) may result in less energy available for reproduction. *Nucella* egg capsules each contain 5–30 developing embryos along with hundreds of nurse eggs that nourish embryos during development (Pechenik et al. 1984). Because embryos consume nurse eggs within the first 10 days of development (Costello and Henley 1971), we dissected a subset of egg capsules and counted their contents one week after egg capsule deposition using the microscopy protocol described above. Because of their developmental stage, we could not differentiate between embryos and nurse eggs (Crothers 1985, Rawlings 1990). Thus, following Spight (1976), we determined the approximate number of nurse eggs available to each embryo by counting the total number of particles (embryos + nurse eggs) in each egg capsule, subtracting the average number of offspring to ultimately emerge from an egg capsule produced by the same parental pair in the same week and exposed to the same conditions as the dissected egg capsule, and dividing by this average.

Nucella produce thicker shells in response to shell-crushing predators such as *Carcinus* to reduce their vulnerability to predation (i.e., induced defense; Trussell et al. 2003). The small size of emerging *Nucella* offspring (~1 mm shell length) prevented us from measuring shell and tissue masses separately using nondestructive techniques (e.g., Trussell et al. 2003). We therefore sacrificed a subset (3 individuals/capsule) of newly emerged offspring to estimate their tissue and shell masses (via organic and inorganic content, respectively) using ash-free dry mass. While shell material has a small fraction of organic content (1.5% in other *Nucella* species; Palmer 1983), the allocation of energy to organic mass should largely reflect investment in the soft tissue mass of offspring. Following Moran and Emlet (2001), we measured offspring immediately after emergence and then froze them individually in micropipette tubes and stored them at −20°C. We later thawed the offspring, rinsed them five times using deionized water, and dried them in a drying oven (60°C) for five days. After drying, we weighed each offspring on a microbalance (Mettler Toledo MX5; Columbus, Ohio, USA) in an individual pre-ashed and pre-weighed aluminum microweigh dish (VWR, Radnor, Pennsylvania, USA). We then ashed offspring in their weigh dish in a muffle furnace for 4 h at 450°C and weighed them again after they cooled to room temperature. We calculated tissue mass (organic content, mg) by subtracting the mass remaining after ashing (inorganic content, mg) from initial dry mass (mg). There were eight replicates, each from a different parental pair, of each treatment combination.

Finally, we examined whether parental and embryonic experiences with predation risk impacted the respiration rates of encapsulated *Nucella* embryos in the presence and absence of current predation risk. Exposure to predation risk can increase prey respiration rates (Hawlena and Schmitz 2010) that, in turn, lead to reduced growth efficiency (Trussell et al. 2006) and smaller body size. We used a FireStingO_2 meter (Pyro Science, Aachen, Germany) to measure oxygen consumption rates ($\mu mol\ O_2 \cdot h^{-1} \cdot mg^{-1}$) in the presence and absence of current risk of egg capsules (six weeks old) from each of the eight parental experience with risk × embryonic experience with risk × duration of embryonic experience treatment combinations. The FireSting system utilized 4-mL glass respiration vials (containing stainless steel beads to adjust the internal volume to 2.16 mL) that were affixed with oxygen sensor spots, which allow contactless detection (via an optical oxygen sensor) of oxygen concentrations within the vial. Due to logistical constraints, we conducted the experiment over four days with 21 separate trial runs. There were four replicates of each treatment combination, which were spread as evenly as possible across each trial. One treatment combination only had three replicates due to egg capsule availability (+Parental Risk/−Embryonic Risk/Short Duration of Embryonic Experience/+Current Risk.).

At the start of each trial, we placed one egg capsule in a respiration vial. We then submerged and capped the vials while in UV filtered seawater that had been conditioned for 24 h (in a water bath to maintain temperature at 14°C) either with or without one adult male green crab (presence and absence of current risk, respectively) and filtered again (to 0.7 μm) at the start of each experiment day; control vials were filled with "no risk" water. We then placed the four sealed vials (three experimental + one control) in a circulating water bath (14°C). Trials began once vials were submerged and settled, and ran for two hours, after which time oxygen levels had plateaued. Oxygen concentrations were recorded using Pyro Oxygen Logger software. After each trial, we measured and weighed each egg capsule and then dissected them to count the developing embryos, which did not differ across treatment combinations (all $P > 0.3$). We calculated egg capsule oxygen consumption rates ($\mu mol\ O_2 \cdot h^{-1} \cdot mg^{-1}$) by subtracting the absolute value of the change in oxygen concentration (initial minus final concentration, μmol O_2) in a control vial in a given trial run from the absolute value of the change in oxygen concentration within each experimental vial from the same trial run, multiplying by the volume of water in each vial corrected for egg capsule volume (2.16 mL minus egg capsule volume, mL), and dividing by the trial duration (2 h). We also explored the possibility that egg capsule respiration rates scaled allometrically with egg capsule mass (Schmidt-Nielsen 1984, Glazier 2005) using $\log_{10}$-$\log_{10}$ plots where the slope of the resulting line of best fit is equivalent to the scaling exponent, b (White and Kearney 2014).

Statistical analyses

We analyzed average *Nucella* offspring shell length at emergence, time to emergence, and number emerging per egg capsule using mixed-model ANOVAs that considered parental experience with risk, embryonic experience with risk, and duration of embryonic experience as fixed effects, and parental pair and experiment year as random effects. We used a similar model to analyze average *Nucella* offspring shell width, shell mass, and tissue mass at emergence, but year was not included as a factor because we only collected these data in 2016. Because two of the treatment combinations (absence of embryonic risk for the short and long duration) potentially inflated Type I error in model terms that included the duration of embryonic experience treatment, we conducted a secondary mixed model ANOVA that combined the embryonic experience with risk and duration of embryonic experience treatments into one term with four levels (see Appendix S2 in Supplementary Materials). The results between the two- and three-factor analyses were similar, and we therefore present the results of the three-factor analysis here for ease of interpretation.

We analyzed the per capita number of nurse eggs using a mixed-model ANOVA with parental experience with risk and embryonic experience with risk as fixed effects and parental pair as a random effect, but duration of embryonic experience was not included as a factor because nurse eggs were counted one week after egg capsule deposition, before we were able to apply this treatment. Egg capsule length and width were analyzed with parental experience with risk as a fixed effect, and parental pair as a random effect. Finally, egg capsule respiration rate was analyzed using parental experience with risk, embryonic experience with risk, duration of embryonic experience, and current exposure to risk as fixed effects, and trial run as a random block effect. To explore the significance of the random effects, we compared models using likelihood ratio tests (Zuur et al. 2009; see Appendix S2).

We also conducted linear regressions on $\log_{10}$ respiration rates (y) as a function of $\log_{10}$ egg capsule mass (x) to explore the possibility of allometric scaling of respiration rates. We found that egg capsule mass did not affect egg capsule respiration rates when analyzed across all treatment combinations (see *Results*; Fig. 3). When analyzed by treatment combination, egg capsule respiration rates did not scale with egg capsule mass for 15 of the 16 treatment combinations (see Appendix S1). Because respiration rates generally did not change allometrically across egg capsule mass, we divided respiration rates by egg capsule mass (mg) to further standardize for egg capsule size. We calculated egg capsule volume using the equation for a prolate ellipsoid ($v = \frac{4}{3}\pi(L/2)(W/2)^2$; Rawlings 1990). We conducted the mixed model analyses using Type III sums of squares in JMP 11 (SAS Institute, Cary, North Carolina, USA) and the likelihood ratio tests in R (v. 3.4.0; R Core Team 2017) using the nlme package (Pinheiro et al. 2017) and methods outlined in Zuur et al. (2009).

Results

Offspring of risk-experienced parents emerged with 8% longer shells (parental experience with risk × embryonic experience with risk, $F_{1,84.2} = 8.30$, $P = 0.005$; least square (ls) contrast, $P = 0.008$, Fig. 1a) and 39.5% more tissue ($F_{1,52.1} = 14.6$, $P = 0.0004$; ls contrast, $P = 0.005$, Fig. 1b) than offspring of risk-naïve parents, but only if they experienced risk as embryos. In contrast, parental experience with risk did not impact offspring shell length

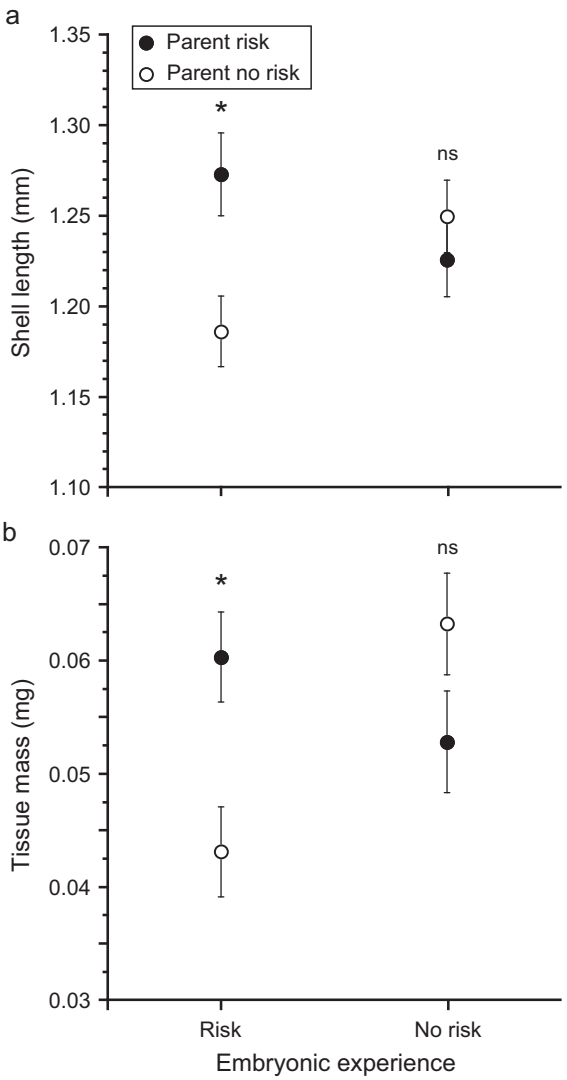

FIG. 1. (a) Shell length (mm) and (b) tissue mass (mg) at emergence of offspring *Nucella lapillus* that experienced the presence or absence of predation risk from the green crab *Carcinus maenas* as embryos. Values are mean ± SE. Offspring were produced by parents that were also exposed to the presence (solid circles) or absence (open circles) of predation risk from *Carcinus*. Results in panel a are from both experimental years, while in panel b, data were collected in 2016 only. Asterisk denotes a difference ($P \leq 0.05$) between means based on least square contrasts and ns denotes no statistical difference ($P > 0.05$) between means; exact P values are given in *Results*.

(ls contrast, $P = 0.6$, Fig. 1a) or tissue mass (ls contrast, $P = 0.1$, Fig. 1b) at emergence in the absence of embryonic risk. Duration of embryonic experience had no effect on offspring shell length ($F_{1,87.1} = 0.06$, $P = 0.81$) or tissue mass ($F_{1,45.0} = 0.36$, $P = 0.54$). Year ($\chi^2 = 18.5$, $P < 0.0001$) and parental pair ($\chi^2 = 7.2$, $P = 0.004$) had a significant effect on offspring shell length, but parental pair did not affect offspring tissue mass ($\chi^2 = 5.4$, $P = 0.1$). There was no effect of any treatment on offspring shell width (all $P > 0.09$; Appendix S2: Fig. S2a), shell mass (all $P > 0.39$; Appendix S2: Fig. S2b), number of offspring emerging per egg capsule (all $P > 0.29$; Appendix S2: Fig. S3a), or per capita number of nurse eggs (all $P > 0.30$, Appendix S2: Fig. S3b). Parents from both risk treatments produced egg capsules of similar length ($F_{1,17.3} = 0.61$, $P = 0.45$; Appendix S2: Fig. S4) and width ($F_{1,17.2} = 0.28$, $P = 0.60$; Appendix S2: Fig. S4).

In the presence of current risk, embryos that experienced risk during development respired 56% less (corrected for egg capsule volume and mass) if their parents were also exposed to risk (parental experience with risk × embryonic experience with risk × current risk, $F_{1,35.0} = 8.36$, $P = 0.006$; ls contrast, $P = 0.0003$, Fig. 2a, b). There was no effect of parental and embryonic experiences with risk in the absence of current risk (ls contrast, $P = 0.5$). Duration of embryonic experience did not affect embryonic respiration rates ($F_{1,40.3} = 0.83$, $P = 0.37$). Trial run had a significant effect on respiration rates ($\chi^2 = 15.9$, $P < 0.0001$). There was no relationship between egg capsule mass and egg capsule respiration rates ($F_{1,61.0} = 0.49$, $P = 0.48$, Fig. 3) across all treatment combinations. Finally, offspring of risk-experienced parents that experienced risk as embryos emerged 4.1 d (7%) sooner on average than other offspring, regardless of the duration of embryonic experience (parental experience with risk × embryonic experience with risk, $F_{1,86.0} = 9.64$, $P = 0.003$; ls contrast, $P = 0.001$, Fig. 4). Year ($\chi^2 = 33.9$, $P < 0.0001$) and parental pair ($\chi^2 = 15.7$, $P < 0.0001$) had a significant effect on time to emergence.

Discussion

Nucella offspring of risk-naïve parents produced shorter shells and less tissue in response to embryonic risk experience (Fig. 1a, b). This result was expected based on previous work that shows that juvenile *Nucella* grow less in the presence of green crab predation risk because of reductions in foraging and growth efficiency (Trussell et al. 2006). In contrast, the offspring of risk-experienced parents grew marginally longer shells (8%) and substantially more tissue (39.5%) than offspring of risk-naïve parents in response to embryonic risk experience (Fig. 1a, b), indicating that parental experience with predation risk positively affected embryonic performance in the presence of risk. Importantly, only the offspring of risk-naïve parents that experienced risk as embryos were smaller relative to those in other treatments. All other offspring were the same size at emergence (Fig. 1a, b). Hence, parental experience with risk only conferred a benefit to those offspring that experienced risk as embryos.

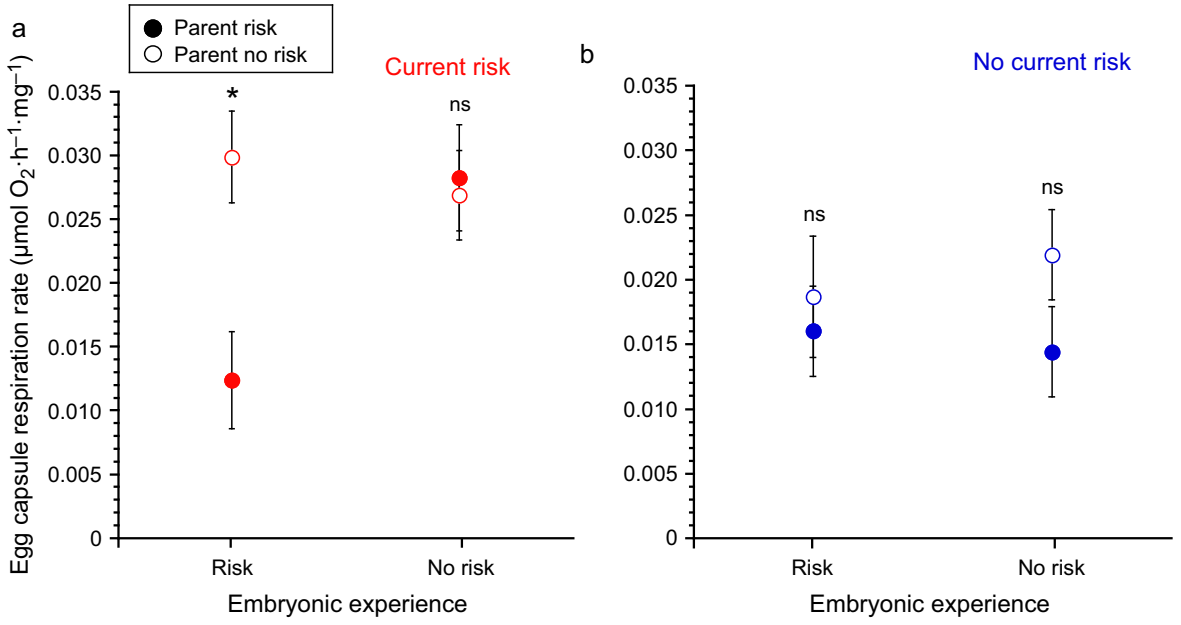

Fig. 2. Respiration rates (μmol $O_2 \cdot h^{-1} \cdot mg^{-1}$) of *Nucella lapillus* egg capsules that were exposed to the (a) presence or (b) absence of current predation risk from the green crab *Carcinus maenas* six weeks after deposition. Values are mean ± SE. Egg capsules had previously experienced the presence or absence of predation risk from *Carcinus* as embryos and were produced by parents that were also exposed to the presence (solid circles) or absence (open circles) of risk. Data were collected in 2016 only. Asterisk denotes a difference ($P \leq 0.05$) between means based on least square contrasts and ns denotes no statistical difference ($P > 0.05$) between means; exact P values are given in *Results*.

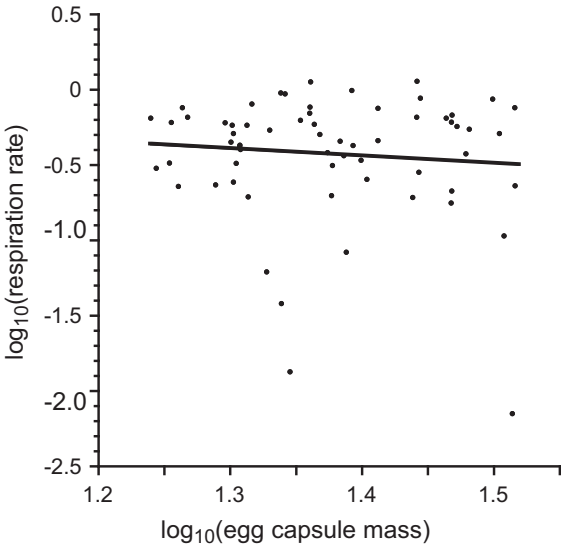

FIG. 3. Log_{10}-log_{10} relationship between egg capsule mass (mg) and egg capsule respiration rates (μmol $O_2 \cdot h^{-1} \cdot mg^{-1}$) of all egg capsules used in the respiration experiment. The slope of the resulting line of best fit is equivalent to the scaling exponent, *b*, which was not significantly different from 0.

A larger size at emergence is often, though not always (Kaplan 1992, McGhee et al. 2012), adaptive because larger offspring can be less vulnerable to predation (Petranka et al. 1987), more competitive (Einum and Fleming 1999), able to reach reproductive age more quickly (Marshall et al. 2003), or more metabolically efficient per unit body mass (Glazier 2005, Pettersen et al. 2015) than smaller offspring. If parental effects are to be adaptive for offspring, they must have a demonstrable positive effect on offspring fitness (Burgess and Marshall 2014). While we cannot be sure that a larger size at emergence confers higher lifetime fitness in our system, larger offspring have higher survivorship in *Nucella ostrina* (Moran and Emlet 2001), which also inhabit rocky shores with similar environmental constraints that likely enhance the adaptive value of larger size. A larger size may improve fitness through effects of energetic state on offspring foraging demands (reduced due to greater energetic reserves) and capabilities (greater prey variety and size options) or impacts of size on metabolic efficiency (due to allometric scaling, see below). For example, despite risk-induced reductions in foraging, larger, sub-adult *Nucella* with enhanced energetic reserves are more likely to remain in the safety of refuge habitats than smaller juveniles (Matassa et al. 2016). In addition, the considerable increase in offspring tissue mass in response to parental and embryonic experiences with risk is noteworthy because it reveals that the size patterns we observed were not a byproduct of induced defenses in shell traits (Appendix S2: Fig. S2a, b).

Offspring size at emergence was not affected by the duration of embryonic experience with predation risk. Hence, even short-term risk exposure was sufficient to prompt a stress response in offspring and may be particularly influential if it coincides with a developmental window when embryos are highly responsive to their external environment (e.g., Lehman and Campbell 2007). For *Nucella*, the first 10 days of development may be a sensitive window because embryos consume all resources (nurse eggs) during that time (Costello and Henley 1971). Because both the short and long duration of embryonic experience treatment levels overlapped with this potentially sensitive period, our experimental design may have limited our ability to distinguish its effects.

Early consumption of embryonic provisioning may also explain why offspring of risk-experienced parents that experienced risk as embryos emerged both larger and sooner than offspring of risk-naïve parents (Fig. 4). While longer development time frequently results in larger, more developed offspring (Sih and Moore 1993, Gillooly and Dodson 2000), larger offspring may also emerge sooner, as they did here, because of mounting developmental costs. For example, greater metabolic demands associated with larger size (Gillooly and Dodson 2000, Brown et al. 2004) and constraints on maximum achievable size within an egg capsule (Warkentin 2011) may make earlier hatching more favorable for larger offspring. Diminishing such costs may be especially important for species with crawl-away juveniles such as *Nucella*, where offspring begin to forage and improve their energetic state immediately after hatching (Crothers 1985, Warkentin 2011). On the other hand, the offspring of

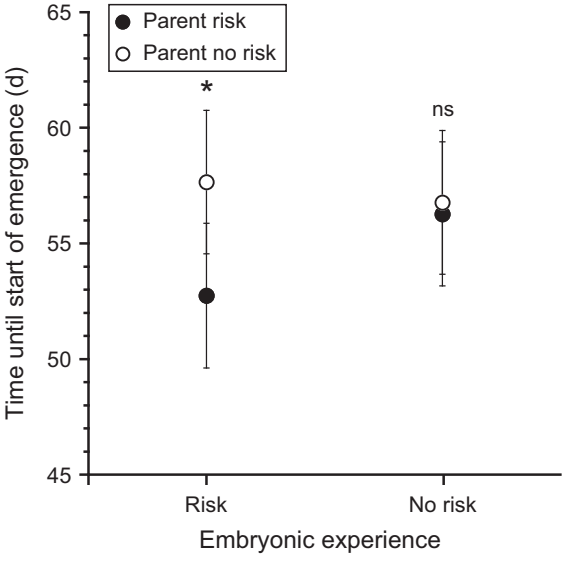

FIG. 4. Number of days until emergence of *Nucella lapillus* offspring that experienced the presence or absence of predation risk from the green crab *Carcinus maenas* as embryos. Values are mean ± SE. Offspring were produced by parents that were also exposed to the presence (solid circles) or absence (open circles) of predation risk from *Carcinus*. Results are from both experimental years. Asterisk denotes a difference ($P \leq 0.05$) between means based on least square contrasts and ns denotes no statistical difference ($P > 0.05$) between means; exact *P* values are given in *Results*.

risk-experienced parents that were also exposed to risk as embryos were the same size at emergence as offspring that were not exposed to risk as embryos (Fig. 1a, b), but still emerged sooner (Fig. 4). This suggests that emergence time might be governed both by size and by the offspring's assessment of their relative risk conditions (Warkentin 2011). All offspring were in risk-free conditions immediately before their emergence. Because offspring of risk-experienced parents that were also exposed to risk as embryos had previously experienced a relatively risky environment, this release from risk, along with their larger size, may have caused them to emerge sooner than offspring that did not experience risk as embryos even though they were all similarly sized. While plasticity in hatching time often exists due to an imminent threat from predators after emergence (Sih and Moore 1993), it is unlikely that such dynamics operated here because of substantial size disparities between newly hatched *Nucella* and *Carcinus*.

Egg capsules containing offspring of risk-experienced parents that experienced risk as embryos had 56% lower respiration rates than other egg capsules in the presence of risk, and their respiration rates were similar to those of egg capsules in the absence of risk (Fig. 2a, b). We suggest that these reduced respiration rates contributed to the ability of offspring to achieve a larger size at emergence by improving their metabolic efficiency (Pettersen et al. 2015). Prey increase their respiration rate in response to predation risk in many systems (Hawlena and Schmitz 2010) and are thus less efficient at converting the energy they consume into body mass (McPeek et al. 2001, Slos and Stoks 2008). This inefficiency likely emerges because of reallocation of energy away from growth into essential metabolic functions that help mitigate cellular damage caused by stress (Wingfield et al. 1998, Slos and Stoks 2008). Indeed, Trussell et al. (2006) found that exposure to green crab risk cues negatively affects *Nucella* growth efficiency and hypothesized that increased respiration rates may have been operating. Interestingly, others have shown that prey become physiologically conditioned after chronic predation risk, exhibiting reduced respiration after previous (Holopainen et al. 1997) or prolonged risk exposure (Steiner and Van Buskirk 2009). Hence, parental experience with risk may serve as a proxy for individual experience with risk that preconditions offspring of risk-experienced parents to expect risk. These effects may then diminish the physiological consequences for offspring exposed to risk. Mommer and Bell (2013) found suggestive evidence for a dampened physiological response (via lower plasma cortisol levels) to predation risk in adult stickleback offspring whose parents had been exposed to risk. Others have found an opposite effect: Giesing et al. (2010), also working with sticklebacks, showed that maternal experience with risk increased embryonic respiration rates early in development but importantly, this pattern only occurred when embryos were in risk-free conditions. Because parental effects are more likely to be adaptive when parental and offspring environments are complementary (Sheriff and Love 2013, Burgess and Marshall 2014), we suggest that the lower respiration rates of *Nucella* embryos that occurred because of parental and embryonic experiences with risk confer a fitness advantage that improves offspring energetic state in the presence of risk.

Respiration rates can scale allometrically with body mass in many organisms, such that larger individuals have lower respiration rates per unit body mass than smaller individuals (e.g., Schmidt-Nielsen 1984, Glazier 2005). The general absence of an allometric relationship between egg capsule size and egg capsule respiration rates (Fig. 3, see also Appendix S1) is perhaps not surprising given that our measurements of egg capsule mass include the non-respiring egg capsule structure. We were limited in our ability to conduct respiration trials on individual embryos because *Nucella* embryos exhibit poor survivorship outside of an egg capsule (Pechenik et al. 1984). Given these constraints, we cannot definitively determine whether reduced respiration rates were the cause or the effect of achieving a larger size. However, respiration rates were generally higher in the presence vs. absence of current risk for egg capsules containing offspring that would ultimately emerge at the same size (i.e., all other treatment combinations, Figs. 1a, b, 2a, b). Because current exposure to predation risk can affect the respiration rates of developing embryos independently of embryo size, it is possible that parental and embryonic risk experiences may influence respiration rates independently of embryo size. Our hypothesis that reduced respiration rates under predation risk lead to a larger size is intriguing and worth exploring in future work. If reduced respiration mechanistically drives the synergistic influence of parental and embryonic effects, it may have lifetime implications for offspring responses to predators. Regardless of the suggestive patterns in the respiration data, our results show that the interplay between parental and embryonic experiences can strongly affect offspring size at emergence and that organisms can be influenced by a complex interplay of environmental history.

Finally, the size patterns we observed operated independently of trade-offs between offspring size and number at emergence (Appendix S2: Fig. S3a). A large body of work exploring this trade-off (e.g., Smith and Fretwell 1974, Stearns 1992) suggests that resource limitation compels mothers to allocate reproductive resources to increase either offspring number or size. We may not have detected this trade-off here because the potential mechanism driving offspring size patterns, changes in respiration rate, operates independently of offspring number. In addition, we found no difference in parental provisioning or egg capsule size based on parental risk experience (Appendix S2: Figs. S3b, S4), so it is unlikely that these factors influenced offspring size at emergence.

Theory predicts that early life experiences, either direct (i.e., embryonic) or indirect (i.e., parental), should

have pronounced phenotypic effects because of their propensity to change the overall trajectory of an individual's phenotype (Fawcett and Frankenhuis 2015, English et al. 2016). Because species that reside in middle trophic levels comprise much of species diversity (60%; Williams and Martinez 2000), the synergistic effects of parental and embryonic exposure to predation risk may be pervasive. Notably, our results encompass a relatively narrow developmental snapshot of the effects of parental and embryonic experiences with risk on offspring fitness, and more work is necessary to understand whether their synergistic effects persist through later stages of ontogeny. In addition, parental and embryonic experiences may also interact in response to other environmental stressors. There is evidence, for example, for the independent effects of transgenerational (Miller et al. 2012) and early life experiences (Hettinger et al. 2012) on organism phenotypes in response to climate change, but our results suggest that exclusive focus on these independent effects may be insufficient. Our results provide clear evidence of a synergistic effect of parental and embryonic experiences with predation risk on offspring performance that is likely adaptive in risk conditions. Thus, attention to the interactive effects of stressors across prey life history will be key to better understanding how factors like predation risk ultimately shape the dynamics of species interactions and their consequences for natural communities.

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Literature Cited

Agrawal, A. A., C. Laforsch, and R. Tollrian. 1999. Transgenerational induction of defences in animals and plants. Nature 401:60–63.

Beaty, L. E., J. D. Wormington, B. J. Kensinger, K. N. Bayley, S. R. Goeppner, K. D. Gustafson, and B. Luttbeg. 2016. Shaped by the past, acting in the present: transgenerational plasticity of anti-predatory traits. Oikos 125:1570–1576.

Bernardo, J. 1996. Maternal effects in animal ecology. American Zoologist 36:83–105.

Bestion, E., A. Teyssier, F. Aubret, J. Clobert, and J. Cote. 2014. Maternal exposure to predator scents: offspring phenotypic adjustment and dispersal. Proceedings of the Royal Society B 281:20140701.

Brown, J. H., J. F. Gillooly, A. P. Allen, V. M. Savage, and G. B. West. 2004. Toward a metabolic theory of ecology. Ecology 85:1771–1789.

Burgess, S. C., and D. J. Marshall. 2014. Adaptive parental effects: the importance of estimating environmental predictability and offspring fitness appropriately. Oikos 123: 769–776.

Chivers, D. P., J. M. Kiesecker, A. Marco, J. Devito, M. T. Anderson, and A. R. Blaustein. 2001. Predator-induced life history changes in amphibians: egg predation induces hatching. Oikos 92:135–142.

Coslovsky, M., and H. Richner. 2011. Predation risk affects offspring growth via maternal effects. Functional Ecology 25:878–888.

Costello, D. P., and C. Henley. 1971. Methods for obtaining and handling marine eggs and embryos. Second edition. Marine Biological Laboratory, Woods Hole, Massachusetts, USA.

Crothers, J. 1985. Dog-whelks: an introduction to the biology of *Nucella lapillus* (L.). Field Studies 6:291–360.

Czarnołęski, M., J. Kozłowski, G. Dumiot, J.-C. Bonnet, J. Mallard, and M. Dupont-Nivet. 2008. Scaling of metabolism in *Helix aspersa* snails: changes through ontogeny and response to selection for increased size. Journal of Experimental Biology 211:391–399.

Donelan, S. C., and G. C. Trussell. 2015. Parental effects enhance risk tolerance and performance in offspring. Ecology 96:2049–2055.

Donelan, S. C., J. H. Grabowski, and G. C. Trussell. 2017. Refuge quality impacts the strength of nonconsumptive effects on prey. Ecology 98:403–411.

Einum, S., and I. A. Fleming. 1999. Maternal effects of egg size in brown trout (*Salmo trutta*): norms of reaction to environmental quality. Proceedings of the Royal Society B 266:2095–2100.

English, S., T. W. Fawcett, A. D. Higginson, P. C. Trimmer, T. Uller, J.-M. Gaillard, and J. L. Bronstein. 2016. Adaptive use of information during growth can explain long-term effects of early life experiences. American Naturalist 187:620–632.

Etter, R. J. 1989. Life history variation in the intertidal snail *Nucella lapillus* across a wave-exposure gradient. Ecology 70:1857–1876.

Fawcett, T. W., and W. E. Frankenhuis. 2015. Adaptive explanations for sensitive windows in development. Frontiers in Zoology 12:S3.

Fraser, D. F., and J. F. Gilliam. 1992. Nonlethal impacts of predator invasion: facultative suppression of growth and reproduction. Ecology 73:959–970.

Giesing, E. R., C. D. Suski, R. E. Warner, and A. M. Bell. 2010. Female sticklebacks transfer information via eggs: effects of maternal experience with predators on offspring. Proceedings of the Royal Society B 278:1753–1759.

Gillooly, J., and S. Dodson. 2000. The relationship of neonate mass and incubation temperature to embryonic development time in a range of animal taxa. Journal of Zoology 251:369–375.

Glazier, D. S. 2005. Beyond the '3/4-power law': variation in the intra- and interspecific scaling of metabolic rate in animals. Biological Reviews 80:611–662.

Harvell, C. D. 1990. The ecology and evolution of inducible defenses. Quarterly Review of Biology 65:323–340.

Hawlena, D., and O. J. Schmitz. 2010. Physiological stress as a fundamental mechanism linking predation to ecosystem functioning. American Naturalist 176:537–556.

Hettinger, A., E. Sanford, T. M. Hill, A. D. Russell, K. N. Sato, J. Hoey, M. Forsch, H. N. Page, and B. Gaylord. 2012. Persistent carry-over effects of planktonic exposure to ocean acidification in the Olympia oyster. Ecology 93:2758–2768.

Holopainen, I. J., J. Aho, M. Vornanen, and H. Huuskonen. 1997. Phenotypic plasticity and predator effects on morphology

and physiology of crucian carp in nature and in the laboratory. Journal of Fish Biology 50:781–798.

Kaplan, R. H. 1992. Greater maternal investment can decrease offspring survival in the frog *Bombina orientalis*. Ecology 73:280–288.

Kats, L. B., and L. M. Dill. 1998. The scent of death: chemosensory assessment of predation risk by prey animals. Ecoscience 5:361–394.

Lehman, E. M., and C. D. Campbell. 2007. Developmental window of response to predator chemical cues in rough-skinned newt embryos. Functional Ecology 21:880–885.

Lima, S. L. 1998. Nonlethal effects in the ecology of predator-prey interactions. BioScience 48:25–34.

Lima, S. L., and L. M. Dill. 1990. Behavioral decisions made under the risk of predation: a review and prospectus. Canadian Journal of Zoology 68:619–640.

Ludwig, D., and L. Rowe. 1990. Life-history strategies for energy gain and predator avoidance under time constraints. American Naturalist 135:686–707.

Mangel, M., and C. W. Clark. 1986. Towards a unified foraging theory. Ecology 67:1127–1138.

Marshall, D. J., T. F. Bolton, and M. J. Keough. 2003. Offspring size affects the post-metamorphic performance of a colonial marine invertebrate. Ecology 84:3131–3137.

Matassa, C. M., S. C. Donelan, B. Luttbeg, and G. C. Trussell. 2016. Resource levels and prey state influence antipredator behavior and the strength of nonconsumptive predator effects. Oikos 125:1478–1488.

McGhee, K. E., L. M. Pintor, E. L. Suhr, and A. M. Bell. 2012. Maternal exposure to predation risk decreases offspring antipredator behaviour and survival in threespined stickleback. Functional Ecology 26:932–940.

McPeek, M. A., M. Grace, and J. M. Richardson. 2001. Physiological and behavioral responses to predators shape the growth/predation risk trade-off in damselflies. Ecology 82: 1535–1545.

Metcalfe, N. B., and P. Monaghan. 2001. Compensation for a bad start: grow now, pay later? Trends in Ecology & Evolution 16:254–260.

Miller, G. M., S.-A. Watson, J. M. Donelson, M. I. McCormick, and P. L. Munday. 2012. Parental environment mediates impacts of increased carbon dioxide on a coral reef fish. Nature Climate Change 2:858–861.

Mommer, B. C., and A. M. Bell. 2013. A test of maternal programming of offspring stress response to predation risk in threespine sticklebacks. Physiology & Behavior 122:222–227.

Moran, A., and R. Emlet. 2001. Offspring size and performance in variable environments: field studies on a marine snail. Ecology 82:1597–1612.

Mousseau, T. A., and C. W. Fox. 1998. Maternal effects as adaptations. Oxford University Press, Oxford, UK.

Orizaola, G., and F. Braña. 2005. Plasticity in newt metamorphosis: the effect of predation at embryonic and larval stages. Freshwater Biology 50:438–446.

Palmer, A. R. 1983. Relative cost of producing skeletal organic matrix versus calcification: evidence from marine gastropods. Marine Biology 75:287–292.

Pechenik, J. A., S. C. Chang, and A. Lord. 1984. Encapsulated development of the marine prosobranch gastropod *Nucella lapillus*. Marine Biology 78:223–229.

Peckarsky, B. L., B. W. Taylor, A. R. McIntosh, M. A. McPeek, and D. A. Lytle. 2001. Variation in mayfly size at metamorphosis as a developmental response to risk of predation. Ecology 82:740–757.

Petranka, J. W., L. B. Kats, and A. Sih. 1987. Predator-prey interactions among fish and larval amphibians: use of chemical cues to detect predatory fish. Animal Behaviour 35:420–425.

Pettersen, A. K., C. R. White, and D. J. Marshall. 2015. Why does offspring size affect performance? Integrating metabolic scaling with life-history theory. Proceedings of the Royal Society B 282:20151946.

Pinheiro, J., D. Bates, S. DebRoy, D. Sarkar, and R Core Team. 2017. nlme: linear and nonlinear mixed effects models. R package version 3.1-131. https://CRAN.R-project.org/package=nlme

R Core Team. 2017. R: a language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria.

Rawlings, T. A. 1990. Associations between egg capsule morphology and predation among populations of the marine gastropod, *Nucella emarginata*. Biological Bulletin 179: 312–325.

Reznick, D., and A. P. Yang. 1993. The influence of fluctuating resources on life history: patterns of allocation and plasticity in female guppies. Ecology 74:2011–2019.

Schmidt-Nielsen, K. 1984. Scaling: why is animal size so important?. Cambridge University Press, Cambridge, UK.

Sheriff, M., and O. Love. 2013. Determining the adaptive potential of maternal stress. Ecology Letters 16:271–280.

Sheriff, M. J., C. J. Krebs, and R. Boonstra. 2010. The ghosts of predators past: population cycles and the role of maternal programming under fluctuating predation risk. Ecology 91:2983–2994.

Shine, R., and S. J. Downes. 1999. Can pregnant lizards adjust their offspring phenotypes to environmental conditions? Oecologia 119:1–8.

Sih, A. 1980. Optimal behavior: can foragers balance two conflicting demands? Science 210:1041–1043.

Sih, A., and R. D. Moore. 1993. Delayed hatching of salamander eggs in response to enhanced larval predation risk. American Naturalist 142:947–960.

Sinervo, B. 1990. The evolution of maternal investment in lizards: an experimental and comparative analysis of egg size and its effects on offspring performance. Evolution 44:279–294.

Slos, S., and R. Stoks. 2008. Predation risk induces stress proteins and reduces antioxidant defense. Functional Ecology 22:637–642.

Smith, C. C., and S. D. Fretwell. 1974. The optimal balance between size and number of offspring. American Naturalist 108:499–506.

Spight, T. M. 1976. Hatching size and the distribution of nurse eggs among prosobranch embryos. Biological Bulletin 150:491–499.

Stearns, S. C. 1992. The evolution of life histories. Oxford University Press, Oxford, UK.

Steiner, U. K., and J. Van Buskirk. 2009. Predator-induced changes in metabolism cannot explain the growth/predation risk tradeoff. PLoS ONE 4:e6160.

Stoks, R., M. De Block, and M. A. McPeek. 2006. Physiological costs of compensatory growth in a damselfly. Ecology 87:1566–1574.

Storm, J. J., and S. L. Lima. 2010. Mothers forewarn offspring about predators: a transgenerational maternal effect on behavior. American Naturalist 175:382–390.

Tollrian, R. 1995. Predator-induced morphological defenses: costs, life history shifts, and maternal effects in *Daphnia pulex*. Ecology 76:1691–1705.

Trussell, G. C., P. J. Ewanchuk, and M. D. Bertness. 2003. Trait-mediated effects in rocky intertidal food chains: predator risk cues alter prey feeding rates. Ecology 84:629–640.

Trussell, G. C., P. J. Ewanchuk, and C. M. Matassa. 2006. The fear of being eaten reduces energy transfer in a simple food chain. Ecology 87:2979–2984.

Uller, T. 2008. Developmental plasticity and the evolution of parental effects. Trends in Ecology & Evolution 23:432–438.

Vonesh, J. R., and B. M. Bolker. 2005. Compensatory larval responses shift trade-offs associated with predator-induced hatching plasticity. Ecology 86:1580–1591.

Warkentin, K. M. 1995. Adaptive plasticity in hatching age: a response to predation risk trade-offs. Proceedings of the National Academy of Sciences USA 92:3507–3510.

Warkentin, K. M. 2011. Plasticity of hatching in amphibians: evolution, trade-offs, cues and mechanisms. Integrative and Comparative Biology 51:111–127.

Werner, E. E., and J. F. Gilliam. 1984. The ontogenetic niche and species interactions in size-structured populations. Annual Review of Ecology, Evolution, and Systematics 15: 393–425.

Werner, E. E., G. G. Mittelbach, D. J. Hall, and J. F. Gilliam. 1983. Experimental tests of optimal habitat use in fish: the role of relative habitat profitability. Ecology 64:1525–1539.

White, C. R., and M. R. Kearney. 2014. Metabolic scaling in animals: methods, empirical results, and theoretical explanations. Comprehensive Physiology 4:231–256.

Williams, R. J., and N. D. Martinez. 2000. Simple rules yield complex food webs. Nature 404:180–183.

Wingfield, J. C., L. M. Donna, W. B. Creagh, J. D. Jacobs, L. Sharon, M. Ramenofsky, and D. R. Ralph. 1998. Ecological bases of hormone-behavior interactions: the "emergency life history stage". American Zoologist 38:191–206.

Zuur, A. F., E. N. Ieno, N. J. Walker, A. A. Saveliev, and G. M. Smith. 2009. Mixed effects models and extensions in ecology with R. Springer, New York, New York, USA.

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