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Parental effects enhance risk tolerance and performance in offspring

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Abstract. Predation risk can strongly influence community dynamics through its effects on prey foraging decisions that often involve habitat shifts (i.e., from risky to refuge habitats). Although the within-generation effects of risk on prey are well appreciated, the effects of parental experience with risk on offspring decision-making and growth are poorly understood. The capacity of parents to prepare their offspring for potential risk exposure may be adaptive when the likelihood of eventual risk exposure is high and be instrumental in shaping how offspring allocate their foraging effort and habitat use. Using a simple rocky intertidal food chain, we examined the influence of parental exposure to predator risk cues on the behavior, foraging, and tissue maintenance of offspring exposed to the presence and absence of risk. We found that offspring of risk-experienced parents were bolder. When confronted with risk, these offspring spent more time out of refuge habitat, foraged more, and maintained more tissue than offspring of risk-free parents. Thus, parental experience with risk was most important when offspring were exposed to risk. These results suggest that the effects of parental experience with predation risk on offspring traits strongly shape the transmission of risk effects in ecological communities.

Key words: Carcinus maenas; *foraging/predation risk trade-off; nonconsumptive effect;* Nucella lapillus; *predation risk; transgenerational phenotypic plasticity*.

Introduction

In many natural systems, environmental variation can strongly influence individual fitness and community organization and dynamics (Menge and Sutherland 1987, Hutchings et al. 2000). Natural selection has favored the evolution of different adaptive strategies to environmental variation (e.g., canalization, genetic polymorphism), but phenotypic plasticity is particularly common in a wide variety of taxa and ecosystems (Levins 1968, Stearns 1989, Tollrian and Harvell 1999). Phenotypic plasticity is expected to be favored in unpredictable environments, especially when phenotypic modification results in phenotypes adapted to local environmental conditions (Stearns 1989, Scheiner 1993, Via et al. 1995). Inducible defenses are a common form of plasticity describing the modification, for example, of prey morphology (Lively 1986, Trussell 1996) or behavior (Turner and Mittelbach 1990, Schmitz et al. 1997, Trussell et al. 2003) in response to cues signaling predation risk.

There has been considerable interest in the effects of predation risk on prey foraging behavior (Werner and Peacor 2003) and how these shape community dynamics (Schmitz et al. 2004, Peckarsky et al. 2008) and ecosystem processes such as nutrient cycling (Schmitz et al. 2010). Moreover, such changes in prey foraging behavior are often accompanied by reduced growth or changes in prey energetic status that can feedback to influence subsequent foraging decisions and their impact on the community (Trussell and Schmitz 2012). Under predation risk, prey often shift their habitat use, such as retreating to refuges, to reduce their risk of being consumed (Crowder and Cooper 1982, Sih 1992, Lima 1998). The positive effects of such habitat shifts, however, can also involve costs such as decreased food availability (Orrock et al. 2013), increased competition with conspecifics (Persson 1993), or lower quality resources (Schmitz et al. 1997). Thus, habitat shifts can be a key factor in the calculus of prey foraging decisions, their consequences for prey fitness, and the strength of trait-mediated trophic cascades both in and out of refuge habitats (Grabowski 2004, Creel et al. 2005, Orrock et al. 2013).

In order for organisms to express the appropriate phenotype in a given environment, cues indicating environmental conditions must be reliable (e.g., Levins 1968, Moran 1992, Scheiner 1993). Individual experience is clearly important to the assessment of an organism's surroundings, but the environment experienced by its parents can also provide reliable information on the conditions that offspring are likely to encounter (Mousseau and Fox 1998). Hence, based on their own environmental experience, parents may program offspring to express specific phenotypes via parental effects (i.e., transgenerational phenotypic plasticity; Mousseau and Dingle 1991). Parental effects, particularly for young offspring, are evident in many taxa and in

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response to a variety of biotic and abiotic environmental cues (see Bernardo 1996, Rossiter 1996, Mousseau and Fox 1998). Growing evidence, however, indicates that parental effects, such as the influence of parental provisioning on offspring body size (Reznick et al. 1996, Sinervo and Doughty 1996), can operate throughout an offspring's lifetime as well as affect multiple generations (Agrawal et al. 1999, Plaistow et al. 2006). Moreover, parental exposure to predation risk can affect offspring morphology (e.g., Agrawal et al. 1999) and behavior (Storm and Lima 2010, McGhee et al. 2012), thereby influencing population and community dynamics by changing the foraging success of predators on offspring (Tollrian 1995, Agrawal et al. 1999, Storm and Lima 2010, McGhee et al. 2012).

Here we examine how parental and offspring exposure to predation risk interact to influence offspring behavior, foraging, and tissue maintenance. We found that the offspring of risk-experienced parents increased the time they spent in risky habitats and their per capita foraging in these habitats, and were better at maintaining their tissue biomass. Importantly, this effect only occurred when offspring were confronted with risk. Our results suggest that parental experience with predation risk and its resulting impact on offspring foraging and performance may have important consequences for individual fitness and population and community dynamics.

Materials and Methods

We conducted a laboratory mesocosm experiment using a tritrophic intertidal food chain consisting of the predatory green crab, *Carcinus maenas*, its prey, the carnivorous snail *Nucella lapillus* (see Plate 1), and barnacles (*Semibalanus balanoides*) as a basal resource for *Nucella*. We examined how exposure of *Nucella* parents to *Carcinus* risk cues interacted with offspring exposure to *Carcinus* risk cues to influence offspring habitat use, foraging, and tissue maintenance. We fully crossed two treatments, each with two levels: (1) parental risk experience (risk/no risk) and (2) offspring risk exposure (risk/no risk), and each treatment combination was replicated six times.

We collected *Nucella* parents from an exposed shoreline (Bennett Head, Nahant, Massachusetts, USA) in early April. *Nucella* fertilize internally and lay individual egg capsules from which juveniles emerge and immediately begin foraging (Crothers 1985). Parental mesocosms consisted of two, smaller chambers: (1) an upstream chamber (11.5 cm diameter × 10 cm tall) that held either a single male green crab (risk; mean carapace width ± SD, 72.4 ± 3.9 mm) or no crab (no risk) to manipulate parental experience with risk and (2) a downstream chamber (8 cm diameter × 10 cm tall) containing *Nucella* parents (one male and one female *Nucella*, mean shell length ± SD, 23.0 ± 1.2 mm) and their food (six blue mussels, *Mytilus edulis*, replenished every four days, mean shell length ± SD, 16.9 ± 3.3 mm). We checked the downstream parent chambers for newly laid *Nucella* egg capsules every four days. Egg capsules laid by a given parent pair were removed, placed together in small containers with seawater (changed every other day), and kept in an incubator at temperatures commensurate with ambient seawater until offspring emerged. Egg capsules were produced consistently throughout the 10 weeks of parental exposure in both risk treatments, and the length of exposure did not vary between treatments for offspring used in the experiment ($P = 0.96$). Upon hatching, offspring were given 10 juvenile mussels (mean shell length ± SD, 3.80 ± 0.98 mm) as food; new mussels were added as needed to provide an ad libitum supply. Before the onset of winter, offspring were transferred to larger containers (8 cm diameter × 10 cm tall) and remained in risk-free conditions until the experiment began the following July (10–12 months after hatching).

In July, offspring risk exposure was manipulated by placing offspring into mesocosms consisting of an outer chamber (14 × 14 × 16.5 cm) that housed two, smaller, perforated chambers: an upstream chamber (13 × 13 × 7.5 cm) for risk manipulation (risk and no risk, as described for parents) and a downstream chamber (12 × 8.5 × 6.5 cm) that housed three *Nucella* offspring (mean shell length ± SD, 19.8 ± 2.9 mm) and two granite tiles (7.5 × 7.5 × 1 cm) sandwiched back to back. *Nucella* offspring from 18 total parent pairs (10 risk-experienced and eight risk-free pairs) were randomly assigned to experimental replicates, but were kept with other offspring from the same parental risk conditions. Each pair of tiles was raised off the bottom of the mesocosm using PVC spacers (1 cm). The top tile was entirely covered with barnacles (mean density ± SD, 397.3 ± 24.3) and provided a risky habitat for offspring foraging. These barnacle communities were established by anchoring granite tiles (7.5 × 7.5 × 1 cm) in the field during the recruitment season (March). Barnacles recruited onto tiles for about two months and were then retrieved and maintained in laboratory tanks supplied with running seawater until their use in the experiment. The bottom tile created a refuge space for the offspring, but contained no barnacles. We manipulated refuge quality in this way to mimic natural refuges, which are often resource poor compared to risky habitats in the field (Schmitz 1998, Trussell et al. 2006*a*).

We monitored *Nucella* offspring tissue maintenance by marking each with a bee tag and weighing them at the beginning and end of the experiment using a nondestructive buoyant weighing technique (see Palmer 1982). Tissue maintenance was calculated by subtracting initial tissue mass (mg) from final tissue mass. The experiment ran for 27 days. Individual offspring behavior was recorded on the first day and every three days thereafter (10 total observations). From these behavioral observations, we calculated the proportion of time spent in the risky habitat by each individual

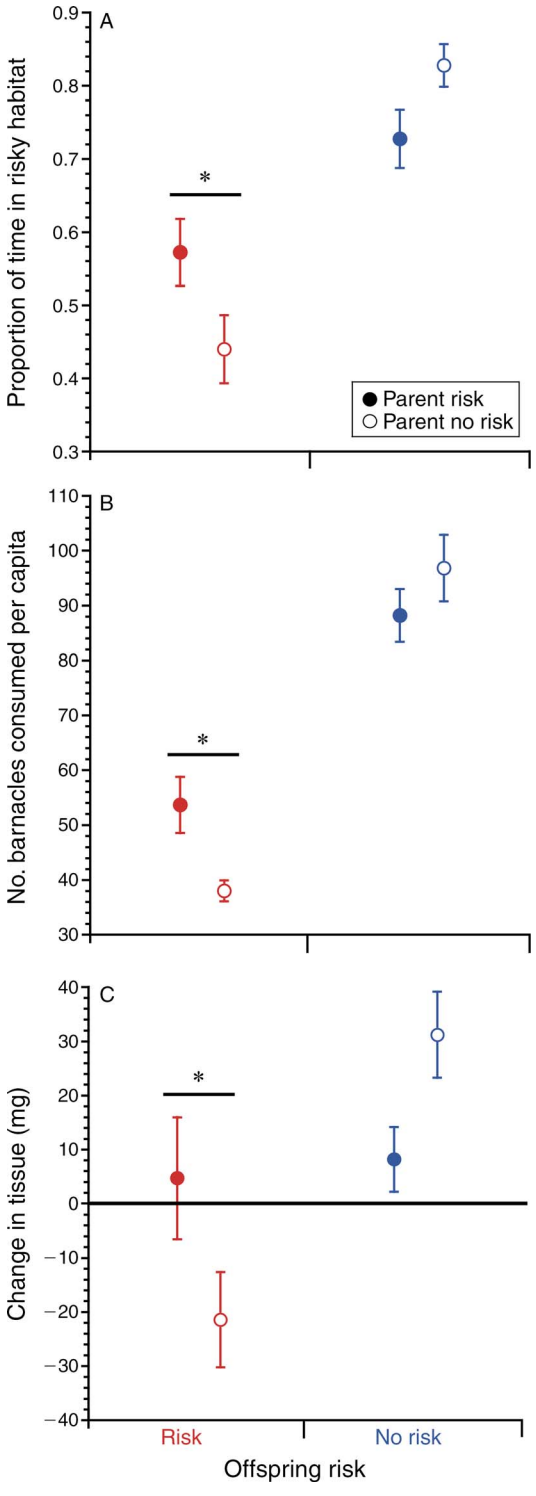

FIG. 1. Responses of *Nucella lapillus* offspring from risk-experienced (solid circles) and risk-free (open circles) parents, which were then maintained in the presence (red) and absence (blue) of predation risk signaled by water-borne cues from the green crab, *Carcinus maenas*, for 27 days. Parents that mated in the presence and absence of green crab risk cues produced these offspring. Shown are (A) proportion of time spent by offspring in the risky habitat, (B) per capita foraging of offspring, and (C) change in offspring tissue. Values are means ± SE. Asterisks denote differences between means based on a priori contrasts comparing the effect of parental experience with risk on offspring in the presence (red) or absence (blue) of risk (linear contrasts, $P < 0.05$).

snail. Each behavioral observation was scored as either risk (1) or no risk (0), and we divided the number of observations in the risky habitat by the total number of behavioral observations. We also digitally photographed barnacle tiles at the beginning and end of the experiment, and resulting images were scored in Photoshop (v. CS4; Adobe Systems, San Jose, California, USA) for live and dead barnacles to track *Nucella* foraging rates (see Matassa and Trussell 2011). We subtracted the number of barnacles alive at the end of the experiment from the number of barnacles alive at the beginning of the experiment to calculate the total number of barnacles consumed and then divided by the density of *Nucella* offspring in each replicate ($n = 3$) to calculate per capita foraging. All analyses were conducted on replicate averages.

We analyzed proportion of time spent in the risky habitat, per capita number of barnacles consumed, and the change in offspring tissue mass using two-way ANOVAs that considered parental experience with risk and offspring exposure to risk as fixed effects. A priori, we sought to determine whether parental experience with risk affected offspring traits when offspring were subjected to the presence and absence of risk. Hence, we performed two a priori linear contrasts after the full analysis for each offspring trait. One replicate (parent no risk, offspring no risk) was excluded from the tissue maintenance analysis due to measurement error. All analyses were performed using JMP software (Version 11; SAS Institute, Cary, North Carolina, USA).

RESULTS

A significant interaction ($F_{1,20} = 8.04$, $P = 0.01$, Fig. 1A) indicated that the proportion of time *Nucella* offspring spent in the risky habitat depended on parental experience with risk ($F_{1,20} = 0.15$, $P = 0.70$) and offspring exposure to risk ($F_{1,20} = 44.05$, $P < 0.0001$). The offspring of risk-free parents spent significantly less time (47% less) in the risky habitat when exposed to risk than offspring that were not exposed to risk. This pattern also emerged for the offspring of risk-experienced parents, but it was not as strong (21%). Moreover, when exposed to risk, the offspring of risk-experienced parents spent significantly more time (30% more) in the risky habitat than the offspring of risk-free parents (linear contrast, $P = 0.03$). Parental experience with risk was unimportant when offspring were not exposed to risk (linear contrast, $P = 0.10$).

We also found an interaction ($F_{1,20} = 6.54$, $P = 0.02$, Fig. 1B) between the effects of parental experience with risk ($F_{1,20} = 0.55$, $P = 0.47$) and offspring exposure to

risk ($F_{1,20} = 97.05$, $P < 0.0001$) on the per capita foraging of *Nucella* offspring (Fig. 1B). The offspring of risk-free parents foraged less (61%) when exposed to risk than when not exposed to risk. This pattern also emerged for the offspring of risk-experienced parents, but was not as strong (39%). Moreover, when exposed to risk, the offspring of risk-experienced parents foraged more (41%, linear contrast, $P = 0.03$) than those of risk-free parents. Parental experience with risk was unimportant to offspring foraging in the absence of risk (linear contrast, $P = 0.21$).

Finally, there was also a significant interaction ($F_{1,19} = 7.75$, $P = 0.01$, Fig. 1C) between the effects of parental experience with risk ($F_{1,19} = 0.03$, $P = 0.87$) and offspring exposure to risk ($F_{1,19} = 10.09$, $P = 0.005$) on tissue maintenance of *Nucella* offspring. The offspring of risk-free parents lost tissue when exposed to risk and therefore produced 168% less tissue than those that were not exposed to risk. In contrast, a different pattern emerged for the offspring of risk-experienced parents because they maintained the same amount of tissue regardless of risk environment. Importantly, when confronted with risk, the offspring of risk-experienced parents maintained their initial tissue mass, but the offspring of risk-free parents lost tissue (linear contrast, $P = 0.04$). Parental experience with risk did not impact tissue maintenance when offspring were not exposed to risk (linear contrast, $P = 0.09$).

Discussion

When prey are confronted with predation risk, they must balance their need to feed with the risk of being eaten (Lima et al. 1985, Werner and Hall 1988, Lima and Dill 1990). In many systems, prey facing this dilemma utilize refuge habitats to reduce their vulnerability to predators at the expense of reduced foraging gains because these habitats are often resource poor (Orrock et al. 2013). Consistent with a considerable body of work (for review, see Werner and Peacor [2003]), this trade-off was evident in the offspring of risk-free parents: when exposed to risk, these offspring spent less time in the risky habitat and foraged and grew less than their counterparts that were not exposed to risk.

Although a number of factors, such as prey state (Luttbeg et al. 2003), predator foraging mode (Schmitz et al. 2004), resource identity (Trussell et al. 2008), and refuge quality (Grabowski 2004, Orrock et al. 2013) can shape whether prey decide to feed or hide, this study reveals that parental experience with risk can strongly shape prey foraging decisions and their consequences. When subjected to risk, the offspring of risk-experienced parents were bolder than those of risk-free parents, spending 30% more time in the risky habitat. As a result, the offspring of risk-experienced parents also foraged 41% more under risk and were able to maintain more tissue than the offspring of risk-free parents.

The increased boldness we observed under risk in the offspring of risk-experienced parents may seem counterintuitive because prey often increase their antipredator behavior when confronted with risk (Lima and Dill 1990, Lima 1998), as we observed in the offspring of risk-free parents. However, prey under high or chronic risk are often bolder, perhaps because the costs of constant vigilance (e.g., starvation) become untenable (Fraser and Gilliam 1987) or the uncertainty regarding the value of foraging out of refuge under low and variable risk is more stressful to prey than constant, high risk (Sih 1992, Trussell et al. 2011). These scenarios are consistent with risk allocation theory, which posits that prey foraging decisions will depend on both the duration of and variation in risk exposure (the Risk Allocation Hypothesis; Lima and Bednekoff 1999). Hence, prey are predicted to increase their foraging effort as periods of risk grow longer because sacrificing foraging for safety becomes less adaptive as the risk of starvation increases. In our study, parental experience with risk may enhance offspring perception of risk and their assessment of the likelihood that risk will persist. Both scenarios may promote bolder foraging behavior in offspring when they are confronted with risk.

In contrast to our results, we note that previous work has found that parental experience with risk can increase offspring antipredator behavior under risk (Shine and Downes 1999, Storm and Lima 2010, Giesing et al. 2011, Bestion et al. 2014). However, unlike our study, offspring in these experiments were exposed to risk for very brief periods of time (<20 minutes). Hence, as predicted by the Risk Allocation Hypothesis, such short risk exposure should enhance antipredator behavior in prey, whereas the longer duration of risk exposure in our study (27 days) may have reduced such behavior in our offspring as the costs of antipredator behavior mount.

It is remarkable that parental experience with risk continued to influence offspring traits even though offspring were held in risk-free conditions for ~10–12 months with ad libitum food prior to the experiment. At present, the precise mechanism underlying these parental effects remains unclear. We think it is unlikely that parental-based differences in offspring energetic state were operating at the beginning of the experiment because offspring had substantial time to make up any potential energetic deficits before the experiment began. Moreover, offspring were the same size (mean shell length ± SD, 1.18 ± 0.17 mm, $P = 0.60$) when they emerged from egg capsules in both parental risk treatments. This similarity in size also suggests that induction at the egg capsule stage was not operating because snails exposed to risk are often smaller due to reduced growth rates (Trussell et al. 2006*b*). In any case, epigenetic programming represents one potential mechanism for the transmission of parental effects, and in other systems such programming varies based on parental environment (Jablonka and Raz 2009, Holeski et al. 2012). Clearly, more work is needed to better

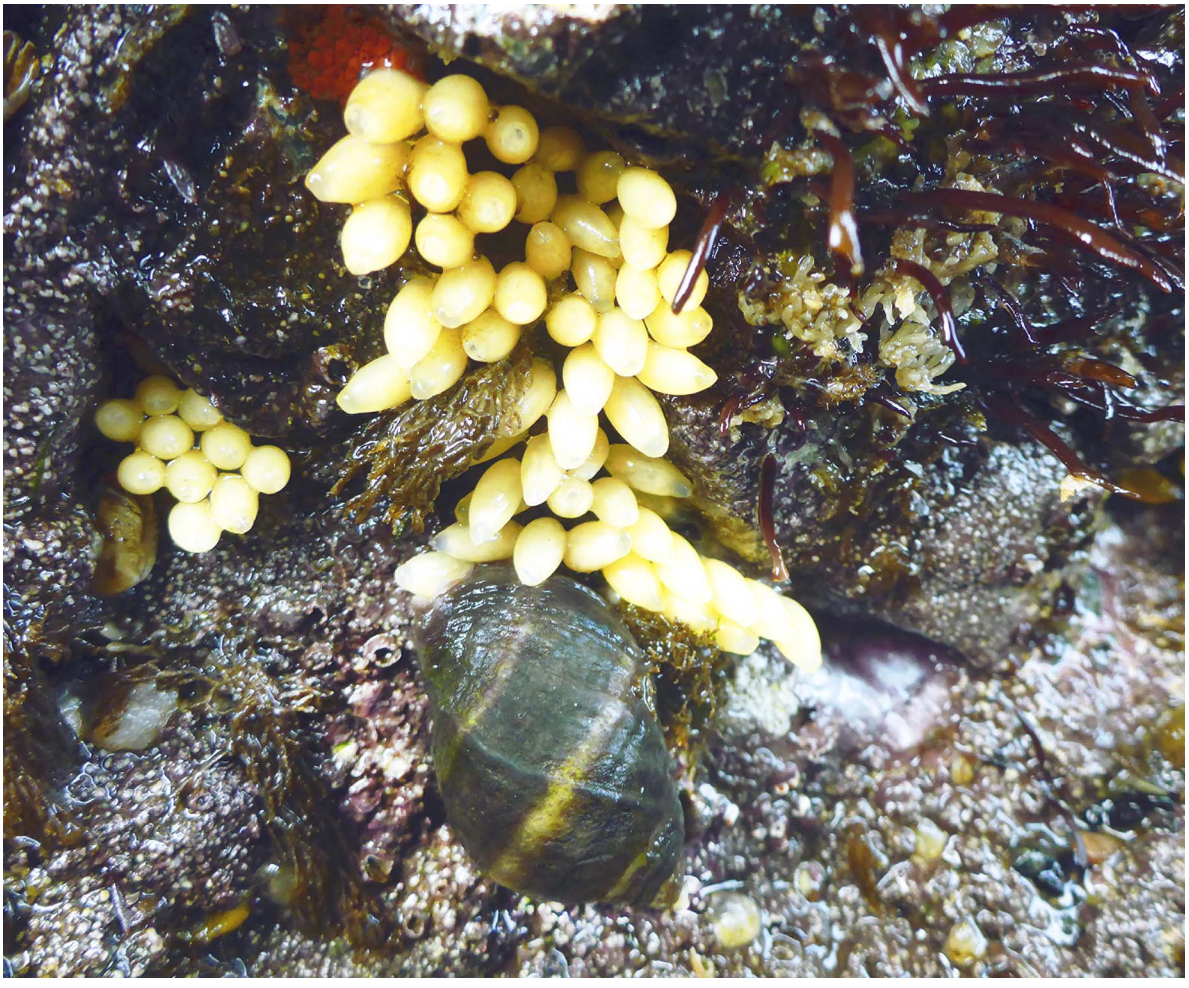

PLATE 1. Adult *Nucella lapillus* laying egg capsules on a rocky shore in Nahant, Massachusetts (USA). Experience of *Nucella* parents with predator risk cues from *Carcinus maenus* impacts the response of their offspring to risk. Photo credit: S. C. Donelan.

understand the potential mechanisms that shape parental effects at both the parental and egg capsule stage and whether offspring in our treatments differ in their willingness to approach potential starvation thresholds that are central to the Risk Allocation Hypothesis. Nevertheless, the persistent influence of parental effects in this study suggests that they are key to risk levels perceived by offspring and the degree of reliability that offspring assign to their assessment of risk once their environment becomes risky.

The effects of parental experience with risk on the foraging of their offspring had important consequences for prey tissue maintenance (change in tissue mass). Under risk, the offspring of risk-experienced parents both foraged more and maintained more tissue than the offspring of risk-free parents. Indeed, the offspring of risk-experienced parents were able to maintain their initial tissue levels (~5 mg above that at the beginning of the experiment) whereas the offspring of risk-free parents lost ~21 mg of their initial tissue mass. Moreover, the offspring of risk-experienced parents maintained the same amount of tissue (Fig. 1C) under both risk treatments despite large differences in foraging (Fig. 1B). Thus, while differences in foraging rate are surely operating, the observed differences in tissue maintenance may also be shaped by how efficiently offspring were able to satisfy basal metabolic requirements as well as translate acquired energy into tissue (Trussell et al. 2006*b*). Work in other systems has found that prey under risk can compensate for their reduced foraging by increasing assimilation efficiency and incorporating more necessary nutrients from their resources than prey held without risk, leading to similar growth in both situations (Thaler et al. 2012).

Elsewhere (Trussell et al. 2006*b*), we have shown that predation risk can greatly reduce prey growth efficiency, perhaps by altering a variety of physiological pathways that adversely impact prey energy budgets (also see McPeek et al. 2001, Stoks et al. 2005, Hawlena and Schmitz 2010*a*, *b*, Trussell and Schmitz 2012). We

suggest that information conveyed by risk-experienced parents may reduce the impact of risk-induced physiological stress on their offspring, thereby allowing them to make the most of their foraging effort. Such effects would explain why offspring of risk-experienced parents were able to maintain just as much tissue under risk as their counterparts in the absence of risk. However, this similarity in tissue maintenance may also partly reflect costs of risk-experienced parents when their offspring are not confronted with risk. Our data suggest that such costs may exist because in the absence of risk, offspring of risk-experienced parents displayed a trend ($P = 0.09$) for lower tissue production than the offspring of risk-free parents even though their foraging rates were similar. Nevertheless, our study reveals that offspring under risk perform better if their parents were exposed to risk, and suggests that parental experience may be costly to offspring when they are not exposed to risk.

It is important to highlight that the offspring used in our experiment were about a year old and thus approaching sexual maturity (Etter 1989). Because of the significant investment associated with reproduction, energetic constraints at this stage of life history may be especially important to reproductive success. In our experiment, the offspring of risk-free parents lost considerable tissue mass when exposed to risk, and such losses may either delay the onset of reproduction or reduce the number of offspring they are able to produce. In contrast, the offspring of risk-experienced parents did not experience such tissue loss under risk and are presumably better positioned for future reproduction, suggesting the potential for parental effects to extend to their grandoffspring.

The within-generation impacts of predation risk can substantially influence prey fitness and habitat use as well as community dynamics (Schmitz et al. 2004, Trussell et al. 2006*b*, Peckarsky et al. 2008) and ecosystem-level processes such as energy transfer (Trussell et al. 2006*b*), nutrient dynamics (Schmitz et al. 2010), and diversity (Schmitz 2003). We found that parental experience with predation risk can strongly influence prey behavior and foraging with important consequences for prey energetic status. These results suggest that parental effects provide essential information to their offspring that enhances their fitness, particularly in systems where risk is chronic. Thus, the legacy of predation risk and its influence across multiple generations should be better integrated into our understanding of the role that risk plays in ecological communities.

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