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Research

Fear and lethality in snowshoe hares: the deadly effects of non-consumptive predation risk

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Predators play a critical, top–down role in shaping ecosystems, driving prey population and community dynamics. Traditionally, studies of predator-prey interactions have focused on direct effects of predators, namely the killing of prey. More recently, the non-consumptive effects of predation risk are being appreciated; e.g. the 'ecology of fear'. Prey responses to predation risk can be morphological, behavioural, and physiological, and are assumed to come at a cost to prey fitness. However, few studies have examined the relationship between predation risk and survival in wild animals. We tested the hypothesis that predation risk itself could reduce survival in wild-caught snowshoe hares. We exposed female snowshoe hares to a simulated predator (a trained dog) during gestation only, and measured adult survival and, in surviving females, their ability to successfully wean offspring. We show for the first time in a wild mammal that the risk of predation can itself be lethal. Predation risk reduced adult female survival by 30%, and had trans-generational effects, reducing offspring survival to weaning by over 85% – even though the period of risk ended at birth. As a consequence of these effects the predator-exposed group experienced a decrease in number, while the control group substantially increased. Challenges remain in determining the importance of risk-induced mortality in natural field settings; however, our findings show that non-lethal predator encounters can influence survival of both adults and offspring. Future work is needed to test these effects in free-living animals.

Introduction

Predation is a central organizing principle in ecology, and ecologists have continuously worked to understand the importance of predators (Volterra 1926, Rosenzweig and MacArthur 1963, Estes et al. 2011). Studies of predator effects on prey and community dynamics have traditionally focused on the killing of prey (Paine 1966, Taylor 1984, Sih et al. 1985). However, the non-consumptive risk of predation can also significantly influence prey traits and life history through long-lasting morphological, behavioural, and physiological changes (Lima 1998, Werner and Peacor 2003, Clinchy et al. 2013,

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Sheriff and Thaler 2014). Predation risk effects have been suggested to be as large as the direct killing of prey (Preisser et al. 2005), and as such, may play a significant role in population dynamics and trophic cascades (Peckarsky et al. 2008, Hawlena and Schmitz 2010, Sheriff et al. 2015, Suraci et al. 2016).

Risk-induced prey trait changes have been well-studied and such effects are often associated with a fitness cost (Werner and Peacor 2003, Hawlena and Schmitz 2010, Clinchy et al. 2013). For example, in wild birds and mammals, perceived risk of predation can alter stress hormone (glucocorticoid) levels and parental behaviour (e.g. nesting and incubation), leading to fewer offspring (Sheriff et al. 2009, Travers et al. 2010, Zanette et al. 2011). Although the effects of risk on reproduction are becoming apparent, there is a relative paucity of evidence to support the link between predation risk and survival in wild, adult animals. Evidence for risk effects on survival is limited to mesocosm studies of invertebrates and vertebrate larvae in aquatic systems (Schmitz 1998, Stoks 2001, Nelson et al. 2004, McCauley et al. 2011, Peacor et al. 2012). For example, McCauley et al. (2011) showed that the presence of a predator decreased the survival of dragonfly larvae *Leucorrhinia intacta* even when direct consumption of larvae was prevented. Yet, how these results can scale up to larger, wild vertebrates remains unknown.

Here, we tested the hypothesis that the risk of predation alone is sufficient to reduce survival using captive-held, wild female snowshoe hares *Lepus americanus*. Further, we investigated the effects of predation risk on the ability of females to successfully wean their young, even though the period of risk ended at birth. The direct, lethal effects of predation on snowshoe hare ecology are well-known (Krebs et al. 2001). We have also demonstrated that snowshoe hares are highly sensitive to the risk of predation, such that when predator densities are greatest snowshoe hares have the highest stress hormone levels (Boonstra et al. 1998, Sheriff et al. 2011). A previous experiment in this system demonstrated that elevated maternal stress hormone levels reduce the number of offspring born, the size and weight of those offspring, and altered their stress hormone levels (Sheriff et al. 2009, 2010). Here we add to those findings by testing for the effects of predation risk on adult survival and offspring survival to weaning using data from the same experiment. We predicted that exposure to predation risk alone will 1) reduce adult female survival, and 2) reduce the number of offspring weaned by risk-exposed females, even though risk-exposure ends at birth. This is the first test of these hypotheses in this system.

Material and methods

This study was conducted in the boreal forest near the Arctic Institute Kluane Lake Research Station in the southern Yukon, Canada (60rctiN, 138ReseW) over two breeding seasons (2006 and 2007). Snowshoe hares are synchronous, seasonal breeders, with first estrus occurring mid-April (Cary and Keith 1979, Stefan and Krebs 2001). Females give birth 36–39 days later to 3–5 precocial young/litter (Sheriff et al. 2009) that are weaned at 24–28 days of age (O'Donoghue and Bergman 1992). Snowshoe hares are subject to predation by both mammalian (e.g. lynx, coyote; O'Donoghue et al. 1997, 1998) and avian predators (e.g. great horned owls, goshawks; Doyle and Smith 2001).

Predator exposure experiment and animal housing

In order to test the effects of predator exposure on female and offspring survival, we experimentally exposed wild female snowshoe hares to a live, simulated predator (a trained dog) during gestation. Details of animal capture, housing, and predator exposure can be found in Sheriff et al. (2009). In brief, pregnant hares were live-trapped in early May of 2006 and 2007, transferred to an outdoor enclosure, and housed in individual 4 × 4 m chicken wire pens, which were separated by burlap-covered walls to prevent visual contact. Pens were designed to allow hares to show their natural response to predators, which is to freeze and/or take cover (O'Farrell 1965): suitable cover and hiding places were provided in all pens. Control pens and predator-exposure pens were separated by black, heavy-duty cloth and an open corridor (width of 4 m). Females were randomly assigned to either the predator-exposure (n = 20) or a control treatment (no exposure, n = 12). We include data on 6 hares in the predator-exposure treatment that were not included in Sheriff et al. (2009) due to mortality prior to reproduction. Hares in the predator-exposure treatment were exposed in their pens to a dog (trained not to bark, whine, chase, or contact the hares) that was brought into the pens, at haphazardly-selected times throughout the day, for 1–2 min every second day for the last 15 days of gestation. This duration and frequency was designed to mimic the likely direct exposure experienced by hares during the decline phase of the hare–predator cycle, when risk is greatest (Hodges et al. 1999). Control hares had no contact with the dog and did not alter their behaviour during predator exposures (i.e. they were observed to continue to forage). Although there was no direct control treatment (e.g. exposure to a non-predatory animal), all pens were checked thoroughly every day for 2–3 min for the birth of offspring, to provide food and water, and for adult mortality. On exposure days, the length of initial checking was minimized and the dog was introduced immediately prior to the check; thus, attempting to maintain a 2–3 min time period for total disturbance.

All pens were checked each day for adult mortality and the birth of offspring, and to provide food and water for animals. At parturition predator-exposure of that female ceased. Families were kept together in the pens for the next 28 days, after which offspring were assumed to have successfully weaned (Rongstad and Tester 1971). After the completion of the experiment, all hares were released back to the site of their capture. Females that died during the experiment were sent to a veterinarian for autopsy to ensure that death was not caused by disease; in all cases, dead females were disease-free.

Females from these groups did not differ in glucocorticoid levels at capture (Sheriff et al. 2009), so subsequent between-group variation in response to predator-exposure is not likely to be explained by initial underlying variation in stress physiology.

This research was approved by the University of British Columbia Animal Care Committee in accordance with the guidelines of the Canadian Council for Animal Care.

Statistical analysis

All statistical analyses were carried out using the statistical software, R ver. 2.1.2 (<www.r-project.org>). Means are reported with standard error. All tests were run with an alpha level of 0.05, but we infer and interpret p-values between 0.05 and 0.1 as biologically significant (Yoccoz 1991).

Female survival throughout the experimental period was recorded as a binary variable (0: did not survive, 1: survived treatment). To determine whether female survival in captivity was influenced by predator exposure, we tested the difference in proportion survival between treatment groups (predator-exposure, control) using a Fisher's exact test. One control female did not give birth during the experiment (likely due to misidentification of pregnancy status at the start of the experiment); to avoid confounding our results with any effects of difference in pregnancy status, we conducted the analysis testing differences in survival with this female included (n = 12 control females), and with this female excluded (n = 11 control females), and present the results from both.

We next analysed the effects of maternal predator exposure on offspring survival between birth and weaning. We restricted our analysis to successful females (those whose litters were carried to term and produced live offspring) This resulted in the exclusion of the same single control female that did not give birth (likely due to misidentification of pregnancy status at the start of the experiment), and predator-exposed females that had stillborn or aborted litters (n = 5) and that died before parturition (n = 6). This resulted in a sample of 11 control females and 9 predator-exposed females. The number of offspring that survived to weaning, and the total number of offspring born in the litter, were bound into a two-column variable using the 'cbind' function – this allows us to test effects of treatment on the proportion of offspring that survived to weaning while still accounting for original litter size. This proportion survival variable was set as the dependent variable in a generalized linear model, with a binomial error structure specified, using the 'lme4' package (Bates et al. 2015).

We separately tested for year effects prior to analyses as we did not expect treatment effects to differ between years. Year had no significant effect on female survival (Kruskal–Wallis $\chi^2_1 = 0.19$, $p = 0.66$), likelihood of producing a live litter (Kruskal–Wallis $\chi^2_1 = 0.11$, $p = 0.74$), or on the total number of offspring weaned in each group (Kruskal–Wallis $\chi^2_1 = 0.94$, $p = 0.33$). We subsequently removed year from our analyses.

Data deposition

Data available from the Dryad Digital Repository: <http://dx.doi.org/10.5061/dryad.c50sf> (MacLeod et al. 2017).

Results

Predator exposure (introduction of a trained dog) resulted in higher adult female mortality than in the control group, with all deaths occurring before parturition (Fisher's exact test $p = 0.061$, $n = 32$; Fig. 1). The exclusion of the single control female that did not produce a litter did not change this result (Fisher's exact test $p = 0.065$, $n = 31$). Although marginally non-significant, this was a strong effect according to Cohen's *d* (0.8, a "large" effect: Cohen 1988). Across both years, 100% of the control females survived the experiment, whereas only 70% of the 20 stress-treated females survived (62.5% in 2006 and 75% in 2007).

The proportion of offspring that survived from birth to weaning was significantly reduced when mothers were exposed to a predator during gestation ($F = 4.80$, $p = 0.04$). On average, predator-exposed females weaned 0.22 ± 0.22 offspring/ female, whereas control females weaned 1.73 ± 0.59 offspring/ female (Fig. 2).

Discussion

Regular exposure to the perceived risk of predation during gestation influenced survival of adult female snowshoe hares, and the subsequent survival of their offspring. At the end of our experiment, spanning one breeding bout, the predator-exposed group (adults + offspring) experienced a 20% decrease in numbers from 20 adult females to

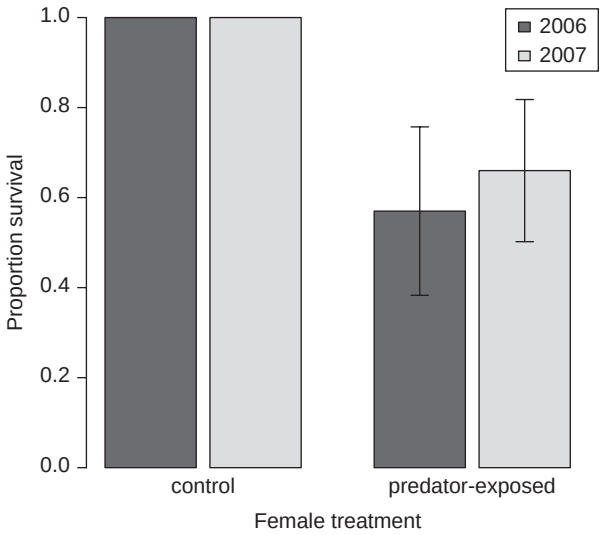

Figure 1. Proportion adult female survival (± SE) in control (n = 11 females) and predator-exposed treatment groups (n= 20 females) from 2006 and 2007. Adult female survival was significantly lower in the predator-exposed treatment.

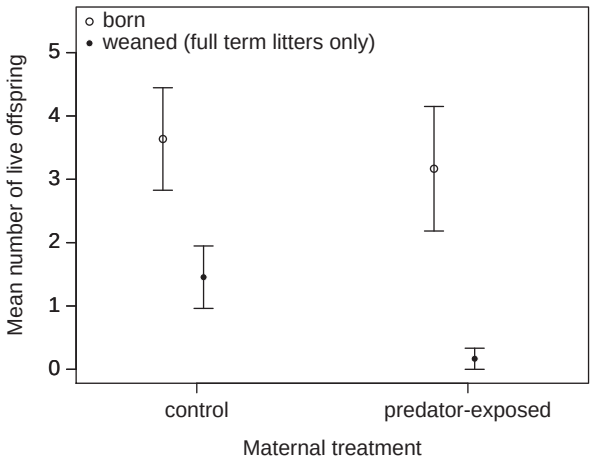

Figure 2. Number of live offspring born and weaned to surviving female hares in the control (n = 11 females) and predator-exposed (n = 9 females) treatment groups (mean ± SE). Predator-exposed females weaned fewer offspring than females in the control group.

14 adults and 2 weaned juveniles, whereas the control group had nearly a 3-fold increase from 12 adult females to 12 adults and 19 weaned juveniles (Table 1). Thus, we show, for the first time in a mammal, that predation risk increases prey mortality, causing a 70% decline in adult survival and an 87% decline in weaning success, with clear implications for group size.

We suggest that physiological changes induced by activation of the hypothalamic–pituitary–adrenal (HPA) axis, and subsequent release of glucocorticoids and its downstream effects may play a role in the risk-induced increases in mortality we found. In our experiment predator-exposed females had, on average, 214% higher faecal cortisol metabolite concentrations (indicative of levels of circulating glucocorticoids) compared to control hares (results reported in Sheriff et al. 2009). In this instance, glucocorticoid levels were increased by predator presence, but other stressors that also result in higher circulating glucocorticoids could potentially have the same effects. Past research has correlatively linked glucocorticoid physiology to survival, for example, by correlating the magnitude of the physiological response to a stressor with later survival (Blas et al. 2007, Cabezas et al. 2007, Romero and Wikelski 2010). However, the mechanistic pathways of this effect are not well known. Acute, but traumatic events may have fatal effects through HPA-mediated increases in adrenaline and downstream cardiovascular events, as has been shown with environmental stressors such as natural disasters and war in humans (Meisel et al. 1991, Leor et al. 1996).

Table 1. The effects of predator exposure on group size via maternal survival and weaning success of offspring.

Treatment group	Initial group size	Surviving females	Offspring weaned	Final group size
Predator-exposed	20	14	2	16
Control	11	11	19	30

Prolonged effects of elevated glucocorticoids may also increase the likelihood of mortality as they have been shown to: 1) reduce body mass through protein catabolism and/or reduced food intake (De Vos et al. 1995, Schmitz et al. 1997, Klein 2015, Osborne 2015); and 2) increase myopathy (Gupta and Gupta 2013). Persistently elevated glucocorticoids can also have immunosuppressing effects (Dhabhar and Mcewen 1997, French et al. 2010), leaving individuals vulnerable to disease. Although we suggest the HPA-axis and subsequent release of glucocorticoids may play a significant role in risk-induced mortality, a strict focus on glucocorticoids is likely limiting and predation risk may have multiple effects that ultimately reduce survival potential. A better understanding of the mechanisms associated with risk-induced mortality is clearly needed.

Previously, in the same experiment, we found that predator-induced maternal stress reduced the number of offspring born (i.e. mothers with greater stress hormone levels produced fewer offspring; Sheriff et al. 2009). Here, we extend those findings and show that offspring born to predator-exposed females had significantly lower survival from birth to weaning compared to offspring born to control females, even though the risk treatment stopped at birth (Fig. 2). Predation risk has been shown to reduce offspring survival from hatching to fledging in free-living song sparrows via changes in parental care, however, risk exposure was continued from nestling through the chick rearing stage in this study (Zanette et al. 2011). To our knowledge, ours is the first study in a wild mammal to find that risk experienced by mothers during gestation can have trans-generational effects reducing weaning success after the period of risk has ended. The lower survival to weaning may be due to risk-induced reductions in offspring's birth weight and birth size of offspring, and alterations in their HPA-axis, as we previously found (Sheriff et al. 2009, 2010), or due to maternal abandonment of young or reductions in lactation, as we observed offspring attempting suckling and mouthing behaviours at the time of death (Sheriff unpubl.). Reduced birth weight, which may be accompanied by a range of other cardiovascular, metabolic, and neuroendocrine disorders (Harris and Seckl 2011), has been directly linked to reduced survival in mammalian neonates (Gardner et al. 1989, Mila et al. 2015) and may therefore be a likely cause of the increased mortality we see in offspring from the predator-exposed group. Increased maternal stress can also alter post-natal maternal care and lactation (Meaney et al. 2007, Zanette et al. 2011, Hinde et al. 2015), both of which may influence offspring mortality. In order to minimise any additional stressors during the experimental period, no observational behavioural data were collected in this study, though this might elucidate the potential for maternal behaviour to play a role in increased offspring mortality. Although these factors (reduced body mass, changes in parental care) may contribute to increased offspring mortality, their weight of influence likely depends on an animal's life history. Understanding the stressor-induced changes in the factors that influence immediate offspring survival under

different contexts (life histories) may be a fruitful topic of further exploration.

Although individuals in this study were wild-caught, our experiment occurred in captivity. How our findings translate to free-living animals and the role they may play in population dynamics remains unknown. The importance of such effects will likely depend upon factors such as the influence of risk cues (e.g. scent or sound) on prey physiological responses, the natural frequency of predator–prey interactions, and the likelihood of individuals surviving predator attacks. Whether such effects are additive vs. compensatory to direct predation is also likely to be an important consideration. For example, if risk-induced mortality reduces direct killing, because predators feed on scavenged carcasses, then it may play a reduced role in population dynamics because an equal number of prey die. However, if predators maintain their level of direct predation while also increasing risk-induced mortality, such additive effects may play an important role in population dynamics. The occurrence of risk-induced mortality may also be rare in free-living systems, particularly if prey require numerous non-lethal predator encounters to induce such effects, and may be difficult to discern from direct predation, particularly if carrion is as readily consumed as a fresh kill. There is evidence of mortality in snowshoe hares not directly related to predation (Hodges et al. 2006); however, whether such mortality is caused by predation risk alone, or in combination with other factors such as food limitation, is not possible to discern from observational data. Thus, inferences about the relative importance of risk-induced mortality in free-living populations should be made cautiously.

In conclusion, we have shown that perceived predation risk alone, only during gestation, is sufficient to increase mortality in pregnant snowshoe hares, and that surviving predator-exposed females weaned fewer offspring. As a consequence, there were substantial differences in final group size between the treatments. As this is the first study in a wild vertebrate to show a risk-induced reduction in adult survival, further work is needed to better understand the mechanisms associated with such effects and in understanding the relative importance of such effects in free-living systems.

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Statement of authorship – KJM wrote the manuscript and analyzed the data; MJS collected the data; and all authors contributed to the conception of the idea, the editing and the writing.

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