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Predator-induced maternal stress and population demography in snowshoe hares: the more severe the risk, the longer the generational effect

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Abstract

The risk of predation can cause strong antipredator behaviors and marked stress-induced changes in physiology. In mothers, predator-induced stress can reduce reproductive fitness and alter offspring phenotypes. Acting via these generational, maternal stress effects, predation risk may continue to influence the demography of prey populations even when the predators are no longer present. The 10-year snowshoe hare cycle is the classic top-down predator-driven example in nature and is caused both by direct mortality and by indirect risk effects. During the decline phase, virtually, all hares die because they are killed and simultaneously hares exhibit pronounced stress effects caused by high predation risk. However, the rapidity of the decline phase varies among cycles. When the decline is extremely rapid, we expect that the risk experienced by hares is much greater than when the decline is prolonged. The enigma of these cycles is the low phase following the decline, when there is little or no population growth in spite of the absence of predators and ample food. Previously, we have shown that predator-induced maternal stress decreases reproduction and compromises the offspring stress axis. Here, we examine how the severity of predation risk during six separate population declines is related to the length of the subsequent low phase. We show that the more severe the decline, as indicated by the greater rate of loss of hares, the longer the subsequent low phase. These results support the hypothesis that the greater the degree of risk, the longer the generational impact on population demography (the longer the low phase of the hare population). Our findings have broad applicability to conservation and management efforts; even when a stressor (predator, human disturbance) is removed or when exposure may be short term (drought, fire or translocation), the signature of the stressor may be evident over several future generations.

Introduction

It is now well recognized that predators can have profound indirect effects on their prey (Schmitz, Beckerman & O'Brien, 1997; Preisser, Bolnick & Benard, 2005; Pangle, Peacor & Johannsson, 2007; Peckarsky *et al*., 2008; Clinchy, Sheriff & Zanette, 2013). Much of our understanding of the impacts of non-consumptive effects stems from work on antipredator behavior and prey trade-off decision making between predation risk and energy intake (Sih, 1987; Lima, 1998; Caro, 2005). We now know that in addition to behavioral changes, predation risk can drive changes in the physiology and morphology of prey and act not only at the level of the individual, but also have generational effects and potentially scale up to population and ecosystem level processes (Hawlena & Schmitz, 2010; Sheriff, Krebs & Boonstra, 2011; Clinchy *et al*., 2013). The risk of predation can have generational effects via maternal stress effects and continue to affect prey population processes well after the threat is gone (Love, McGowan & Sheriff, 2013; Sheriff & Love, 2013). Maternal stress effects occur when maternally derived stress hormones (i.e. glucocorticoids) have developmental and organizational effects on offspring, altering offspring phenotype (Dantzer *et al*., 2013; Love *et al*., 2013; Sheriff, 2015). Although some studies have quantified the degree to which predation risk impacts offspring via maternal stress effects (e.g. Sheriff, Krebs & Boonstra, 2010; Monclús, Tiulim & Blumstein, 2011; Bestion *et al*., 2014), these studies examined the immediate effects on a single generation. However, no study on wild animals has examined whether the severity of predation risk affects the length of its impact. If the effects last only a single generation, it is referred to as an intergenerational effect; if it

lasts more than one generation, it is referred to as a transgenerational effect. For simplicity, we refer to these effects as generational. Here, we examine whether greater predation risk results in a longer signature in populations of free-living snowshoe hares.

Snowshoe hares are an excellent species in which to examine the generational effects of predation risk for four reasons. First, both hares and their predators undergo cyclic population fluctuations every 8–10 years with predator numbers lagging 1–2 years behind hare numbers (Krebs *et al.*, 1995, 2014). During the hare population decline, almost all hares die and of these, virtually, all are killed by their predators (Boutin *et al.*, 1986; Hodges *et al.*, 2001). Second, hares are highly sensitive to increasing predation risk and this is reflected both by a marked decline in reproductive output as risk intensifies (Cary & Keith, 1979; Stefan & Krebs, 2001) and by a dramatic change in their stress physiology indicating pronounced chronic stress as risk intensifies (Boonstra *et al.*, 1998*a*, 2014; Sheriff *et al.*, 2011). Third, the majority of the breeding population of hares is made up of yearlings (Krebs *et al.*, 1995), such that carry-over effects from one year to the next are generational. Fourth, following the decline of both the hares and their predators, hare populations remain low for 2–5 years (Krebs *et al.*, 1995, 2014). The variation in the length of the low phase is an enigma that we examine here.

We hypothesize that this variation may be related to the severity of the predation risk experienced by hares during the decline. We have shown that predator-induced maternal stress is negatively correlated to hare reproductive fitness and offspring body condition and is positively correlated to offspring stress levels (Sheriff, Krebs & Boonstra, 2009; Sheriff *et al.*, 2010). Thus, if there is variation from one cycle to the next in the severity of predation risk during the decline phase, this variation will be expressed by the proportion of the population lost to direct mortality and potentially also expressed as variation in the severity of indirect effects operating through differential chronic maternal stress effects. The latter will then cause variable changes in offspring phenotype in proportion to the intensity of predation risk, which may have short- or long-term effects on fitness (reproduction and survival), resulting in short or long low phases. We test the hypothesis that the greater the risk of predation during the decline phase, the longer the subsequent low phase. As a measure of the severity of predation risk, we use the percentage reduction per year of the hare population during the decline phase (rate of decline). This severity is a consequence both of the numerical and functional response of predators (i.e. the number of predators killing hares plus their efficacy in doing so). Alternatively, the variation in the length of the low phase may simply be driven by hare density at the beginning of the low phase, with a smaller initial density prolonging recovery (i.e. a lower density at the beginning of the low phase results in a longer low phase).

Methods

Our data set consisted of six complete cycles (60 years of data), spanning the years from 1961 to 2013, in which we were able to obtain actual spring estimates of hare population change (Fig. 1). It included two cycles from Rochester, Alberta (1961–1977) (Meslow & Keith, 1968; Keith & Windberg, 1978; Keith, 1990) and four from Kluane, Yukon (1977–2013) (Krebs *et al.*, 2014). We determined phase changes based on the criteria of Keith (1990), who reviewed hare cycles across North America from 1940 to 1978 including those from Rochester and Kluane. He found that during increase years, hare

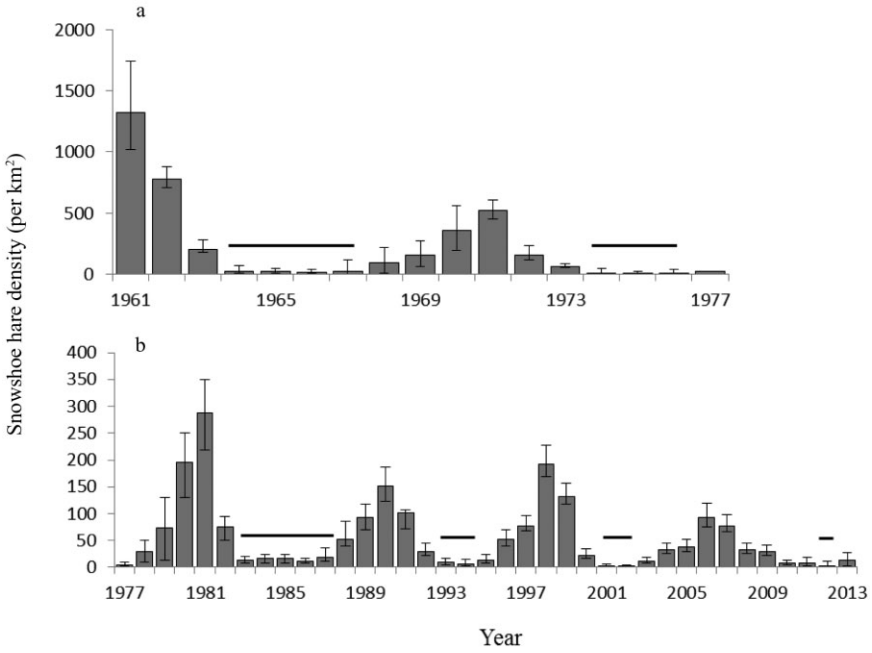

Figure 1 Changes in spring hare abundance from the peak to the end of the low phase across six hare cycles. Graph (a) is from Rochester, Alberta (1962–1977) (Meslow & Keith, 1968; Keith & Windberg, 1978) and graph (b) is from Kluane, Yukon (1977–2013) (Krebs *et al.*, 2014). Note that although the increase phase is not included for the first population cycle from Rochester, 1961 is the peak in the hare population (Meslow & Keith, 1968). The line designates the length of estimated low phase.

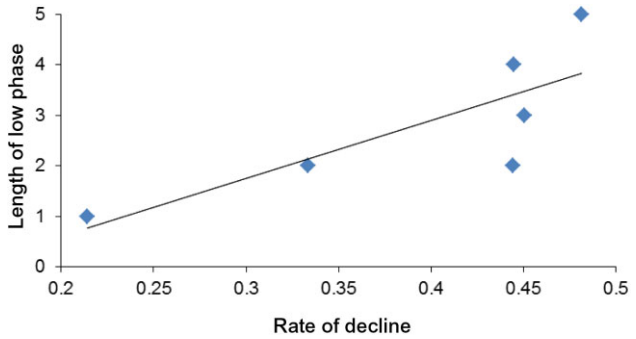

Figure 2 The length of the low phase (years) and previous rate of decline from six snowshoe hare population cycles two from Rochester, Alberta, and four from Kluane, Yukon, Canada (y = –1.7 + 11.49×).

populations averaged finite rates of population growth of 1.89 per year. In contrast, during decline years, the finite rates of population growth averaged 0.44 per year. We took these rates to estimate the length of the low phase as the number of years from the end of the decline (when rates were ≤ 0.44) to the start of the increase (when rates were ≥ 1.89 per year). Importantly, the length of the low phase was characterized not only by the low density of hares, but the constancy of low numbers. For example, at Kluane, from 1983 to 1987 spring density ranged between 12 and 18 per km^2 during the 5-year low phase (average rate of change 1.1), but between 2011 and 2013 spring density declined from 7 per km^2 (in 2011) to a single-year low phase of 3 per km^2 (in 2012; rate of decline of 0.43) and then increased to 12.5 per km^2 (in 2013; rate of increase of 4.17; Fig. 1).

We calculated the rate of decline as: rate of decline = $1–e^{(P)}$; where P equals proportion lost per year calculated as: $P = \text{Log}_e[(N_d)/(N_p)]$/length of decline (in years); where N_d equals the number of hares per km^2 in the first year of the low phase and N_p equals the number of hares at the peak. For example, at Kluane, spring hare density was 288.16 per km^2 in 1981 (peak) and 14 per km^2 in 1983 (first year of low); $P = \text{Log}_e[(14/288.16)]/2 = –0.657$; rate of decline = $1–e^{(–0.657)} = 0.48$ (Fig. 2). We used a general linear model to compare (1) the rate of decline to the length of the subsequent low phase and (2) the density of hares at the beginning of the low phase to the length of that low phase (Statistica v.10; Statsoft, Tulsa, OK, USA).

Results

The length of the low phase for these six cycles varied between 1 and 5 years, with one cycle having a 1-year low phase, two having a 2-year low phase, one having a 3-year low phase, one having a 4-year low phase and one having a 5-year low phase (Fig. 1). On average the low phase lasted 2.88 ± 0.25 (SE) years. The rate of loss per year during the decline was positively related to the length of the low phase ($r^2 = 0.63$, $P = 0.05$, Fig. 2). We did not find a relationship between the population density in the first year of the low phase and the length of that low phase ($r^2 = 0.46$, $P = 0.14$). Thus, the greater the rate of loss during the decline phase, the longer the subsequent low phase.

Discussion

Our results are consistent with our hypothesis that the more rapid the decline phase, the longer the subsequent low phase (Fig. 2). Since essentially all of the loss of hares during the decline is caused by predators, this must mean that predation risk is higher when a greater proportion of the hare population is being killed. It implies that negative consequences of greater predation risk for those hares that survive is more severe and long-lasting, prolonging the generational impact.

Alternate hypotheses

We will begin our discussion by briefly examining two alternate hypotheses to explain the variation in the length of the low phase. The first hypothesis is that the length is driven by external factors, carry-over effects of food availability and direct predation (Bryant *et al.*, 1985; Krebs *et al.*, 1995; Boonstra, Krebs & Stenseth, 1998b; Bryant, 2003). The second hypothesis is that the length of the low phase is driven by an Allee effect (Stephens, Sutherland & Freckleton, 1999; Lidicker Jr, 2010). Of critical importance is an understanding that in a single population cycle, survival is highest (and relatively constant) during the low phase (Hodges, Krebs & Sinclair, 1999) and reproduction is lowest at the beginning of the low phase but increases to a peak by the beginning of the increase phase (Cary & Keith, 1979; Stefan & Krebs, 2001). Thus, it seems that mechanisms which drive changes in reproduction are those which influence the length of the low phase.

The hypothesis that something is 'wrong' with the environment during the low phase of cyclic populations has been discussed in detail by Boonstra *et al.* (1998*b*). Briefly, there is no support that either food or predation is a major driver of the low phase or that these factors influence the length of the low phase. We deal first with the food hypothesis. The prediction related to food availability is that poor nutrition caused by either a lack of quantity or quality of food could explain the variable length of the low phase. However, virtually, all studies of food availability have found that food plants quickly recover following peak hare densities and that there is no food shortage by the beginning of the low phase (Smith *et al.*, 1988; Keith, 1990; Hodges *et al.*, 1999). Food quality also does not seem to play a role in determining the length of the low phase. Although, there is evidence that severe browsing, as occurs during the peak, can reduce overwinter nutritional value of feltleaf willows (a preferred food item) for two to three subsequent winters (Fox & Bryant, 1984; Bryant *et al.*, 1985), this severe browsing also increases nutritional value of willows during summer (Bryant, 2003). Furthermore, the impact of severe browsing is likely no longer a factor by the time the low phase begins (average decline phase lasts > 3 years). Sinclair *et al.* (1988) found that as hare populations declined, plant secondary compounds also declined and fecal

crude protein of hares (an index of dietary protein) increased. Thus, by the beginning of the low phase, there is ample, high quality food.

The predator hypothesis proposes that there are still sufficient predators in the system to keep hare numbers depressed for a number of years. There are two arguments against this. First, predators are virtually absent during the low phase, having a far lower density than during the increase phase when the hare population is growing (Krebs *et al*., 1995). Particularly in winter, only red squirrels are a viable alternative food for predators and, although these are occasionally killed, show no decline in density over time after hare numbers have collapsed and remain low (Boonstra *et al*., 2001, fig 9.2). Second, direct predation may be able to influence hare density by killing hares but it cannot directly alter the number of babies born to an individual, and, as discussed earlier, variation in the length of the low phase seems to be driven by mechanisms which influence reproduction not survival.

An alternative hypothesis is one based on the Allee effect: that is the hare density is either so low that they cannot find each other to reproduce or that some social benefit of a critical density reduces fitness – there is a positive relationship between density and individual fitness (Stephens *et al*., 1999; Lidicker Jr, 2010). The prediction would thus follow that a lower density at the beginning of the low phase would result in a longer low phase because of reduced initial individual fitness. We think that this is unlikely. First, we did not find a relationship between initial population density and length of the low phase; in fact, the longest low phase had the highest initial and subsequent density (Fig. 1, 1983–1986, Kluane, Yukon). Second, population density does not change significantly throughout the low phase but reproductive output significantly increases at the end of the low phase just prior to the population increase (Cary & Keith, 1979), thus there is no correlation between density and reproductive fitness within the low phase. Third, throughout the population cycle, individual fitness does not positively correlate to density, with the highest survival occurring during the low phase and the highest reproduction occurring at the beginning of the increase phase when densities are still low (Cary & Keith, 1979; Hodges *et al*., 1999; Stefan & Krebs, 2001).

Maternal stress and the length of the low phase

The variation in the length of the low phase could be due to inherent, long-lasting changes within the animal caused by factors acting during the decline. Previously, we have shown that hares are highly sensitive to the risk of predation (Boonstra *et al*., 1998*a*; Sheriff *et al*., 2011). When the numbers of predators were greatest (during the decline phase), hares were more stressed (as indicated by greater basal cortisol levels and a greater response to a standardized stressor, a greater ability to mobilize energy and a poorer body condition). This predator-induced maternal stress decreased reproductive fitness in hares (Sheriff *et al*., 2009). Furthermore, maternal stress was inherited by their offspring (Sheriff *et al*., 2010). We have proposed that predator-induced changes in hare stress physiology may affect their demography through its negative impact on reproduction and that the low phase of cyclic populations may be the result of predator-induced maternal stress acting through offspring fitness (Boonstra *et al*., 1998*b*; Sheriff *et al*., 2011).

Here, we argue in favor of this hypothesis and suggest that the variation in the length of low phases may be the result of differences in the severity of predator-induced maternal stress during declines. It is important to note that in our previous findings, the more severe the predator-induced maternal stress, the greater the reduction in reproductive fitness and the greater the inheritance of stress in offspring (Sheriff *et al*., 2009, 2010). Thus, during times of rapid population decline, indicating intense predation pressure, predator-induced maternal stress is much greater causing a more severe decline in reproduction and exposing offspring to much greater stress hormones *in utero*. This results in a greater and longer generational inheritance of stress; a longer 'memory of the past', likely expressed in the offspring's reproductive fitness. During times when the population decline is more gradual, predator-induced maternal stress is reduced, resulting in a less severe decline in reproduction and reduced *in utero* stress exposure for offspring. This leads to a shorter generational time that elevated stress levels persist in the population, more rapid recovery of high reproductive fitness and ultimately to a shorter low phase.

A critical understanding in the findings we present here and that of others is that risk does not simply create a two-case scenario of a stressed or unstressed mother, or of a stressed or unstressed offspring, but that the *severity of risk* is related to the *strength of response*. In terms of the generational impact, differences in predation risk create a gradient, such that the severity of risk is reflected in the degree of maternal stress, which in turn would be reflected in the degree of change in offspring phenotype (stress and behavior). This hypothesis is supported by evidence from the laboratory-based biomedical studies which demonstrate that the severity of prenatal maternal stress exposure is correlated with methylation patterns in offspring brains and has dose-dependent effects on neurophysiology and behavior of offspring (Mychasiuk *et al*., 2011; Cao-Lei *et al*., 2014). In free-living animals, we demonstrate (Fig. 2) that the greater the severity of initial risk, the longer (more generations) the consequences (greater offspring stress, antipredator behaviors and cost to reproduction) will persist in the population even after the risk has been removed.

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