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Predicting population-level risk effects of predation from the responses of individuals

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Abstract. Fear of predation produces large effects on prey population dynamics through indirect risk effects that can cause even greater impacts than direct predation mortality. As yet, there is no general theoretical framework for predicting when and how these population risk effects will arise in specific prey populations, meaning that there is often little consideration given to the key role predator risk effects can play in understanding conservation and wildlife management challenges. Here, we propose that population predator risk effects can be predicted through an extension of individual risk trade-off theory and show for the first time that this is the case in a wild vertebrate system. Specifically, we demonstrate that the timing (in specific months of the year), occurrence (at low food availability), cause (reduction in individual energy reserves), and type (starvation mortality) of a population-level predator risk effect can be successfully predicted from individual responses using a widely applicable theoretical framework (individual-based risk trade-off theory). Our results suggest that individual-based risk trade-off frameworks could allow a wide range of population-level predator risk effects to be predicted from existing ecological theory, which would enable risk effects to be more routinely integrated into consideration of population processes and in applied situations such as conservation.

Key words: bottlenose dolphin, Tursiops truncates; *harbor porpoise,* Phocoena phocoena; *indirect effects; individual-based theory; lethal porpoise–dolphin interactions; mass-dependent predation risk; nonconsumptive effects; nonlethal predator effects; sandeel,* Ammodytes marinus; *Scotland; starvation–predation risk trade-off.*

Introduction

It has traditionally been assumed that predators impact population dynamics only by causing direct mortality of the individuals that they kill. Recently, it has been shown that the indirect fear or risk effects of predators (also known as nonlethal or nonconsumptive effects) can have as great, or even greater, impacts on population regulation (Werner and Peacor 2003, Schmitz et al. 2004, Trussell et al. 2006, Creel and Christianson 2008, Heithaus et al. 2008, Zanette et al. 2011). Such predator risk effects can lead to significant changes in population dynamics, with effects cascading through communities and across different trophic levels in both terrestrial and marine ecosystems (Schmitz 2003, Schmitz et al. 2004, Creel et al. 2005, 2007, Baum et al. 2007). The key role of risk effects in ecological systems has generated considerable interest in trying to predict their effects from general principles, with habitat effects, predator hunting mode, and prey characteristics all being shown to provide some ability to predict impacts (Trussell et al. 2006, Schmitz 2008, Creel 2011, Matassa and Trussell 2011). However, there is, as yet, no general theoretical framework in population ecology for predicting when and how such risk effects will arise or how strong they will be (Creel and Christianson 2008, Creel 2011). As a result, there is often too little consideration given to the key role that predator risk effects can play in understanding conservation and wildlife management challenges (e.g., Zanette et al. 2011).

Population-level predator risk effects on prey can only arise if individuals respond to predation risk and if these responses result in individual fitness costs, so it has been suggested that understanding the functional responses of prey may provide the most direct general framework for predicting risk effects (Creel 2011). In behavioral ecology, individual-based starvation–predation risk trade-off theory provides a general framework for understanding antipredator responses at the individual level (Houston et al. 1993). This body of work predicts that in order to maximize fitness, individuals should respond to their risk of starvation and their risk of predation by adopting behaviors that will minimize their

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overall risk of mortality rather than minimizing starvation or predation risk independently (McNamara and Houston 1987, Houston et al. 1993). It has long been expected that these individual effects, when taken cumulatively across a population, should have the potential to impact population dynamics (McNamara and Houston 1987, Rogers 1987, Houston et al. 1993, Lima 1998, MacLeod et al. 2006, 2007*b*) and so help to explain how population-level predator risk effects arise. However, despite these predictions, much of this theory has never been applied to help achieve a general understanding of the role of predator risk effects in population ecology.

In this study, we therefore test for the first time whether individual-based starvation–predation theory has the ability to predict the occurrence, timing, and type of a population-level predator risk effect. Population mortality rates are, by definition, the sum of individual deaths in the population and individual deaths are dependent on the risks that each individual experiences. Based on starvation–predation risk trade-off theory, we therefore suggest that changes in population mortality rates should be related to the behavioral response of individuals, and predator risk effects should produce increases in population mortality rates due to causes other than direct predation. Specifically, risk trade-off theory suggests such increases should be linked to the interaction between starvation and predation risk at the level of the individual (McNamara and Houston 1987).

To explore these ideas, we focus on a well-developed branch of risk trade-off theory based on the concept of mass-dependent predation risk, MDPR (Lima 1986, Witter and Cuthill 1993, Bednekoff and Houston 1994, Gosler et al. 1995). This states that when body mass influences predation risk, individual animals will trade off the risk of predation and the risk of starvation in their local environment to optimize their level of energy stores (and therefore body mass) to minimize their overall risk of mortality (Brodin 2007). When predation risk is high, it is predicted that energy stores (and therefore body mass) will be reduced to improve foraging efficiency and escape response, at the cost of a poorer ability to survive periods of low food availability. In contrast, when starvation risk is high, energy stores (and body mass) will be increased to insure against starvation, but with the cost of a poorer escape performance (in terms of reduced speed, acceleration, and maneuverability) resulting in a reduced ability to escape from a predator (Krams 2002) and/or greater exposure to predation risk because of increased energy requirements linked to greater body maintenance costs (Brodin 2001, 2007). MDPR theory was originally developed to explain energy reserve and body mass dynamics in small birds (Lima 1986, Bednekoff and Houston 1994), but evidence of widespread applicability now comes from a diverse range of vertebrate organisms in both terrestrial and aquatic ecosystems, including passerine birds (Gosler et al. 1995, Gentle and Gosler 2001), larger birds (Zimmer et al. 2011), large aquatic mammals (MacLeod et al. 2007*c*), rodents (Tidhar et al. 2007), and reptiles (Perez-Tris et al. 2004). The wide taxonomic applicability of MDPR theory makes this a useful test case for developing an understanding of predator risk effects. However, it should not be assumed to apply universally, because in some large mammals, especially herbivores, energy reserve storage may be determined more by habitat use (e.g., Creel et al. 2005).

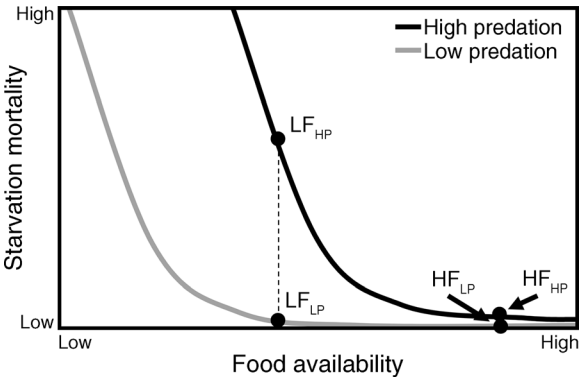

FIG. 1. Predicted population-level starvation mortality of prey under high and low predation risk conditions. Based on mass-dependent predation risk (MDPR) theory, we predict that because of strategic changes in energy reserves for any given level of food availability (high food, HF; low food, LF), starvation mortality will be higher under high predation risk (HP, black line) than under low predation risk (LP, gray line). Although this difference may be relatively small and insignificant when food availability is high (e.g., the difference between points HF_{LP} and HF_{HP}), when food availability is relatively low, this difference increases substantially (e.g., the difference between points LF_{LP} and LF_{HP}). Thus, we predict that under conditions of high predation and relatively low food availability, there will be a substantial amount of additional population mortality from starvation (indicates by the dashed line) that is not present at similar food levels when predation is low.

Overall, this body of theory implies that the risk effects of predators have the potential to cause otherwise unexpected starvation mortality within populations of their prey. Such potential starvation mortality caused by predators is not due to the presence of a predator or predators preventing prey reaching a resource (which would only impact individuals when in close proximity to a predator). Rather, we predict that starvation mortality would increase across the population because each prey individual will adopt strategic responses that reduce its predation risk (e.g., by reducing either predator attack rate or predator attack success), but at the cost of an increased starvation risk. In the case of a mass-dependent predation risk, the simple possibility that prey will be attacked by a predator results in the individuals within the prey population adopting the strategic response of reducing energy reserves (Gosler et al. 1995, Gentle and Gosler 2001). Fig. 1 outlines conceptually how such individual strategic responses would result in increased population starvation mortal-

ity through predator risk effects. Specifically, in a high-quality foraging environment where most individuals can easily meet daily energy expenditure requirements, we suggest that carrying reduced energy reserves will have little impact on mortality rates (see the points marked HF, higher food availability, in Fig. 1). However, if an environment deteriorates so that food availability is less predictable, then strategically low individual energy reserves will mean higher starvation mortality rates in the population due to the stochastic nature of foraging. Fig. 1 predicts that the increase in the probability of starvation with high predation risk depends on environmental quality (as measured by food availability) and occurs at a much higher level when environmental quality is low. The two points marked LF (lower food availability) show how starvation mortality in a population could differ substantially due to differences in predation risk (and consequent reduction in energy reserves carried), even though the underlying food availability is exactly the same.

To test these predictions, we use a model study system based around harbor porpoises *Phocoena phocoena* in Scottish coastal waters (Ross and Wilson 1996, MacLeod et al. 2007*c*). In this system, harbor porpoises suffer from a form of intra-guild predation where they are killed, but importantly not consumed, by a larger sympatric predator, the bottlenose dolphin *Tursiops truncatus* (Ross and Wilson 1996, Patterson et al. 1998). This form of predation only occurs in some areas and not others, despite the presence of bottlenose dolphins (and even some of the same individual bottlenose dolphins) in both types of area (Ross and Wilson 1996, Patterson et al. 1998, Robinson et al. 2012). Under these conditions, harbor porpoises have been found to vary their levels of energy stores in the manner predicted by mass-dependent predation risk theory and in a manner inconsistent with other potential explanations such as suboptimal foraging due to predator-induced habitat constraints or reduced foraging efficiency (MacLeod et al. 2007*c*). In addition, evidence consistent with harbor porpoises suffering an increased level of starvation when the availability of a favored food species, the sandeel (*Ammodytes* sp.), is low has been identified (MacLeod et al. 2007*a*). Finally, the abundance of sandeels is known to be negatively related to water temperatures (Arnott and Ruxton 2002). Therefore, the availability of a key food species will be influenced by changes in water temperature, which provides a foraging environment that changes between years and allows us to test our hypotheses about the impact of predators on starvation mortality. In this study we therefore investigate whether individual harbor porpoises adjust their body mass over time as predicted by MDPR theory, and whether this is linked to otherwise unexpected changes in recorded starvation mortality as environmental conditions have changed over time.

Specifically, we test the following hypotheses. Hypothesis 1, at the level of individual response, is that body mass will vary temporally in response to temporal fluctuations in predation risk (Lima 1986, Houston et al. 1993). For this hypothesis, we also consider two potential confounding effects, very low food availability (such that energy reserve constraints rather than strategic management are driving body mass variation), and the effect of fat reserves carried for insulation (a specific characteristic of our porpoise study system) (Watts et al. 1993). We predict that: (hypothesis 1a) mass will vary inversely with predation risk; (1b) lowest body mass will be found at times of the year when predation risk is highest; (1c) lowest body mass will not be found when abundance of preferred food is low (as might be predicted if body mass reserves were directly determined by food availability alone); (1d) body mass will not peak when temperatures are lowest (as might be expected if fat reserves were determined by a greater need for insulation in colder conditions). Hypothesis 2, at the level of population mortality, is that mortality rates will show additional starvation mortality of otherwise healthy individuals associated with body mass reduction due to increased predation risk (Fig. 1). We predict that: (2a) decreases in mass associated with predation risk will be sufficient to increase the individual's risk of starvation by reducing the time animals could survive without feeding; (2b) at times of high predation risk, average body mass levels within the population will be low enough to lead to increased starvation mortality relative to times of low predation risk; (2c) starvation mortality will peak when body mass response to predation risk is highest; (2d) there will be higher starvation mortality and different relationships between food availability and starvation mortality in areas of high predation risk compared to areas of low predation risk (as illustrated in Fig. 1).

Material and Methods

For this study, biological data on porpoises from Scotland, including information on cause of death, were obtained from the Cetacean Strandings Investigation Programme (Jepson et al. 2005) and were processed and checked for consistency prior to use by one of the authors (J. A. Learmonth) as part of the Bioaccumulation of Persistent Organic Pollutants in Small Cetaceans in European Waters (BIOCET) project (Pierce et al. 2008). These data included information on cause of death, which was determined during necropsies carried out by qualified and experienced strandings personnel. Data on annual variations in sandeel abundance, as measured by spawning stock biomass (SSB) were obtained from the International Council for the Exploration of the Seas (ICES 2008); data on sea temperatures were obtained from the Meteorological Office's HADiSST data set (Rayner et al. 2003). Fuller information about these data sources is provided in Appendix A.

Data analysis and modeling

Two initial analyses were conducted to investigate and control for the effects of potential confounding variables. Firstly, a generalized additive model (GAM) was used to confirm that starvation deaths were not confounded by bottlenose dolphins preferentially targeting starving individuals. This model, referred to as GAM 1, used body mass as the dependent variable, and body length and cause of death as the independent variables. Secondly, a coefficient of body mass in each month was generated in a second GAM analysis. Again body mass was the dependent variable, while body length, month, and interannual variations in food availability of a key prey species were the independent variables. Food availability was indicated by regional sandeel spawning stock biomass (SSB, the number of mature sandeels that occur within a specific area) of the main sandeel species *Ammodytes marinus*. Full details of this model are provided in Appendix B. To avoid having starving animals, or those in generally poor health at time of death, causing us to overestimate the potential for a starvation risk effect due to MDPR, only data from otherwise healthy animals that were killed by bottlenose dolphins were used in this analysis of model GAM 2. In previous work, we demonstrated that in terms of the reduced body mass response, animals killed by dolphins are representative of the data set as a whole (see Fig. 1 in MacLeod et al. 2007*c*). The coefficient of body mass from GAM 2 provides a measure of how body mass varies between months, while controlling for variability in this measure of food availability and body length, that was then used within the main analyses for this study.

Next, a model of starvation in harbor porpoises was created based on thermo-energetic processes and was quantified using data from stranded animals (see Appendix C). This model provided a detailed understanding of how long it would take a porpoise of a given size and energy reserves to starve to death if food availability were disrupted. This is the first time such a model has been created and quantified for a small cetacean, and it provides an unprecedented understanding of the starvation process. In addition, it provided physiological evidence to support the classification of starvation as an actual cause of death, based on information obtained from necropsies. This model was used to quantify changes in estimated days to starvation for harbor porpoises, based on the monthly changes in body mass (as measured by the coefficient of body mass generated from GAM 2; see Appendix B).

Finally, generalized additive modeling was used to test the hypotheses and predictions just outlined. A GAM framework was used because we suspected that many of the relationships being analyzed might have nonlinear components and it was important to make no a priori assumptions as to their exact form. These GAM models (referred to as GAMs 3–9) used either a Gaussian or a Poisson distribution, depending on the nature of the dependent variable, with cross-validation to select the most appropriate number of degrees of freedom. A knot value of 4 was used to prevent over-fitting in this cross-validation process (MacLeod 2010). Categorical variables were included as fixed factors as required. All variables under investigation were included in the model either as test variables or as control variables; therefore, no model selection was necessary. The exact data set and variables included in the GAM model to test each prediction are described in Appendix D.

Results

Between 1992 and 2005, the body mass of harbor porpoises stranded around the coast of Scotland was 32 $\pm$ 0.83 kg (mean $\pm$ SE). When the relationship between body length and body mass is controlled for (GAM 1: body length, $P < 0.001$; deviance explained (DE) = 85.2%; $n = 294$ porpoises; for full details, see Appendix D: Fig. D1), mass varies significantly with cause of death. Animals killed by bottlenose dolphins were significantly heavier than animals dying of starvation ($P < 0.001$; mean difference = 4.4 kg or 13.8% of average mass) or other causes of death, ($P < 0.001$; mean difference = 3.3 kg or 10.3% of average mass). Animals that died from starvation were, on average, 1.1 kg lighter than animals that died from other causes of death, such as disease. However, this difference was not significant ($P = 0.34$). This suggests that bottlenose dolphins are attacking otherwise healthy individuals and not preferentially targeting diseased or starving animals that are in poor condition. Therefore, in general, individuals that were preyed upon would not have been likely to die in the short term due to starvation, if they had not been killed by predators.

Individual mass responses

To test Hypothesis 1 (body mass will vary temporally in response to temporal fluctuations in predation risk), we focused on the otherwise healthy animals that had been killed by bottlenose dolphins. Body mass was highest in January and February when predation mortality was low (Fig. 2A). Then as predation mortality increased, body mass dropped rapidly so that lowest body mass was maintained in March, April, and May before rising again as predation mortality deceased (Fig. 2A). There was a highly significant negative relationship between the number of recorded deaths from bottlenose dolphin predation per month and the monthly variation in body mass (GAM 3: $P < 0.001$, deviance explained, DE = 43.3%; Fig. 2B). Therefore, as predicted by MDPR theory (Predictions 1a and 1b), temporal variations in mass are related to predation risk.

Conversely, mass did not vary in relation to food availability (GAM 2: body mass in high food availability years vs. low food availability years, $P = 0.27$; Fig. 2C; see Appendix B for full details), as might be expected if energy reserves were driven directly by food availability

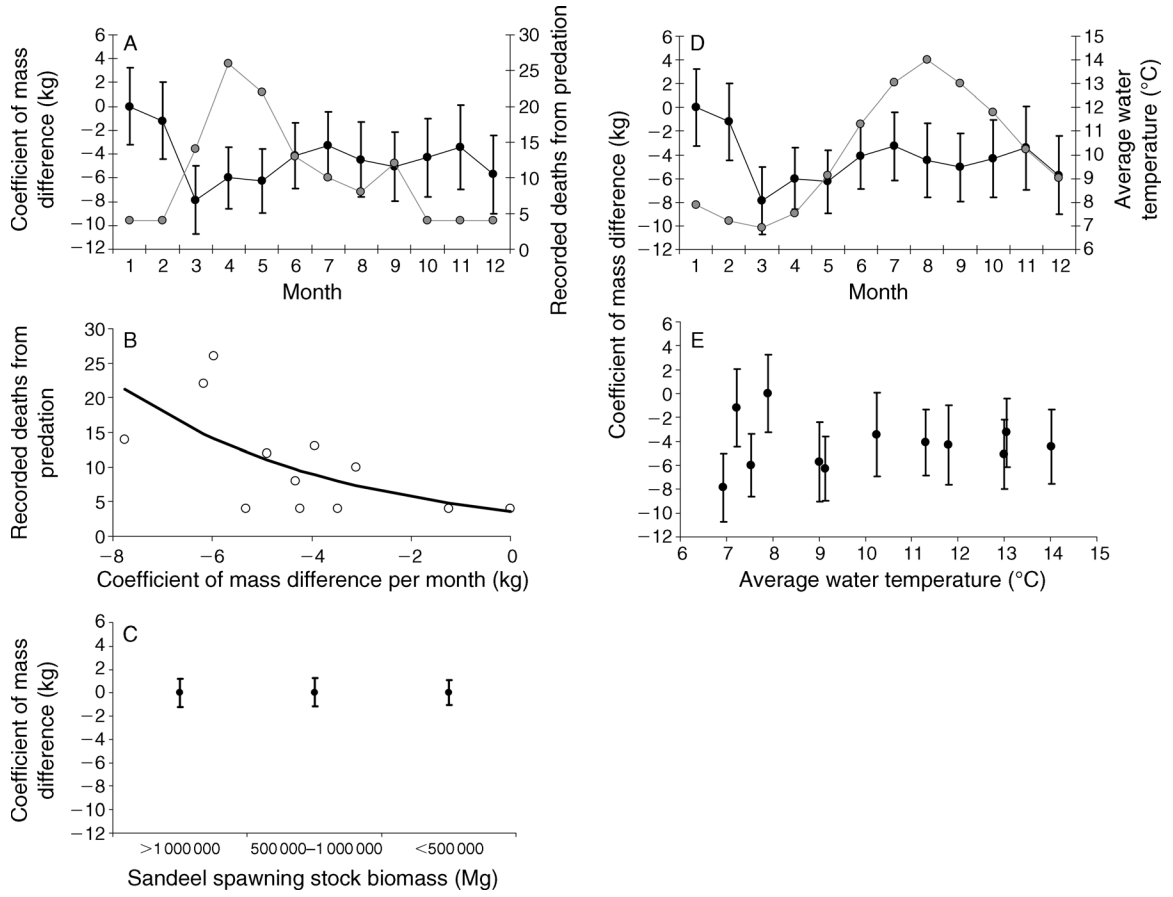

FIG. 2. Potential causes of body mass variation in the harbor porpoise (*Phocoena phocoena*). (A) Monthly changes (January is month 1) in porpoise variation in mass (black circles and line, mean ± SE of monthly coefficient of body mass from the generalized additive model GAM 2) and recorded porpoise deaths from predation (gray circles and line) by its sympatric predator, the bottlenose dolphin (*Tursiops truncatus*). (B) The relationship between monthly body mass variation and the number of recorded deaths from predation (GAM 3: $P < 0.001$, deviance explained is DE = 43.3%, $n = 12$ months). (C) Body mass in relation to annual regional abundance of sandeels, *Ammodytes marinus*, important in the porpoise diet (mean ± SE of coefficient of body mass in years with different prey abundances from GAM 2). Spawning stock biomass of sandeels was measured in metric tons; 1 ton = 1 Mg. No line was fitted because this relationship was not significant (see Appendix A for details). (D) Body mass variation (black symbols and line, mean ± SE) and average water temperature by month (gray circles and line). (E) Average water temperature and monthly body mass variation (mean ± SE). Note that abundance of sandeels is known to be negatively related to water temperature. No line was fitted because this relationship was not significant (GAM 4: $P = 0.95$, variation explained (DE) = 0.04%, $n = 12$ months).

(Prediction 1c). Finally, body mass was not highest in March when regional sea temperatures are lowest and we found no evidence of a direct relationship between temperature and monthly mass in animals killed by bottlenose dolphins (GAM 4: $P = 0.95$; DE = 0.04%, $n = 12$; Fig. 2D, E). Therefore, in the component of the population targeted by bottlenose dolphins, monthly variations in body mass were not related to water temperature and this is not consistent with changes in body mass being driven by seasonal changes in the need for insulation in these animals (Prediction 1d).

Population mortality responses

We tested Hypothesis 2 (there will be additional starvation mortality of otherwise healthy individuals associated with mass reduction due to increased predation risk) by generating a physiological model of the starvation process in harbor porpoises and how it varies with food availability, energy requirements, and ambient temperature (see *Methods* and Appendix C for details). We found that the monthly changes in body mass associated with predation are sufficient to greatly reduce the period of time that individual harbor porpoises could survive without feeding successfully (Prediction 2a; Fig. 3A). In January, when body mass was greatest, the mean survival time without feeding of an average individual in the population could be as high as 20 days, based on average water temperature in that month. In contrast, in March, when body mass was at its lowest, the reduction in energy stores would result in animals dying from starvation in less than one day without feeding successfully (Fig. 3A). Therefore, the

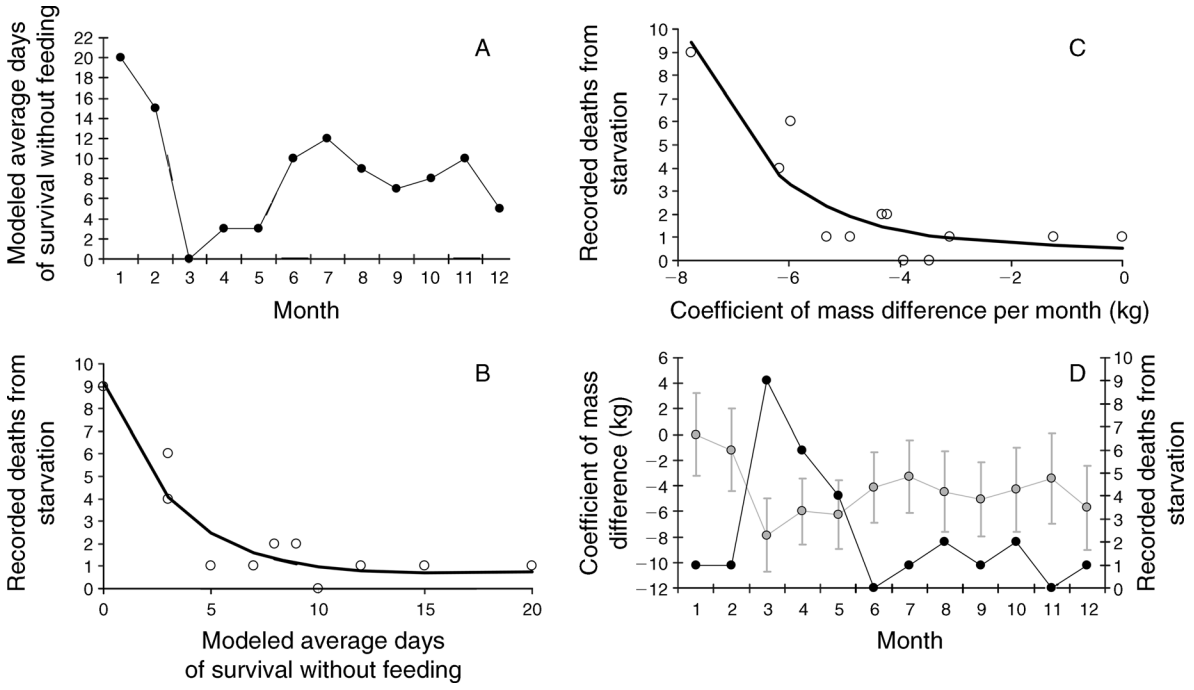

FIG. 3. Days to death from starvation, actual starvation mortality, and changes in monthly body mass of harbor porpoises. (A) Modeled days of survival without feeding for an average healthy porpoise killed by bottlenose dolphins, based on monthly mass coefficient. (B) The relationship between modeled average days of survival without feeding and the number of recorded deaths from starvation per month (GAM 5: estimated degrees of freedom, EDF = 1.75, DE = 76.2%, $P < 0.001$, $n = 12$ months). (C) The relationship between the coefficient of mass change per month and the number of animals recorded dying of starvation (GAM 6: EDF = 1.59, DE = 70.5%, $P < 0.001$, $n = 12$ months). (D) Monthly patterns of body mass variation and death by starvation (gray circles and line, mean ± SE monthly coefficient of body mass from GAM 3); black circles and line, recorded deaths from starvation.

temporal variation in body mass resulting from temporal variations in predation risk was sufficient to greatly increase the risk of starvation if food availability changes (Prediction 2a).

The number of individuals recorded as dying from starvation peaked in the same month as the body mass coefficient was lowest (Prediction 2c; Fig. 3D), and for the average level of body mass in otherwise healthy individuals in a particular month, there was a strong, nonlinear relationship between the modeled survival time without feeding and the number of animals actually recorded as having starved to death in the same month (Prediction 2b; GAM 5: estimated degrees of freedom (EDF) = 1.75, DE = 76.2%, $P < 0.0001$; Fig. 3B). When survival time was greater than ~10 days, there was no relationship between survival time and the number of animals recorded dying from starvation in a particular month (Fig. 3B). In contrast, when survival time was less than 10 days, there was a strong negative relationship between survival time and the number of recorded deaths from starvation (Fig. 3B). Additionally, when monthly body mass variation in animals killed by bottlenose dolphins was directly compared to the number of individuals recorded dying from starvation, there was also a strong, nonlinear, and significant negative relationship (GAM 6: EDF = 1.59, DE = 70.5%, $P < 0.001$; Fig. 3C). Therefore, based on the thermo-energetic model, the MDPR response to high dolphin predation is sufficient to greatly increase the risk of starvation during the peak predation period.

When we tested the final central hypothesis of the study (2d), that there will be higher starvation mortality and different relationships between food availability and starvation mortality in areas of high predation risk compared to areas of low predation risk, we found that there was a significantly higher starvation mortality in areas of high predation risk (GAM 7: predation risk, parameter estimate ± SE = 8.74 ± 4.40, $z = 1.99$, $P = 0.047$). There also were significantly different relationships between the number of recorded deaths from starvation and sandeel availability (SSB) in high- and in low-predation areas (GAM 7: nested ANOVA comparison of single relationship model vs. two relationship model, $F = 5.97$, $P = 0.007$). In both cases, the relationship was significant and negative (for high-predation areas, EDF = 1.46, $P = 0.032$; for low-predation areas, EDF = 1.00, $P = 0.018$; Fig. 4A). Overall, this model explained 80.9% of the deviance in recorded starvation mortality. In the low predation risk areas, starvation mortality only starts to occur at the

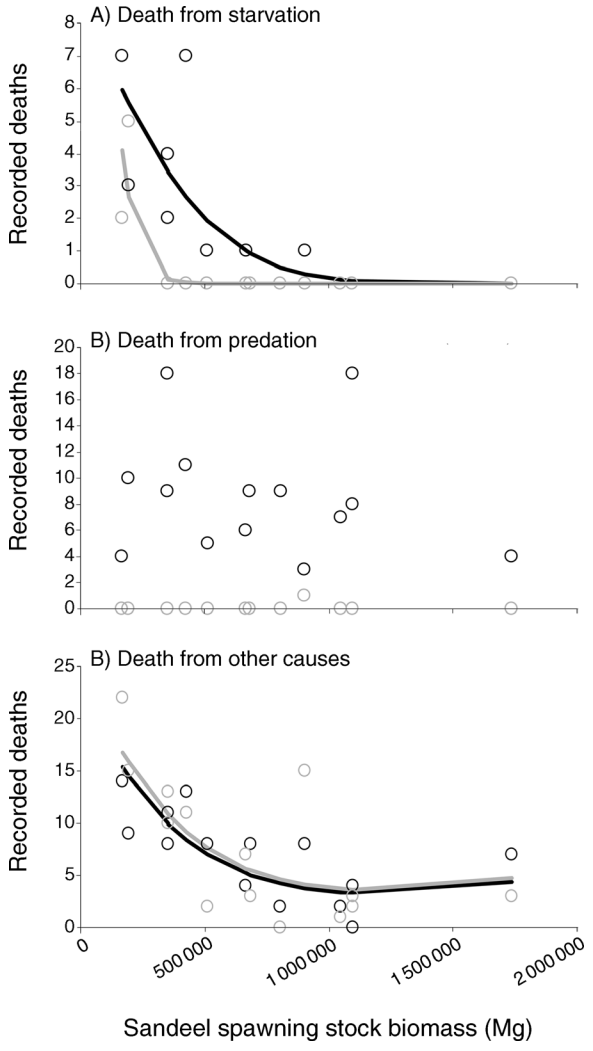

FIG. 4. Prey abundance and recorded mortality. Sandeel spawning stock biomass (measured in metric tons; 1 ton = 1 Mg) and the number of recorded harbor porpoise deaths in areas of high predation risk (black-outlined circles and lines) and low predation risk (gray-outlined circles and lines). Lines represent significant relationships; where there are no lines, relationships are not significant. Recorded deaths are shown (A) from starvation, (B) from predation, and (C) from other causes.

very lowest sandeel abundances (less than ~200 000 tons). However, the situation is very different in the high predation risk areas. Here, deaths from starvation start to occur at much higher sandeel abundances (~1 000 000 tons; Fig. 4A). These relationships show that recorded starvation mortality is, on average, higher in high-predation areas and the difference in mortality rates between high- and low-predation areas is greater at lower levels of food availability (Fig. 4A), as predicted in Fig. 1. In contrast, although there were significantly more deaths from predation in high predation risk areas (GAM 8: predation risk, parameter estimate ± SE = 4.80 ± 0.99, $t = 4.85$, $P < 0.001$), there was no relationship between recorded deaths from dolphin predation and sandeel abundance, SSB (GAM 8: for sandeels, EDF = 2.0, $P = 0.44$; Fig. 4B). Similarly, there was a strong negative relationship between the number of deaths from other causes and sandeel abundance (GAM 9: for sandeels, EDF = 2.0, $P < 0.001$). However, there was no evidence that this relationship differed significantly between high and low predation risk areas (GAM 9: for predation risk, parameter estimate = 0.088 ± 0.14, $t = 0.63$, $P = 0.53$; for ANOVA comparison of a single-relationship model vs. two-relationship model, $F = 2.51$, $P = 0.070$).

Discussion

Here we have shown for the first time that the timing (in specific months of the year), occurrence (at low food availability), cause (reduction in individual energy reserves), and type (starvation mortality) of a population-level predator risk effect can be successfully predicted from individual responses using a widely applicable theoretical framework (individual-based risk trade-off theory). Our results demonstrate that the response of individual animals to high predation risk is sufficient from a physiological stand point to increase the risk of starvation mortality in a population and is tightly linked over time to changes in actual recorded mortality from starvation. Furthermore, we show that the relationship between recorded deaths from starvation and food availability is dependent on the level of predation risk, as we predicted from behavioral risk trade-off theory (see Fig. 1). Therefore, the bottlenose dolphin–harbor porpoise–sandeel system, provides, for the first time for a wild vertebrate population, strong empirical evidence to support our general prediction that predator risk effects (indirect effects or nonconsumptive effects) can increase starvation mortality at the population level. Specifically, our thermo-energetic model suggests that in the system studied, the increase in starvation is driven by body mass change in individual animals in response to predation risk.

The potential for starvation–predation risk trade-off theory to help our understanding of population-level predator risk effects is unlikely to be unique to the dolphin–porpoise–sandeel system. Such a mechanism has been proposed to explain changes in House Sparrow populations within the UK (MacLeod et al. 2006) and differences in the conservation status and population status of passerine birds more generally (MacLeod et al. 2007*b*). Due to the ecological principles on which it is based, this phenomenon is likely to occur in many other systems. Where the dolphin–porpoise–sandeel system is exceptional is in the availability of a long-term data set that provides information on the condition of individuals at time of death, cause of death, and measures of food availability of a key prey species. Therefore, although the porpoise system is unusual in terms of the data that are available for it, there is nothing to

suggest that it is likely to be unique in terms of the understanding it provides on how individual starvation–predation responses can contribute to population dynamics.

Creel (2011) has recently proposed that understanding the functional traits of prey will provide a direct general framework for predicting predator risk effects. The approach described here was developed independently, but provides a successful test of this hypothesis. Our results not only strongly support the ability of prey traits to provide a general framework for predicting population predator risk effects, but also suggest that the way individual prey trade off their predation risk against their starvation risk is a key trait that needs to be understood. Individual starvation–predation risk trade-off theory focuses on understanding and predicting how combinations of risks from different sources can be balanced to optimize individual chances of survival (McNamara and Houston 1987, Houston et al. 1993). We therefore suggest that it has the capacity to unite all of the key factors that have so far been shown to predict strength of predator risk effects: resource availability (Belovsky et al. 2011), habitat effects (Trussell et al. 2006), landscape features (Heithaus et al. 2009), prey escape tactics (Heithaus et al. 2009), and predator hunting mode (Schmitz 2008).

Until now, the possibility that prey respond to predators by strategically accepting a higher starvation risk, and that this will result in a predator risk effect of increased starvation mortality in the population, has received relatively little attention in the burgeoning literature on population-level predator risk effects. Classic studies, for example, with snowshoe hares *Lepus americanus* (Krebs et al. 1995, Sheriff et al. 2009) and Arctic ground squirrels *Spermophilus parryii* (Karels et al. 2000), have shown that combined food limitation and predator exclusion produce more than additive population responses due to increased reproduction attributed to the removal of predator risk effects. More recent work, including the series of detailed investigations of predator risk effects of wolves (*Canis lupus*) on elk (*Cervus elaphus*) populations, has shown significant predator risk effects on behavior, habitat selection, and reproductive physiology linked to elk population declines resulting from reduced reproductive success (Creel et al. 2005, 2007, 2009, Christianson and Creel 2010). Schmitz et al. (2004: Table 1) summarized a large number of studies showing predator risk effects that result in tropic cascades. The risk effects scrutinized in each case were habitat shifts and reduced feeding, both leading to reduced growth and reproductive success. However, as yet it has rarely been considered how indirect predator risk effects might alter mortality patterns in a predictable manner because of strategic responses of the prey. On occasions when reduced survival has been considered, it has most often been attributed to "fear" or "stress" (e.g., McCauley et al. 2011) forcing the prey into a less than optimal habitat or diverting resources from the immune system or other critical processes such as metamorphosis. As McCauley et al. (2011) highlight, the extent to which predator-induced nonconsumptive mortality occurs in animals is currently largely unknown due to a lack of previous studies.

In contrast, in this study we are suggesting that, due to prey individuals trading off their starvation risk against their predation risk (Houston et al. 1993), increased starvation mortality will be a likely and perhaps unavoidable population-level predator risk effect. If predator risk effects regularly induce mortality from starvation, this has implications for the study of ecology and ecosystem functioning more generally. First, a widely held idea in ecology has been that predation mortality might be considered compensatory because predators may be killing individuals that are close to starving (Bowyer et al. 2013) and that even measured direct predator mortality may therefore overestimate the impact of predators on population dynamics. However, as we show here, if indirect predator effects are leading individuals to strategically accept a higher starvation risk in order to minimize their overall mortality risk, then it could equally well be justified that it is the starvation mortality that is compensatory. We suggest that, in reality, in the presence of nonlethal or indirect predator effects, predation and starvation mortality cannot be considered separately, so widespread ideas of compensatory or additive mortality become largely meaningless concepts. Instead, these two causes of mortality will represent different end points that may result from an individual being anywhere along a continuum of combined starvation and predation mortality risk; whether a prey individual has died of one or another cause may be largely due to chance (Cresswell 2011).

Our results also have implications for the long-running debate in population ecology on the relative importance of top-down and bottom-up forces in ecosystem function. Our findings suggest that recorded starvation mortality in a population cannot be used uncritically as evidence of bottom-up processes contributing to population dynamics. Instead starvation–predation risk trade-off theory suggests that predator risk effects mean that a proportion of population starvation mortality can be due to top-down predator risk effects. This, in turn, suggests that a greater understanding of predator risk effects at the population level may be achieved by taking into account individual strategic responses to combined predation and starvation risk rather than studying either in isolation or at the population level alone. This will be particularly important when trying to predict how populations will change in response to novel environmental conditions, such as those resulting from global climate change.

In conclusion, we have investigated one individual-level predator risk response in detail to show that existing theory used to understand how individuals

respond to predation risk can also be used to understand and predict the occurrence of a population-level predator risk response. Although we have concentrated on mass-dependent predation risk responses, we believe that there is no reason to suspect that such population-level predator risk effects could not also arise from the many other behavioral responses of individual animals to their predators, including spatial avoidance of predators, increased vigilance, use of refuges, and distance from cover (Caro 2005). Additionally, there exists an extensive literature and theory underpinning our understanding of how individuals alter reproductive investment and patterns in response to predation risk, so we suggest that population level risk effects on reproductive investment will also be predictable from individual-based risk trade-off theory. We therefore suggest that the application of frameworks based on individual behavioral theory, such as the one that we developed here, will provide a productive means of understanding and predicting the potential impacts of population-level predator risk effects (also known as indirect, nonlethal, and nonconsumptive effects) in general. Such knowledge will increase our ability to predict changes in populations as environments change, whether we are interested in understanding future ecosystem responses, in conservation of species and habitats, or in managing populations for food or sport.

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Literature Cited

Arnott, S. A., and G. D. Ruxton. 2002. Sandeel recruitment in the North Sea: demographic, climatic and trophic effects. Marine Ecology Progress Series 238:199–210.

Baum, J. K., R. A. Myers, T. D. Shepherd, S. P. Powers, and C. H. Peterson. 2007. Cascading effects of the loss of apex predatory sharks from a coastal ocean. Science 315:1846–1850.

Bednekoff, P. A., and A. I. Houston. 1994. Dynamic models of mass-dependent predation, risk-sensitive foraging, and premigratory fattening in birds. Ecology 75:1131–1140.

Belovsky, G., A. Laws, and J. Slade. 2011. Prey change behaviour with predation threat, but demographic effects vary with prey density: experiments with grasshoppers and birds. Ecology Letters 14:335–340.

Bowyer, R. T., J. Kie, D. Person, and K. Monteith. 2013. Metrics of predation: perils of predator–prey ratios. Acta Theriologica 58:329–340.

Brodin, A. 2001. Mass-dependent predation and metabolic expenditure in wintering birds: is there a trade-off between different forms of predation? Animal Behaviour 62:993–999.

Brodin, A. 2007. Theoretical models of adaptive energy management in small wintering birds. Philosophical Transactions of the Royal Society B 362:1857–1871.

Caro, T. 2005. Antipredator defenses in birds and mammals. University of Chicago Press, Chicago, Illinois, USA.

Christianson, D., and S. Creel. 2010. A nutritionally mediated risk effect of wolves on elk. Ecology 91:1184–1191.

Creel, S. 2011. Toward a predictive theory of risk effects: hypotheses for prey attributes and compensatory mortality. Ecology 92:2190–2195.

Creel, S., and D. Christianson. 2008. Relationships between direct predation and risk effects. Trends in Ecology and Evolution 23:194–201.

Creel, S., D. Christianson, S. Liley, and J. A. Winnie. 2007. Predation risk affects reproductive physiology and demography of elk. Science 315:960.

Creel, S., J. A. Winnie, and D. Christianson. 2009. Glucocorticoid stress hormones and the effect of predation risk on elk reproduction. Proceedings of the National Academy of Sciences USA 106:12388–12393.

Creel, S., J. Winnie, B. Maxwell, K. Hamlin, and M. Creel. 2005. Elk alter habitat selection as an antipredator response to wolves. Ecology 86:3387–3397.

Cresswell, W. 2011. Predation in bird populations. Journal of Ornithology 152:251–263.

Gentle, L. K., and A. G. Gosler. 2001. Fat reserves and perceived predation risk in the great tit, *Parus major*. Proceedings of the Royal Society of London Series B 268: 487–491.

Gosler, A. G., J. J. D. Greenwood, and C. Perrins. 1995. Predation risk and the cost of being fat. Nature 377:621–623.

Heithaus, M. R., A. Frid, A. J. Wirsing, and B. Worm. 2008. Predicting ecological consequences of marine top predator declines. Trends in Ecology and Evolution 23:202–210.

Heithaus, M. R., A. J. Wirsing, D. Burkholder, J. Thomson, and L. M. Dill. 2009. Towards a predictive framework for predator risk effects: the interaction of landscape features and prey escape tactics. Journal of Animal Ecology 78:556–562.

Houston, A. I., J. M. McNamara, and J. M. C. Hutchinson. 1993. General results concerning the trade-off between gaining energy and avoiding predation. Philosophical Transactions of the Royal Society B 341:375–397.

ICES. 2008. ICES ADVICE 2008: Report of the ICES Advisory Committee. International Council for the Exploration of the Sea, Copenhagen, Denmark.

Jepson, P., R. C. Sabin, C. J. H. Spurrier, P. D. J. Chimonides, R. Deaville, M. Perkins, and R. J. Law. 2005. Trends in cetacean strandings around the UK coastline and cetacean and marine turtle post-mortem investigations, 2000 to 2004 inclusive (Contract CRO 238). Department for Environment, Food and Rural Affairs, UK Cetacean Strandings Investigation Programme, Institute of Zoology, London, UK.

Karels, T. J., A. E. Byrom, R. Boonstra, and C. J. Krebs. 2000. The interactive effects of food and predators on reproduction and overwinter survival of arctic ground squirrels. Journal of Animal Ecology 69:235–247.

Krams, I. 2002. Mass-dependent take-off ability in wintering great tits (*Parus major*): comparison of top-ranked adult males and subordinate juvenile females. Behavioral Ecology and Sociobiology 51:345–349.

Krebs, C. J., S. Boutin, R. Boonstra, A. R. E. Sinclair, J. N. M. Smith, M. R. T. Dale, K. Martin, and R. Turkington. 1995. Impact of food and predation on the snowshoe hare cycle. Science 269:1112–1115.

Lima, S. L. 1986. Predation risk and unpredictable feeding conditions: determinants of body mass in birds. Ecology 67: 377–385.
Lima, S. L. 1998. Nonlethal effects in the ecology of predator–prey interactions: What are the ecological effects of anti-predator decision-making? BioScience 48:25–34.
MacLeod, C. D., M. B. A. Santos, R. J. Reid, B. E. Scott, and G. J. Pierce. 2007*a*. Linking sandeel consumption and the likelihood of starvation in harbour porpoises in the Scottish North Sea: could climate change mean more starving porpoises? Biology Letters 3:185–188.
MacLeod, R., P. Barnett, J. Clark, and W. Cresswell. 2006. Mass-dependent predation risk as a mechanism for house sparrow declines? Biology Letters 2:43–46.
MacLeod, R., J. Lind, J. Clark, and W. Cresswell. 2007*b*. Mass regulation in response to predation risk can indicate population declines. Ecology Letters 10:945–955.
MacLeod, R., C. D. MacLeod, J. A. Learmonth, P. D. Jepson, R. J. Reid, R. Deaville, and G. J. Pierce. 2007*c*. Mass-dependent predation risk and lethal dolphin–porpoise interactions. Proceedings of the Royal Society B 274:2587–2593.
Matassa, C. M., and G. C. Trussell. 2011. Landscape of fear influences the relative importance of consumptive and nonconsumptive predator effects. Ecology 92:2258–2266.
McCauley, S. J., L. Rowe, and M. J. Fortin. 2011. The deadly effects of "nonlethal" predators. Ecology 92:2043–2048.
McNamara, J. M., and A. I. Houston. 1987. Starvation and predation as factors limiting population size. Ecology 68: 1515–1519.
Patterson, I. A. P., R. J. Reid, B. Wilson, K. Grellier, H. M. Ross, and P. M. Thompson. 1998. Evidence for infanticide in bottlenose dolphins: An explanation for violent interactions with harbour porpoises? Proceedings of the Royal Society B 265:1167–1170.
Perez-Tris, J., J. A. Diaz, and J. L. Telleria. 2004. Loss of body mass under predation risk: cost of antipredatory behaviour or adaptive fit-for-escape? Animal Behaviour 67:511–521.
Pierce, G. J., et al. 2008. Bioaccumulation of persistent organic pollutants in female common dolphins (*Delphinus delphis*) and harbour porpoises (*Phocoena phocoena*) from western European seas: Geographical trends, causal factors and effects on reproduction and mortality. Environmental Pollution 153:401–415.
Rayner, N. A., D. E. Parker, E. B. Horton, C. K. Folland, L. V. Alexander, D. P. Rowell, E. C. Kent, and A. Kaplan. 2003. Global analyses of sea surface temperature, sea ice, and night marine air temperature since the late nineteenth century. Journal of Geophysical Research (Atmospheres) 108(14): 4407.
Robinson, K. P., et al. 2012. Discrete or not so discrete: Long-distance movements by coastal bottlenose dolphins in UK and Irish waters. Journal of Cetacean Research and Management 12:365–371.
Rogers, C. M. 1987. Predation risk and fasting capacity: do wintering birds maintain optimal body mass? Ecology 68: 1051–1061.
Ross, H. M., and B. Wilson. 1996. Violent interactions between bottlenose dolphins and harbour porpoises. Proceedings of the Royal Society B 263:283–286.
Schmitz, O. J. 2003. Top predator control of plant biodiversity and productivity in an old-field ecosystem. Ecology Letters 6: 156–163.
Schmitz, O. J. 2008. Effects of predator hunting mode on grassland ecosystem function. Science 319:952–954.
Schmitz, O. J., V. Krivan, and O. Ovadia. 2004. Trophic cascades: the primacy of trait-mediated indirect interactions. Ecology Letters 7:153–163.
Sheriff, M. J., C. J. Krebs, and R. Boonstra. 2009. The sensitive hare: sublethal effects of predator stress on reproduction in snowshoe hares. Journal of Animal Ecology 78:1249–1258.
Tidhar, W. L., F. Bonier, and J. R. Speakman. 2007. Sex- and concentration-dependent effects of predator feces on seasonal regulation of body mass in the bank vole *Clethrionomys glareolus*. Hormones and Behavior 52:436–444.
Trussell, G. C., P. J. Ewanchuk, and C. M. Matassa. 2006. Habitat effects on the relative importance of trait- and density-mediated indirect interactions. Ecology Letters 9: 1245–1252.
Watts, P., S. Hansen, and D. M. Lavigne. 1993. Models of heat loss by marine mammals: thermoregulation below the zone of irrelevance. Journal of Theoretical Biology 163:505–525.
Werner, E. E., and S. D. Peacor. 2003. A review of trait-mediated indirect interactions in ecological communities. Ecology 84:1083–1100.
Witter, M. S., and I. C. Cuthill. 1993. The ecological costs of avian fat storage. Philosophical Transactions of the Royal Society B 340:73–92.
Zanette, L. Y., A. F. White, M. C. Allen, and M. Clinchy. 2011. Perceived predation risk reduces the number of offspring songbirds produce per year. Science 334:1398–1401.
Zimmer, C., M. Boos, N. Poulin, A. Gosler, O. Petit, and J. P. Robin. 2011. Evidence of the trade-off between starvation and predation risks in ducks. PLoS ONE 6(7):e22352.

Supplemental Material

Appendix A

Data sources, selection, processing, and other considerations (*Ecological Archives* E095-175-A1).

Appendix B

Analysis of monthly variation in body mass in harbor porpoises that died due to bottlenose dolphin predation (*Ecological Archives* E095-175-A2).

Appendix C

A thermo-energetic model of the starvation process in the harbor porpoise (*Ecological Archives* E095-175-A3).

Appendix D

Detailed descriptions of individual analyses used for testing specific hypotheses and predictions (*Ecological Archives* E095-175-A4).