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Relationships between direct predation and risk effects

Scott Creel and David Christianson

Department of Ecology, 310 Lewis Hall, Montana State University, Bozeman, MT 59717, USA

Risk effects arise when prey alter their behavior in response to predators, and these responses carry costs. Empirical studies have found that risk effects can be large. Nonetheless, studies of predation in vertebrate conservation and management usually consider only direct predation. Given the ubiquity and strength of behavioral responses to predators by vertebrate prey, it is not safe to assume that risk effects on dynamics can be ignored. Risk effects can be larger than direct effects. Risk effects can exist even when the direct rate of predation is zero. Risk effects and direct effects do not necessarily change in parallel. When risk effects reduce reproduction rather than survival, they are easily mistaken for limitation by food supply.

Predation risk effects are common and can be strong, but are rarely considered in conservation or wildlife management

Predators affect prey demography through direct predation and through the costs of antipredator behavioral responses, or risk effects [1,2] (Figure 1). Responses to risk can be morphological [3] or behavioral [4], including changes in habitat use [5–7], vigilance [8,9], foraging [10,11], aggregation [12,13], movement patterns [14–16] and sensitivity to environmental conditions [17]. The costs of these responses can be manifest by reduced survival, growth or reproduction [1,18–20] (Figure 2). Recent empirical research has shown that risk effects on prey dynamics can be as large as direct effects, or even larger [15,18,21,22]. Although such research has established that risk effects can be an important part of the total effect of predation, many analyses of predation as a limiting factor still consider only direct effects, particularly in studies focusing on conservation and management, and particularly in studies of vertebrates (e.g. [23–25]). When risk effects are considered, many analyses focus on effects that cascade from the prey to other species [26–29], rather than effects on the prey species itself [2] (Figure 3). Perhaps risk effects are often ignored in this context (risk effects are well studied in other contexts) simply because there is no general theory for the expected relative magnitudes of direct and risk effects, or for the expected relationship between the two.

Here we use a simple conceptual model to examine two questions about the expected relationship between direct predation and risk effects. First, do direct effects or risk effects comprise the larger portion of the total effect of

predation? Second, how are direct effects and risk effects correlated? If risk effects are small and positively related to direct effects, then measurements of direct predation rates should provide a good estimate of the total effect of predators on prey. This is the assumption implicit in many studies of predation in natural systems. However, if risk effects are large and negatively related to direct effects, then measurements of direct predation could be unrelated or negatively related to the total effect of predation, a hypothesis that remains largely untested. Without considering this possibility, it would be easy to mistake risk effects for bottom-up limitation, particularly when risk reduces the reproductive rate, rather than increasing mortality [1,2,19]. For example, analyses of snowshoe hare dynamics originally suggested that predation affects survival, but reproduction is controlled by density-dependent access to food. When Boonstra *et al.* [2] extended prior analysis to consider risk effects, they found that ‘predation risk, not high hare density or poor nutritional condition, accounted for...the marked deterioration of reproduction during the decline phase’ (p. 371).

Are direct effects or risk effects expected to comprise the larger portion of the total effect of predation?

Empirical studies have recently shown that the risk effects of predation can be large, sometimes substantially larger than direct effects. For example, Pangle *et al.* [18] estimated direct effects and risk effects of predatory spiny water fleas (*Bythotrephes longimanus*) on three species of zooplankton in Lake Michigan and Lake Erie, and found that over six combinations of location and depth, risk effects on population growth rates were more than seven times larger than the effect of direct predation. Nelson *et al.* [21] amputated the mouthparts of predatory damselfly nymphs (*Nabis* spp) so they could not kill their prey (pea aphids, *Acyrtosiphon pisum*). Comparing treatments with manipulated and normal predators, they found that risk effects accounted for 39%–80% of the total impact of predation on prey population growth. Schmitz *et al.* [15] used this approach with predatory spiders (*Pisurina mira*) and grasshopper prey (*Melanoplus femurrubrum*), and found that ‘risk spiders’ caused the same level of grasshopper mortality as normal, lethal spiders.

Despite such empirical studies (and many others [22]), we have no general theory to predict whether these should be common results. We suggest that the logic by which the principle of allocation is applied to life-history trade-offs can also be applied to this question. In life-history theory, reproductive value is the sum of current fecundity and

Corresponding author: Creel, S. (screel@montana.edu).

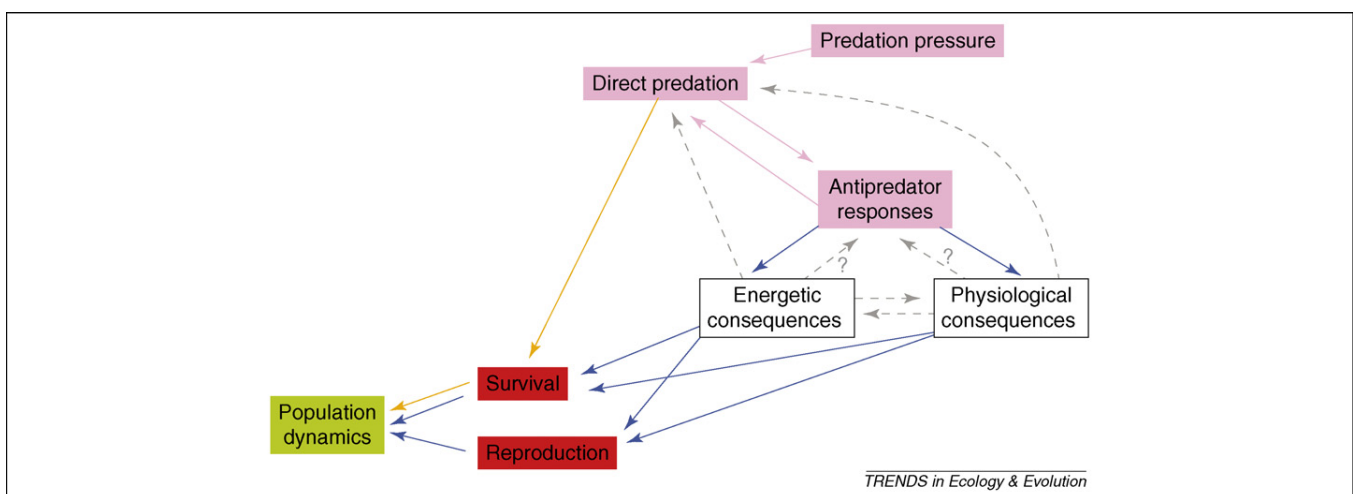


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Figure 1. To understand the total effect of predation on prey populations, we must understand the relative importance of direct predation (a) and risk effects, which are the costs of antipredator behaviors that reduce direct predation but carry fitness costs, such as vigilance and retreat to safe habitats (b).

residual reproductive value, and the shape of the curve relating residual reproductive value to current reproductive effort determines the optimal trade-off between a fitness benefit and its opportunity cost. Similar logic can be used to examine trade-offs between direct predation and risk effects. Figure 4 shows several ways that direct and risk effects might be related. Generally, as the effort invested in antipredator behavior increases, risk effects will increase and direct predation will decline. If an increase in antipredator behavior is equally effective at reducing direct predation for any level of antipredator response, then the trade-off curve would be linear. Evolutionary logic suggests that the slope of a linear trade-off

should be -1 . Positive slopes would quickly be eliminated by selection, because they represent a situation in which antipredator behavior *increases* the rate of direct predation. Behavioral responses that yielded a linear fitness trade-off with any negative slope other than -1 would also be eliminated by selection, because selection would favor individuals who decreased direct effects to zero (for slopes steeper than -1) or decreased risk effects to zero (for slopes shallower than -1), reducing such cases to a single point on one of the axes. The case of pure direct costs might not be surprising, but the case of pure risk effects raises the important point that predation might affect prey fitness (and thus dynamics) even in the absence of direct killing.



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Figure 2. Pathways by which predation can affect the population dynamics of prey: the restricted view that predation operates only through direct offtake is illustrated by two orange arrows linking predation rates to prey dynamics by way of survival; this is only the tip of the iceberg. Pink boxes and arrows illustrate that direct predation rates are not simply an external ecological force imposed on prey by predators, but are determined jointly by the intensity of predation pressure and the antipredator response of prey to that pressure. This creates a feedback loop between direct predation and antipredator responses. In turn, antipredator responses can have physiological or energetic costs for prey, which could alter prey dynamics via either reproduction or survival. These risk effects are illustrated by blue arrows, with likely feedbacks illustrated by grey arrows.

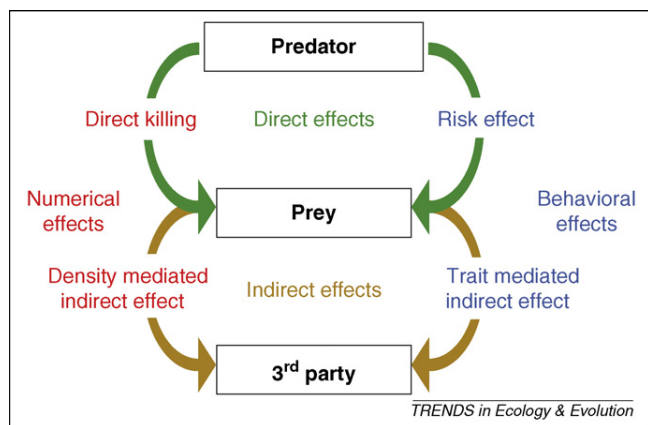


Figure 3. A classification of predation effects into four basic types using two dichotomies: numerical effects of predation operate through changes in prey numbers. Numerical effects are well studied, both as direct killing and as density-mediated indirect effects. Indirect effects are traditionally defined as effects that cascade from the predator to the prey and on to a third species. Although studies of behavioral responses to predation risk are common, the effects of risk on population and community dynamics have received much less attention. Trait-mediated indirect effects have (surprisingly) received more attention than risk effects. Risk effects are often called ‘sublethal’ or ‘nonlethal’ effects, although they can be lethal. Risk effects are also called ‘fear effects,’ although few studies actually test the hypothesis that fear drives antipredator responses. More care is warranted with the connotations of these terms.

We will return to this point. For the case of linear trade-offs with slope -1 , any level of antipredator behavior yields the same total effect of predation. Both risk effects and direct effects are expected, but we cannot predict their relative magnitudes.

The curvature of fitness cost-benefit curves has been shown to be an important determinant of the way that prey should optimize trade-offs between predation risk and starvation risk [30], and curvature is also important here (for similar reasons). For example, it is possible that antipredator responses are initially effective at reducing direct predation, but then yield diminishing returns with increasing effort, as might be the case for antipredator vigilance [31]. In this case, the trade-off curve for direct predation and risk effects would be concave-up (Figure 4, green curve): consequently, both direct and risk effects are expected. It is also possible that antipredator behavior is initially ineffective at reducing direct predation, but antipredator responses become more effective as the level of response increases, as could be the case for antipredator changes in patterns of grouping [32], particularly when prey disaggregate into smaller groups to avoid detection [11,33]. In this case, the trade-off curve would be concave-down (Figure 4, red and blue curves): consequently, the total cost of predation is expected to be either completely direct (Figure 4, red curve) or completely indirect (Figure 4, blue curve). Concave-down trade-off curves with different shapes show that prey species with very different direct predation rates might experience equal total effects of predation (even if one species has a direct predation rate of zero).

To summarize, the manner in which antipredator behavior reduces direct predation should determine the shape of the trade-off curve for direct and risk effects, which helps to predict the expected magnitudes of the effects. Many ecological studies implicitly assume that when direct predation rates are low, the total effect of predation is neces-

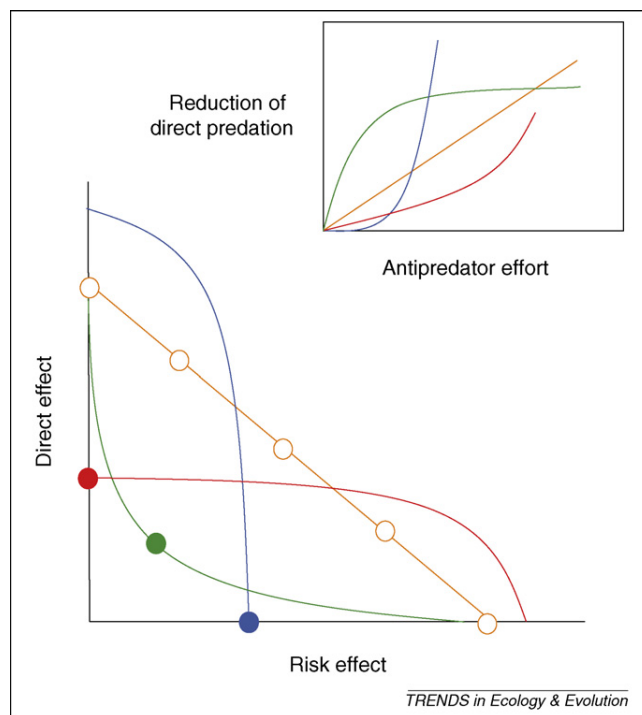


Figure 4. The relative magnitudes of fitness costs manifest as direct predation or as risk effects: one can imagine several reasonable relationships between the demographic cost of direct predation (direct effect on fitness) and the demographic cost of avoiding predation (risk effect on fitness). The inset figure shows the effectiveness with which antipredator effort reduces direct predation, for each of the fitness trade-off curves in the main figure (matched by color). For these trade-off curves, natural selection will favor strategies that minimize the total cost of predation, which is the sum of the fitness costs of direct predation and risk effects. Solid points identify the strategy that minimizes the total cost of predation for trade-off curves with asymptotic benefits of antipredator behavior (green) or accelerating benefits of antipredator behavior (red and blue). Open points identify multiple strategies that yield equal total costs of predation, if antipredator behavior has constant benefits, which yields a linear trade-off curve with slope -1 (orange). For these trade-off curves, the costs of direct predation and risk effects must be measured in the same units, ideally total fitness. In practice, both fitness costs will usually be measured by changes in demographic components of fitness such as rates of survival or reproduction. Note that risk effects can comprise from 0% to 100% of the total effect of predators on prey.

arily weak, but this assumption is questionable, because risk effects can logically be larger than direct effects. McNamara and Houston [30] used a simple model of trade-offs between starvation and predation to reach a similar conclusion, that “the number of animals that die from starvation may not provide a reliable indication of the importance of food” (p. 1515).

How are direct effects and risk effects expected to be correlated?

Ecologists concerned with identifying which factors most strongly limit a population (for example by comparing the effects of food, predators and weather) often measure the direct rate of predation and ignore risk effects when assessing the impact of predators on prey. This traditional approach implicitly assumes that direct effects are larger than risk effects, or that risk effects (typically unmeasured) correlate positively with direct effects. Empirical studies and the conceptual model above suggest that the first assumption is often not safe. Is it any safer to assume that risk effects are always positively correlated with direct effects?

Opinion

If the level of attempted predation on a prey species is driven mainly by the decisions of the predator, and not by the prey, then direct effects and risk effects could be positively related (Figure 5a). This is a logical expectation for comparisons on an ecological timescale, over which there are no evolutionary changes in the shape of the trade-off curve relating risk effects to direct predation. For example, consider a comparison among several populations of a single prey species that are exposed to different densities of their primary predator. A high rate of attempted predation would favor strong expression of antipredator responses, in comparison to a population with a low rate of attack, and this would produce a positive association between direct predation and risk effects (Figure 5a).

This argument, however, assumes that the antipredator behavior does not reduce the rate of predation. Feedback between antipredator behavior and the rate of predation will tend to eliminate or reverse the positive relationship between direct predation and risk effects. To illustrate this point, consider an example in which a predator attacks prey species *A* and *B* equally often, and both prey have the same level of antipredator response. If *A* increases its level of antipredator behavior effectively, and predation pressure consequently shifts to *B*, then direct effects and risk effects would become negatively correlated. This scenario seems plausible, particularly for situations in which a predator chooses from a set of morphologically similar prey of comparable sizes. As Figure 5b illustrates, evolutionary changes in the trade-off between direct predation and risk effects (as tested by comparisons between species, or between the sexes within a species) should also produce negative correlations. To summarize, predation pressure mediated only by the predator should produce a positive relationship between direct and risk effects, whereas predation pressure that is modified by prey responses should reduce this correlation or produce negatively correlated effects.

In summary, risk effects can be large in comparison to the effect of direct predation, and the correlation between risk effects and direct predation need not be positive. If risk effects are not explicitly considered, they could easily be mistaken for limitation by food supply, particularly in observational studies and particularly when they are manifested as reductions in reproduction (as shown in snowshoe hares, arctic ground squirrels, elk, bluegill sunfish and several invertebrates [1,2,19,34,35]). Logically, if one does not consider risk effects, then predation can be expected to affect prey dynamics only through survival, so decreased reproduction will probably be attributed to another limiting factor, such as food supply.

Given these possibilities, measurements of the total effect of predation on prey dynamics should consider risk effects (Box 1). Why have risk effects been ignored in most studies that relate the impacts of predation to other limiting factors? First, there are the common assumptions that risk effects must be smaller than direct effects and positively related to them. Second, risk effects are the product of hundreds or thousands of small actions, with individually small effects on fitness that eventually add up, whereas direct predation is a dramatic event with

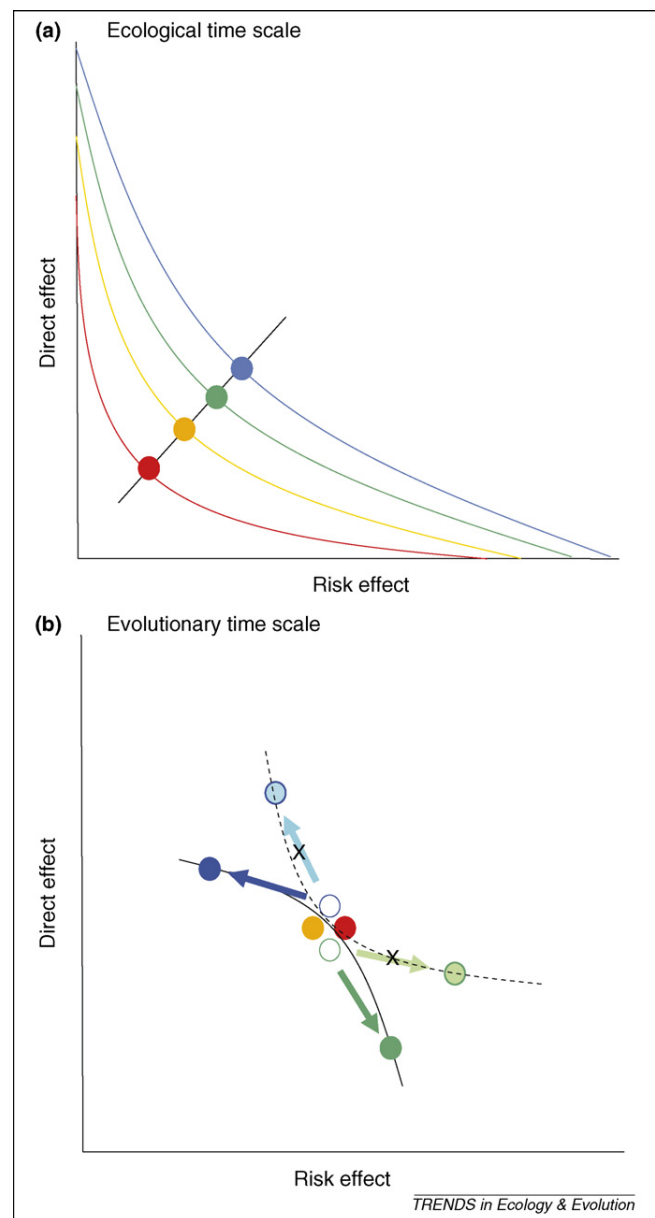


Figure 5. Possible correlations between risk effects and direct effects of predation: **(a)** considers the likely relationship between risk effects and direct predation rates on an ecological timescale, over which there are no evolutionary changes in the shape of the trade-off curve relating risk effects to direct predation (see Figure 1). This logic would apply, for example, to a comparison of multiple populations of a single prey species that faced varying levels of predation pressure from a given predator. As direct predation pressure increases (from the red curve to the blue curve), the optimal balance of risk and predation effects (identified by solid points) causes risk effects to also increase. Consequently, a positive correlation between direct predation and risk effects (black line) is expected. **(b)** considers the likely relationship between risk effects and direct predation rates over evolutionary timescales. The four central points represent four ancestral prey species with similar antipredator behavior and morphology, with similar direct predation rates and risk effects. A behavior arises in one species (open green point) that allows a large reduction in direct predation with little increase in the risk effect (dark green arrow to solid green point). A different behavior arises in another species (open blue point) that allows a large reduction in antipredator effort with little increase in direct predation (dark blue arrow to solid blue point). Both of these behaviors reduce the total cost of predation and would consequently be favored by natural selection. The result is a negative correlation between risk effects and direct predation rates, across the set of four prey species. The lighter blue and green arrows/points indicate new behaviors that would increase the total cost of predation, and thus would not be favored by natural selection. Comparing the favored and disfavored possibilities suggests that negative correlations between risk effects and direct predation (for evolutionary comparisons across species) can have concave-down shapes.

Box 1. Risk effects and predator–prey dynamics

To illustrate the potential effects of antipredator behavior on predator–prey dynamics, consider an extension of the Lotka–Volterra equations [39]:

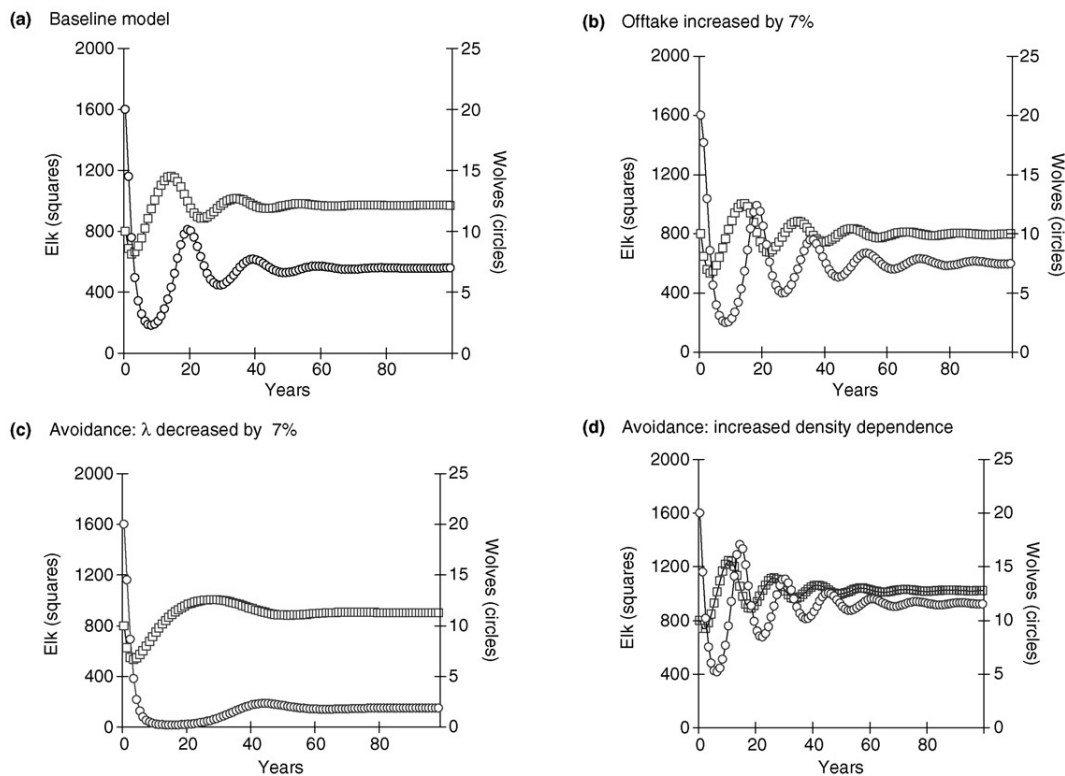
$$N_{t+1} = \frac{N_t \lambda}{1 + aN_t^b} - eP_t N_t \quad P_{t+1} = \frac{feP_t N_t - dP_t}{1 + a'P_t^{b'}}$$

Here, prey numbers (N_t) are controlled by their finite rate of increase (λ), two parameters that describe density dependence (a, b), and by direct predation ($eP_t N_t$). Direct predation depends on the numbers of predators (P_t) and prey (N_t), which determine the likelihood that an encounter will occur, and the efficiency (e) with which predators make a kill upon encountering prey. Predator (P_t) population dynamics are also density dependent (with parameters a' and b'), but the finite rate of increase is decomposed into a death rate in the absence of prey (d) and a birth rate that depends on two variables: the efficiency with which predators kill prey (e) and a factor (f) that converts each prey item killed into a demographic impact on the predator's growth rate (f is large for prey of high net energetic value). In Figure 1, this model is applied with parameter values that mimic elk and wolf dynamics in the Gallatin Canyon population of the Yellowstone ecosystem [6,11].

In most discussions of predator–prey dynamics, the impact of predation is evaluated by direct offtake (in this model, ePN) [23,40–43]. The basic results of these models are well known: as direct offtake increases (via e), a tendency toward a joint equilibrium is replaced by damped, stable and expanding cycles (compare Figures 1a and 1b). However, more novel results emerge if we examine risk effects, or population dynamics that incorporate explicit costs of antipredator behavior. To isolate risk effects in a simple way, consider a scenario in which predation pressure increases but offtake does not increase, because prey respond with an increase in antipredator behavior (vigilance [11], aggregation [33] or redistribution [6,16]). As the level of antipredator behavior increases, we assume that it carries increasing costs. Intuitively, the simplest manner to incorporate these

costs is by decreasing the rate of prey population growth (λ), independent of prey density (Figure 1c). This approach assumes that energy is limiting and that antipredator responses require an increase in energy expenditure and/or reduced foraging efficiency. At this stage of the argument, the effect on λ could be manifest through survival or fecundity. The critical point is this: when antipredator behavior causes a reduction in λ , population dynamics become more stable (Figure 1c versus 1a). This is in sharp contrast to an increase in direct predation, which is destabilizing (Figure 1b versus 1a). The picture is complicated by considering that another effect of antipredator behavior might be to increase the strength of density dependence in the prey population, rather than simply decreasing λ in a density-independent manner. Stronger density dependence could arise if prey concentrate in low-risk habitats or aggregate in larger herds. Figure 1d differs from Figure 1a through a 25% change in the value of b , which shifts from under-compensating to compensating density dependence in the absence of predation. In this case, an increase in the cost of predator avoidance destabilizes the dynamics (Figure 1d versus Figure 1a or 1c).

These simple (but plausible) scenarios show that the costs of antipredator behavior can have complex effects on predator–prey dynamics (also shown by other models [26,44–46]). Although the parameter values above were tailored to elk and wolves on a given study site (Box 2), we do not imply that this model realistically describes wolf–elk dynamics. The model is a tool to highlight the importance of measuring demographic risk effects, and ultimately incorporating these costs into models of predator–prey dynamics [26], which has not previously been considered in the models used to evaluate the quantitative effects of wolves on elk dynamics [23,24,47,25]. This is fundamentally an empirical issue, because the optimal responses by elk depend on the costs and effectiveness of antipredator behavior, processes that we cannot expect to predict by logic alone. Theory does not cleanly reveal the balance between predation risk and the cost of antipredator behavior for a given species.



TRENDS in Ecology & Evolution

Figure 1. Predator–prey dynamics with variation in direct offtake and risk effects. Adding risk effects to a simple model of predator–prey dynamics does not have the same effect as increasing direct offtake, and different types of risk effects alter dynamics in different ways.

immediately obvious effects on fitness. Finally, even once one has established a research focus on risk effects, it is very difficult to isolate and measure the impact of behavior on demography and dynamics, because behavior and demography change on very different timescales. (Of course, the direct predation rate can itself be hard to measure in practice, although the challenges are conceptually simpler. Radiotracking and direct observation of predators or prey can yield good estimates of direct predation rates [35–37]. Most other approaches do not allow estimation of the direct predation rate, and consequently aggregate direct predation and risk effects into a total effect of predation, without distinguishing between the two mechanisms.)

Measuring risk effects

Risk effects on prey dynamics have been quantified in several ways. To date, most research has used experimental approaches. One of the most productive lines of research has employed experimental studies with manipulated predators. Peckarsky *et al.* [1], Schmitz *et al.* [15] and Nelson *et al.* [21] conducted very revealing experiments by gluing together the mouthparts of invertebrate predators, and comparing the impacts of lethal and 'risk-only' predators on prey demography [1], density [15] and population dynamics [21]. These seminal experiments have played a central role in identifying risk effects, and in demonstrating that risk can account for a large proportion of the total impact of predators on prey [22]. Still, experiments in captivity with manipulated predators leave room to wonder how the results generalize to natural conditions.

Large-scale field experiments with snowshoe hares (*Lepus americanus*) [2,38] and arctic ground squirrels (*Spermophilus parryii plesius*) [35,36] have combined food supplementation and predator exclosures (as in Werner *et al.*'s [34] classic experiments on fish) to show that when combined, food limitation and predation had more-than-additive effects on population density. Exclosure of predators increased the density of both hares [2] and squirrels [35], largely by increasing reproduction (a risk effect). The more-than-additive effects of predation and food limitation is also consistent with risk effects, because a given level of anti-predator response is likely to carry larger demographic costs when animals are in poor condition (and see Figure 5a). This result would also arise if food limitation forces prey to reduce antipredator behavior, increasing their vulnerability to predation. For example, Winnie and Creel [11] showed that male elk, with significantly lower fat stores than females, mounted weaker antipredator responses to wolves than females did, despite facing higher per capita predation risk.

Small-scale experiments that manipulate the lethality of predators and field experiments that manipulate food supply and exposure to predators have been central to understanding risk effects. Still, a major challenge and opportunity remains to develop field studies measuring the relative magnitudes of direct predation and risk effects in the wild, without manipulations that could alter their relative magnitudes. Pangle *et al.* [18] recently demonstrated a promising approach, estimating direct and risk effects for two zooplankton prey (*Daphnia* and *Bosmina*) using a combination of field data on antipredator responses and experimental data on the demographic costs of these responses. Box 2 suggests another possible method to

Box 2. A challenge and an opportunity: measuring risk effects in field studies

To illustrate the difficulties of estimating the magnitudes of risk effects and direct predation using observational data, consider wolf-elk interactions. Since the reestablishment of wolf predation on the Gallatin elk winter range in the Yellowstone ecosystem, the proportion of female elk entering winter with a calf has declined by more than a factor of two (Figure 1). In parallel, the population has declined [6], although most Montana elk populations simultaneously grew strongly during this period [48]. Much of the decline in Gallatin calf recruitment can be attributed to reduced reproduction. The progesterone levels of female elk declined significantly with increasing wolf:elk ratios, and these changes in progesterone were significantly correlated with changes in calf recruitment [19]. For Gallatin elk, the calf:cow ratio immediately after the annual birth pulse has declined by 39% since wolf recolonization. These changes in reproduction were associated with strong changes in female elk behavior [11], habitat selection [6], grouping [33] and sensitivity to environmental conditions [17]. Reduced calf numbers after the birth pulse account for a decline of 17.3 calves/100 cows (Figure 1). Increased bear predation on very young calves could potentially contribute to this decline, by increasing the number of calves killed before they can be counted. Contrary to this hypothesis, summer calf:cow ratios were not detectably related to estimated bear density (general linear model: Wald statistic = 0.004, $P = 0.95$, using breeding bear numbers in Yellowstone National Park [49]), whereas wolf presence remains a strong predictor of summer calf:cow ratios after controlling for the potential effect of bears and snow accumulation during the gestation period (GLM: Wald statistic = 6.12, $P = 0.013$). Grizzly bear numbers in the Yellowstone ecosystem have increased mainly by population expansion, but the density of breeding bears within the ecosystem

core has not changed detectably (Figure 5 in Ref. [49]).

How does the impact of risk on elk reproduction compare to the impact of direct calf predation? Calf survival from birth to early winter has declined by 37% from the pre-wolf period to the post-wolf period, but this decline in survival cannot be neatly attributed to direct predation or risk effects. At least in principle, it is a combination of the two. Even if all of the change in calf survival from the pre-wolf period to the post-wolf period was due to direct wolf predation (i.e. no risk effects on calf survival), this would account for a decline of only 6.3 calves/100 cows. (In Figure 1, 6.3 is the difference between the pre-wolf and post-wolf periods for the decrease in calf:cow ratios from summer to winter.) Radiotelemetry of wolves [37] and elk calves [19] has been used to directly estimate the rate of predation. In the winter, observations of Gallatin wolf kills yield a direct predation rate of 0.030 calves killed/wolf/day, closely comparable to the rate of 0.027 calves killed/wolf/day for a neighboring population [37]. If this winter predation rate applied also to summer and fall, then wolves on the Gallatin site would kill an average of 74 calves in 180 days between birth and early winter. (Note that studies of radiotagged elk calves show lower rates of wolf predation in summer/fall than in winter, but do not allow precise estimates of the rate [19].) Direct killing of 74 calves accounts for a decline of 9.0 calves/100 cows, given the size of the Gallatin herd ($X \pm \text{SEM} = 1138 \pm 86$ elk, 1996–2006) and the proportion that are adult females (72%).

Thus, for elk calf recruitment, risk effects on reproduction were ~2–3 times larger than the effect of direct predation (17.9 versus 6.3 to 9.0 calves/100 cows), and the total effect of predation on recruitment was ~3–4 times larger than was captured by measures of direct predation.

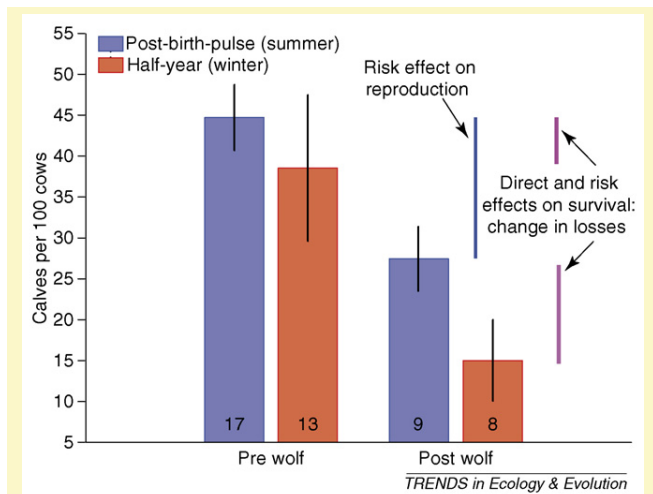


Figure 1. Wolf predation and elk calf number: elk dynamics are strongly affected by calf recruitment, and the proportion of females entering winter with a calf has declined significantly ($t_{17} = 5.12$, $P < 0.0001$) since the reestablishment of wolf predation on the Gallatin elk winter range in the Yellowstone ecosystem. A large proportion of this decline in winter calf recruitment can be attributed to reduced reproduction (a risk effect), as revealed by a significant drop in calf:cow ratios immediately following the summer birth pulse ($t_{23} = 6.79$, $P < 0.0001$). The sum of direct wolf predation and risk effects on calf survival are manifest as changes in survival rates from birth to 6 months for the pre-wolf and post-wolf periods. Bars show means; whiskers show 95% confidence intervals. Numbers within bars are sample sizes (annual surveys).

compare the demographic costs of direct predation and risk effects, using observational data from the wild.

Conclusions

The benefit of antipredator behavior is a decreased risk of predation, and this is automatically incorporated into measures of the direct rate of predation. By contrast, most analyses of vertebrate predator–prey dynamics do not account for the costs of antipredator behavior (risk effects). Logically, risk effects can be larger than the effects of direct predation. By the same logic, risk effects can be either negatively or positively correlated with direct effects, depending on the type of comparison being made. Excellent experimental studies of a broad range of species have confirmed that risk effects on prey demography, density and dynamics can be large, sometimes exceeding direct effects. If risk effects are not considered explicitly, it seems likely that they will go undetected or be attributed to processes other than predation, particularly when risk effects reduce reproduction. The challenge now is to design field studies that determine the relative magnitudes of risk effects and direct predation in wild populations that are not manipulated. Finally, risk effects are becoming well recognized in pure ecology, but are still largely ignored in the fields of conservation and management. It is important that measurement of risk effects penetrates into work on predation as a limiting factor in problems of conservation and wildlife management.

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References

- 1 Peckarsky, B.L. *et al.* (1993) Sublethal consequences of stream-dwelling predatory stoneflies on mayfly growth and fecundity. *Ecology* 74, 1836–1846
- 2 Boonstra, R. *et al.* (1998) The impact of predator-induced stress on the snowshoe hare cycle. *Ecol. Monogr.* 68, 371–394
- 3 Tollrian, R. and Harvell, C.D. (1999) The evolution of inducible defenses: current ideas. In *The Ecology and Evolution of Inducible Defenses* (Tollrian, R. and Harvell, C.D., eds), pp. 306–321, Princeton University Press
- 4 Lima, S.L. (1998) Nonlethal effects in the ecology of predator–prey interactions. *Bioscience* 48, 25–34
- 5 Anholt, B.R. and Werner, E.E. (1999) Density-dependent consequences of induced behavior. In *The Ecology and Evolution of Inducible Defenses* (Tollrian, R. and Harvell, C.D., eds), pp. 218–230, Princeton University Press
- 6 Creel, S. *et al.* (2005) Elk alter habitat selection as an antipredator response to wolves. *Ecology* 86, 3387–3397
- 7 Hebblewhite, M. *et al.* (2005) Human activity mediates a trophic cascade caused by wolves. *Ecology* 86, 2135–2144
- 8 Childress, M.J. and Lung, M.A. (2003) Predation risk, gender and the group size effect: does elk vigilance depend upon the behaviour of conspecifics? *Anim. Behav.* 66, 389–398
- 9 Armitage, K.B. (2004) Badger predation on yellow-bellied marmots. *Am. Midl. Nat.* 151, 378–387
- 10 Lima, S.L. and Bednekoff, P.A. (1999) Temporal variation in danger drives antipredator behavior: the predation risk allocation hypothesis. *Am. Nat.* 153, 649–659
- 11 Winnie, J., Jr and Creel, S. (2007) Sex-specific behavioural responses of elk to spatial and temporal variation in the threat of wolf predation. *Anim. Behav.* 73, 215–225
- 12 Boesch, C. (1991) The effects of leopard predation on grouping patterns in forest chimpanzees. *Behaviour* 117, 220–242
- 13 Barta, Z. *et al.* (2004) The effects of predation risk on the use of social foraging tactics. *Anim. Behav.* 67, 301–308
- 14 Sih, A. and McCarthy, T.M. (2002) Prey responses to pulses of risk and safety: testing the risk allocation hypothesis. *Anim. Behav.* 63, 437–443
- 15 Schmitz, O.J. *et al.* (1997) Behaviorally mediated trophic cascades: effects of predation risk on food web interactions. *Ecology* 78, 1388–1399
- 16 Fortin, D. *et al.* (2005) Wolves influence elk movements: behavior shapes a trophic cascade in Yellowstone National Park. *Ecology* 86, 1320–1330
- 17 Winnie, J., Jr *et al.* (2006) Elk decision-making rules are simplified in the presence of wolves. *Behav. Ecol. Sociobiol.* 61, 277–289
- 18 Pangle, K.L. *et al.* (2007) Large nonlethal effects of an invasive invertebrate predator on zooplankton population growth rate. *Ecology* 88, 402–412
- 19 Creel, S. *et al.* (2007) Predation risk affects reproductive physiology and demography of elk. *Science* 315, 960
- 20 Ruxton, G.D. (1997) Predator-induced breeding suppression and its consequences for predator–prey population dynamics. *Proc. Biol. Sci.* 264, 409–415
- 21 Nelson, E.H. *et al.* (2004) Predators reduce prey population growth by inducing changes in prey behavior. *Ecology* 85, 1853–1858
- 22 Preisser, E.L. *et al.* (2005) Scared to death? The effects of intimidation and consumption in predator–prey interactions. *Ecology* 86, 501–509
- 23 Eberhardt, L.L. *et al.* (2003) Assessing the impact of wolves on ungulate prey. *Ecol. Appl.* 13, 776–783
- 24 White, P.J. and Garrott, R.A. (2005) Yellowstone's ungulates after wolves—expectations, realizations, and predictions. *Biol. Cons.* 125, 141–152
- 25 Varley, N. and Boyce, M.S. (2006) Adaptive management for reintroductions: updating a wolf recovery model for Yellowstone National Park. *Ecol. Model.* 193, 315–339
- 26 Bolker, B. *et al.* (2003) Connecting theoretical and empirical studies of trait-mediated interactions. *Ecology* 84, 1101–1114
- 27 Werner, E.E. and Peacor, S.D. (2003) A review of trait-mediated indirect interactions in ecological communities. *Ecology* 84, 1083–1100
- 28 Luttbeg, B. *et al.* (2003) Prey state and experimental design affect relative size of trait- and density-mediated indirect effects. *Ecology* 84, 1140–1150

- 29 Dill, L.M. *et al.* (2003) Behaviorally mediated indirect interactions in marine communities and their conservation implications. *Ecology* 84, 1151–1157
- 30 McNamara, J.M. and Houston, A.I. (1987) Starvation and predation as factors limiting population size. *Ecology* 68, 1515–1519
- 31 Pulliam, H.R. (1973) On the advantages of flocking. *J. Theor. Biol.* 38, 419–422
- 32 Caro, T. (2005) *Antipredator Defenses in Birds and Mammals*, University of Chicago Press
- 33 Creel, S. and Winnie, J.A., Jr (2005) Responses of elk herd size to fine-scale spatial and temporal variation in the risk of predation by wolves. *Anim. Behav.* 69, 1181–1189
- 34 Werner, E.E. *et al.* (1983) An experimental test of the effects of predation risk on habitat use in fish. *Ecology* 64, 1540–1548
- 35 Karels, T.J. *et al.* (2000) The interactive effects of food and predators on reproduction and overwinter survival of arctic ground squirrels. *J. Anim. Ecol.* 69, 235–247
- 36 Byrom, A.E. *et al.* (2000) Experimental manipulation of predation and food supply of arctic ground squirrels in the boreal forest. *Can. J. Zool.* 78, 1309–1319
- 37 Smith, D.W. *et al.* (2004) Winter prey selection and estimation of wolf kill rates in Yellowstone National Park, 1995–2000. *J. Wildl. Manag.* 68, 153–166
- 38 Krebs, C.J. *et al.* (1995) Impact of food and predation on the snowshoe hare cycle. *Science* 269, 1112–1115
- 39 Volterra, V. (1926) Variations and fluctuations of the numbers of individuals in animal species living together. *Mem. R. Accad. Naz. dei Lincei* (translated in Chapman, R. [1931] *Animal Ecology*, McGraw-Hill)
- 40 Abrams, P.A. and Ginzburg, L.R. (2000) The nature of predation: prey dependent, ratio dependent or neither? *Trends Ecol. Evol.* 15, 337–341
- 41 Eberhardt, L.L. (2000) Reply: predator-prey ratio dependence and regulation of moose populations. *Can. J. Zool.* 78, 511–513
- 42 Hayes, R.D. and Harestad, A.S. (2000) Wolf functional response and regulation of moose in the Yukon. *Can. J. Zool.* 78, 60–66
- 43 Messier, F. and Joly, D.O. (2000) Comment: regulation of moose populations by wolf predation. *Can. J. Zool.* 78, 506–510
- 44 Abrams, P.A. and Matsuda, H. (1997) Prey adaptation as a cause of predator-prey cycles. *Evolution Int. J. Org. Evolution* 51, 1742–1750
- 45 Ives, A.R. and Dobson, A.P. (1987) Antipredator behavior and the population dynamics of simple predator-prey systems. *Am. Nat.* 130, 431–447
- 46 Luttbeg, B. and Schmitz, O.J. (2000) Predator and prey models with flexible individual behavior and imperfect information. *Am. Nat.* 155, 669–683
- 47 Boyce, M.S. (1995) Anticipating consequences of wolves in Yellowstone: model validation. In *Ecology and Conservation of Wolves in a Changing World* (Carbyn, L.C. *et al.*, eds), pp. 199–209, Canadian Circumpolar Institute
- 48 Hamlin, K.L. (2004) *Montana Elk Management Plan*. Montana Fish, Wildlife & Parks
- 49 Schwartz, C.C. *et al.* (2006) Temporal, spatial, and environmental influences on the demographics of grizzly bears in the Greater Yellowstone Ecosystem. *Wildl. Monogr.* 161, 1–68

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