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REVIEW

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Non-consumptive predator effects on prey population size: A dearth of evidence

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Abstract

1. There is a large and growing interest in non-consumptive effects (NCEs) of predators. Diverse and extensive evidence shows that predation risk directly influences prey traits, such as behaviour, morphology and physiology, which in turn, may cause a reduction in prey fitness components (i.e. growth rate, survival and reproduction). An intuitive expectation is that NCEs that reduce prey fitness will extend to alter population growth rate and therefore population size.
2. However, our intensive literature search yielded only 10 studies that examined how predator-induced changes in prey traits translate to changes in prey population size. Further, the scant evidence for risk-induced changes on prey population size have been generated from studies that were performed in very controlled systems (mesocosm and laboratory), which do not have the complexity and feedbacks of natural settings. Thus, although likely that predation risk alone can alter prey population size, there is little direct empirical evidence that demonstrates that it does. There are also clear reasons that risk effects on population size may be much smaller than the responses on phenotype and fitness components that are typically measured, magnifying the need to show, rather than infer, effects on population size.
3. Herein we break down the process of how predation risk influences prey population size into a chain of events (predation risk affects prey traits, which affect prey fitness components and population growth rate, which affect prey population size), and highlight the complexity of each transition. We illustrate how the outcomes of these transitions are not straightforward, and how environmental context strongly dictates the direction and magnitude of effects. Indeed, the high variance in prey responses is reflected in the variance of results reported in the few studies that have empirically quantified risk effects on population size. It is therefore a major challenge to predict population effects given the complexity of how environmental context interacts with predation risk and prey responses.
4. We highlight the critical need to appreciate risk effects at each level in the chain of events, and that changes at one level cannot be assumed to translate into changes in the next because of the interplay between risk, prey responses, and the environment. The gaps in knowledge we illuminate underscore the need for more evidence to substantiate the claim that predation risk effects extend to prey

population size. The lacunae we identify should inspire future studies on the impact of predation risk on population-level responses in free-living animals.

KEYWORDS

anti-predator response, fear effects, indirect effects, non-lethal effects, phenotypic plasticity, predation risk, predator–prey interactions, trait-mediated effects

1 | INTRODUCTION

Predation is a central organizing process in ecology, expected to influence population dynamics, community structure and ecosystem function (Krebs et al., 1995; Schmitz, 2008). Classically, ecologists have focused on the consumptive effects (CEs) of predators, namely the killing of prey (e.g. Paine, 1969; Sih, Crowley, McPeek, Petranka, & Strohmeier, 1985). Ecologists now appreciate that predators can also influence prey via non-consumptive effects (NCEs); herein defined as risk-induced trait responses that alter individual prey fitness components. Because the terminology is often used differently, we denote the first response as risk-induced trait response (i.e. of prey phenotype), and that NCEs involve changes to prey fitness components, population growth rate and prey population size (Clinchy, Sheriff, & Zanette, 2013; Lima, 1998; Peckarsky et al., 2008). Thus, predation risk can influence prey behaviour, morphology, life history and physiology (Lima, 1998; Sheriff & Thaler, 2014; Werner & Peacor, 2003), which can alter prey fitness components such as individual somatic growth rate, survival and reproduction (McCauley, Rowe, & Fortin, 2011; Peckarsky, Cowan, Penton, & Anderson, 1993; Sheriff, Krebs, & Boonstra, 2009; Trussell, 2000; Turner & Montgomery, 2003; Werner & Anholt, 1996; Zanette, White, Allen, & Clinchy, 2011), which can in turn affect population growth rate and prey population size (Figure 1). How risk influences prey responses across this chain of events, from individual trait changes through to population size, has important implications for understanding how predator–prey interactions shape communities and affect ecosystems (Estes et al., 2011; Schmitz, Hawlena, & Trussel, 2010).

Whereas it may seem self-evident that large risk effects on prey traits and fitness components should result in large consequences to population size, surprisingly few studies have actually quantified the effects of predation risk on population size (Table 1). We acknowledge that there is a strong argument, validated by experimental

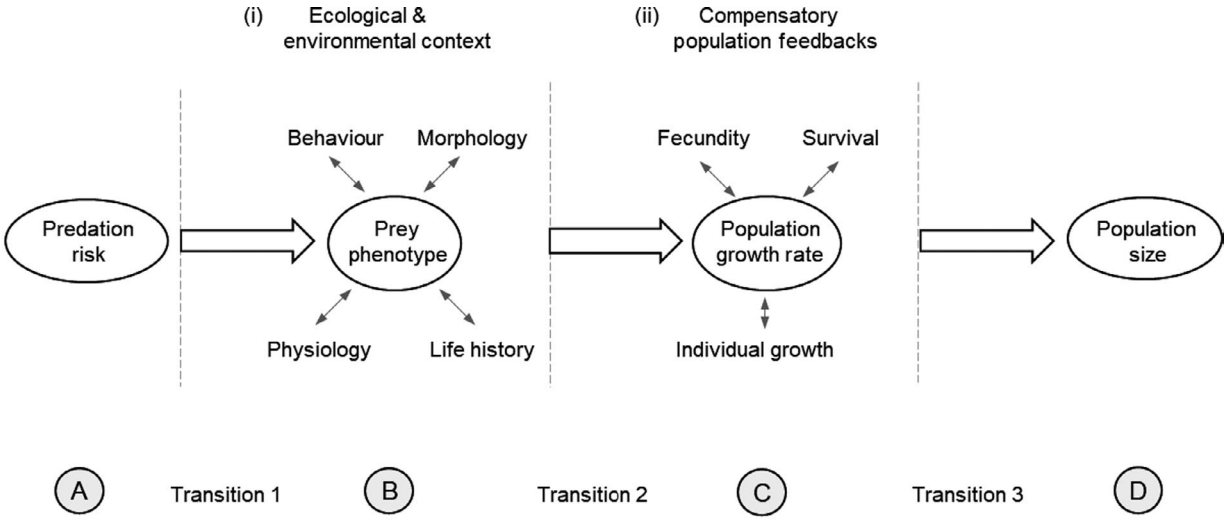

FIGURE 1 The chain of events from predation risk to prey population size. Predation risk (level A) acts to alter prey phenotype (level B), which can influence fitness components (growth rate, fecundity and survival; double-headed arrows again represent the feedbacks possible) and population growth rate (level C). These effects on prey fitness components and population growth rate have the potential to scale up and alter population size (level D). Double-headed arrows for prey phenotype and fitness components represent the fact that a risk-induced change in one aspect may result in changes in another aspect (for e.g. change in behaviour can result in physiological compensation). Key to this chain of events is that even very large changes at one level may not lead to changes in another, because of the factors that influence the Transitions (1–3) between levels. (i) Ecological or environmental context will play an important role in Transition 1, in the way that prey respond to predation risk (e.g. predator/prey diversity, refuge prevalence), and in Transition 2, in how induced-trait changes impact prey fitness (e.g. compensatory trait changes, how the trait change effects other species including resource abundance). (ii) Compensatory population feedbacks may influence the form of Transition 3, in how predation risk influences population size (e.g. density-dependent immigration may compensate for risk-induced emigration, risk-induced reductions in reproduction may not limit recruitment). See Section 4 for detailed discussion of factors that influence transitions

TABLE 1 Studies that have examined the influence of predation risk and risk-induced trait changes on prey population size (number of individuals)

Time-scale	Predation risk from	Prey taxa	Reference
One generation of prey (no consumption)	Zooplanktivorous cladoceran (chemical cues)	Diaptomid copepods	Bourdeau et al. (2016)
	Damsel bugs (free-ranging with surgically altered mouths)	Pea aphid	Nelson et al. (2004)
	Redear sunfish (caged in mesocosm)	Freshwater snails	McCollum et al. (1998)
Multiple generations of prey (no consumption)	*Stenostomum* (freeze killed and strained; chemical cue)	Protist community	Forbes and Hammill (2013)
	Bluegill sunfish (caged in mesocosm)	Multiple zooplankton species	Peacor et al. (2012)
	Common sunfish, water bugs, crayfish (caged in mesocosm)	Freshwater snails	Hoverman and Reylea (2012)
	Roach fish kairomone	Multiple *Daphnia* species	Dawidowcz and Wielanier (2004)
	Chaoborus kairomone	*Daphnia pulex* clones	Boeing et al. (2005)
Multiple generations of prey (with consumption)	*Chaoborus*	*Daphnia pulex* clones	Boeing et al. (2005)
	Planktivorous fish (golden shiner)	*Daphnia pulex* clones	Boeing and Ramcharan (2010)
	Asplanchna rotifers	*Brachionus* rotifers	van der Stap et al. (2007)

studies to support the claim that the consequences of predation risk on prey could affect prey population size (Abrams, 1984, 1995, Brown & Kotler, 2007; Peacor, Peckarsky, Trussell, & Vonesh, 2013; Sheriff, Krebs, & Boonstra, 2011); however, we contend that a lack of empirical evidence motivates a need for renewed attention to this problem, especially if we are to understand under what circumstances (e.g. which species or habitats) predation risk effects are likely to influence population size. Further, we put forth three arguments for why risk effects at the population level are difficult to predict and may not occur in the direction or magnitude assumed from findings on risk-induced trait responses (Lima, 1998; more detail in latter sections). First, the interaction between predation risk, prey responses and other food web interactions (such as predation itself, alternate prey, apparent competition and food availability) may negate or interfere with risk-induced changes to prey population size (Abrams, 2010). Second, classic ecological theory illustrates many conditions in which changes in survival and reproduction do not alter population size (Hutchinson, 1978; Krebs, 1994). Third, theoretical studies suggest that inferences from short-term experiments on risk-effects may overstate long-term effects (e.g. Luttbeg, Rowe, & Mangel, 2003; Persson & De Roos, 2003). Thus, there is little empirical and theoretical evidence to form a basis for predictions on when and how predation risk may alter population size.

Despite the lack of evidence, claims of strong population-level changes in prey population size from predation risk alone are numerous in the scientific literature. From a survey (using search terms: predat* and non-consumptive, or predat* and nonconsumptive) of published papers limited to two recent years (2016 and 2017), we found that over 40% of papers make an implicit or explicit claim that predation risk reduces population size via NCEs (from $N = 60$ papers total, our search yielded 138 papers but we removed studies that did not strictly pertain to predation risk and either phenotypic changes or NCEs, see Supporting Information). Most of these claims were made in the introduction and included statements such as, '*…NCEs of predators were shown to have equal-or-greater effects on prey population size than direct consumption…*', '*…Evidence suggests that predators not only regulate animal populations through consumptive effects but also by the mere threat of predation…*', '*…predators can negatively impact prey populations either by directly consuming prey individuals or through non-consumptive effects…*'. Although these statements were used to justify the motivation for these studies, none of them (and the many other papers that make similar claims) cite studies that provide direct evidence to support such statements about NCEs on prey population size in natural communities as implied.

To be clear, predation risk has been examined and demonstrated to have significant effects on multiple aspects of individual phenotype and fitness components, that provide evidence of the potential to scale up and alter population size (see below). For example, the mean fitness of all individuals within a population equates to Malthusian population growth rate (r; Fisher, 1930), and predation risk may alter population growth rate via change in fitness components (Figure 1; Pangle, Peacor, & Johannsson, 2007; Reede & Ringelberg, 1995). But the link between changes

in mean fitness (population growth rate) and changes in population size is not straightforward, given that the context under which individual fitness components are estimated may not reflect those found in natural systems. There can be many factors that can affect both the fitness components of an individual and the mean fitness in a population in different ways (see sections below). This makes it difficult to scale from risk-induced effects on a single fitness component (which may be used to estimate population growth rate) to risk-induced effects on population size in natural systems. There have also been many studies and reviews comparing the relative effects of CEs versus NCEs on community and ecosystem level processes that suggest that risk effects can be as large, or larger, than effects due to consumption (e.g. Abrams, 1995; Peacor & Werner, 2001; Preisser, Bolnick, & Benard, 2005; Schmitz et al., 2010). The preponderance of such studies, however, has led to the general assumption that NCEs have an inevitable effect on population size, when in fact studies testing this hypothesis are greatly lacking.

In this paper we address the large disconnect in what is being claimed concerning the effects of predation risk on prey population size, and what has actually been shown in the literature. After reviewing this disconnect in more detail, we propose that a key reason for this disconnect is an underappreciation for the many and complex links that occur between the initial trait change and a potential effect on population size. For example, given the vast number of studies examining effects of adaptive prey responses on prey fitness components, many of which suggest that effects on population size are common and well understood, ecologists may incorrectly surmise that there is evidence that the induction of anti-predator responses itself affects population size. Here we explore the relationship between predation risk and prey populations, focusing broadly on how the effects of risk scale across levels, from influencing prey traits to population size. We first examine the empirical evidence and acknowledge the various ways that predation risk has been shown to alter individual traits and fitness components (see below), and then highlight the lack of sufficient evidence for the extension to population size. We argue that there is a need to appreciate how risk effects translate across this chain of events, and that changes at one level may not necessarily alter the next. In examining the prevailing empirical evidence, we provide insights for future directions that will help inform and extend our understanding of the impact of predation risk on prey responses and their population size.

2 | BEING EXPLICIT ABOUT A POPULATION

It is critical that when we discuss predation risk effects on population size, we make explicit what is meant by a population. Often, as

BOX 1 Risk-induced dispersal and its effects on population size

Risk-induced dispersal of prey (a behavioural effect) and its influence on population size requires separate consideration when evaluating the influence of predation risk across the chain of events (Figure 1). Many studies have shown that predation risk can influence local prey density within a designated area through increased emigration or dispersal rates (e.g. Forrester, 1994; Peckarsky, 1980; Sih, 1982; Sih, Kats, & Moore, 1992). For example, in a Trinidadian forest stream system, Fraser and Gilliam (1992) found that an experimental increase in the piscivorous fish *Hoplias malabaricus* altered local space use, increased emigration and reduced local population size of the killifish *Rivulus hartii*. However, for such risk-induced trait changes (movement) to affect the size of a population, not just the number of individuals in a designated local area, it must alter the fitness of the moving individuals (and those remaining). If it does not, the change in local population size, because of risk-induced movement, is scale dependent and changes in the total size of the population (the group of individuals influenced by risk exposure) remain undetected or unexamined in many studies. For example, many studies have found that invertebrate prey migrate deeper in the water column in response to predation risk (e.g. Werner & Peacor, 2006) or use different areas within their habitat (e.g. Schmitz, Beckerman, & O'Brien, 1997); however, the overall population size in these mesocosm studies has not changed. Further, even if there is a cost to the movement, the net cost may be low because it is offset by other benefits (Hawlena & Schmitz 2010a, 2010b; Peacor et al., 2013); i.e. it is the balance of all negative and positive outcomes of migration that must be considered.

Alternatively, if prey fail to perceive the risk of predation in a given area and immigrate into areas of high risk, the spatial and temporal nature of the landscape can create ecological trap dynamics. For example, Hawlena, Saltz, Abramsky, and Bouskila (2010) showed that the critically endangered lizard *Acanthodactylus beershebensis* dispersed into areas that were risky. This resulted in extirpation of lizards from both risky and surrounding less-risky habitats. As such, through both the risk-induced emigration from risky areas and also the naive immigration into risky areas, predation risk in one area may have far reaching landscape-level effects which are difficult to predict based only on local responses. Thus, although we recognize that risk-induced dispersal and migration may have local effects on the number of individuals and trophic interactions at that locale, we caution the interpretation that such effects alter overall population size without a clear understanding of the fitness consequences for both remaining and moving individuals. Future studies are needed to test how predation risk may interact with migration to alter population size at both local and broad scales.

we define here, a population is the number of individuals of the same species that live within a given area at the same time; however, it is then critical to distinguish between localized predation risk that may affect individual fitness in a subpopulation and broader scale effects that may determine the total population size. For instance, consider a system of two ponds, pond A and B, on an island. An aquatic beetle species is found on the island which inhabits water for its larval and adult stages, only leaving the pond to disperse. Given many insects avoid ovipositing in ponds with predators (Binckley & Resetarits, 2005) researchers become interested in examining this effect for our hypothetical species. Caged fish are introduced into pond A and the beetle population is drastically reduced because they are no longer ovipositing in this pond. Observations now show that beetles preferentially oviposit in pond B and the population there has increased. A focused study on pond A would find that population size was strongly negatively affected by predation risk. In this paper, however, we view the population size as including all individuals affected by predation risk in the entire population. Thus, an understanding of the influence of predation risk on the population would require an investigation of both ponds, while an understanding of predation risk at the local scale would require an investigation in only the pond of interest. Clearly, viewing the population as that in pond A, or as that in pond A and B, address different aspects of predation risk effects. However, in many systems one of the trait responses to predators is a change in habitat preference, as with fish in streams, tadpoles in tanks, elk in different valleys and grasshoppers in fields, and thus narrowing the population to that in a habitat preferred in a study of predation risk effects on prey population size before predators are present exaggerates the influence of the predator on prey population size across their entire range (see further discussion in Box 1).

3 | A BROKEN CHAIN OF EVENTS: RISK EFFECTS AT ONE LEVEL DO NOT IMPLY CHANGES IN THE NEXT

In the study of the cascading effects of predation risk, a major goal is to understand adaptive prey responses and determine whether and how predation risk affects population size. In other words, does risk influence prey population size and alter how species are represented in a given environment? To address this question, we decompose the potential chain of events leading from predation risk to prey population size into multiple levels (Figure 1): (a) predation risk, (b) prey traits, (c) prey population growth rate, (d) prey population size. We then examine the transition between levels separately: (a) perceived predation risk induces a change in prey traits (behaviour, morphology, physiology and/or life history), which can (b) cause a change in estimated prey population growth rate via changes in fitness components (growth and developmental rates, fecundity and survival), which can (c) alter prey population size. This decomposition makes more explicit the process examined in individual studies, the status of the problem and gaps in our knowledge.

Key to this chain of events is understanding that there are multiple processes involved in the transition between levels, thus creating conditions wherein changes in one level may have unpredicted effects on another (see Section 4 below). For example, a large change in activity or habitat use may have little effect on fitness components if that change is accompanied by compensatory physiological and behavioural responses (Dewitt, Sih, & Hucko, 1999; McPeek, 2004) or if such behavioural changes are associated with little cost (Peacor et al., 2013). Further, a large reduction in individual reproduction or survival may not directly translate to changes in population size (Hutchinson, 1978). The transition between levels could also be strongly context dependent (Figure 1), influenced by attributes of the predator, prey and environment (Creel, 2011, 2018). For example, Kaplan and Thaler (2010) found that plant resistance attenuated the effects of both NCEs and CEs of predators on insect prey. It is therefore crucial that empirical evidence is generated that determines when effects at one level will translate to alter the next. This further implies a critical need for studies performed in natural conditions, where context dependencies and natural feedbacks can occur that influence the transition from one level to the next.

3.1 | Transition 1: Risk-induced trait responses in prey

There is a large and growing body of work, across taxa, showing that predation risk can alter prey traits (Transition 1; Figure 1). This has been reviewed extensively elsewhere (e.g. Agrawal, 2001; Creel, 2011; Lima, 1998; Lima & Dill, 1990; Sheriff & Thaler, 2014; Werner & Peacor, 2003), so we will only discuss this transition briefly here. Behavioural responses of prey are among the best studied, and in general, prey decrease foraging activity and increase vigilance (e.g. Armitage, 2004; Kotler, Brown, Slotow, Goodfriend, & Strauss, 1993; Lima & Bednekoff, 1999), increase aggregation and group size (e.g. Caro, 2005; Thaker, Vanak, Owen, Ogden, & Slotow, 2010) and spatio-temporally shift habitat use and refuge use (e.g. Donelan, Grabowski, & Trussell, 2017; Sih & McCarthy, 2002; Smith, Donadio, Pauli, Sheriff, & Middleton, 2019; Thaker et al., 2011; Valeix et al., 2009; Werner & Anholt, 1993). These behavioural tactics of prey across multiple spatial and temporal scales attempt to minimize predation risk. Such studies have formed the basis for much of the theories on predator risk effects and predator–prey interactions (reviewed in Abrams, 2010; Peacor & Cressler, 2012), such as the risk allocation hypothesis (Lima & Bednekoff, 1999). For prey that have the capacity for morphological plasticity, alterations to avoid predation pressure have been shown in body shape and size or the development of defensive structures (e.g. Bronmark & Miner, 1992; Lass & Spaak, 2003; Relyea, 2004). For example, classic work shows that *Daphnia* form a helmet, neck teeth and a tail spine in the presence of predators (Dodson, 1989). Much of the work on predator-induced physiological responses of prey has been on changes in stress hormone levels, however, other physiological responses such as changes in metabolic rate, assimilation efficiency and gene

expression have also been demonstrated (reviewed in Sheriff & Thaler, 2014; Zanette, Clinchy, & Suraci, 2014). Additionally, life-history characteristics can be altered in response to increased predation risk (Reznick, Bryga, & Endler, 1990; Reznick, Butler, & Rood, 2001), which include alteration in age and size at maturity (e.g. Beckerman, Rodgers, & Dennis, 2010; Stoks, Block, Slos, Doorslaer, & Rolff, 2006), timing of metamorphosis (e.g. Pangle et al., 2007; Skelly & Werner, 1990; Stoks, Block, Meutter, & Johansson, 2005) and aspects of offspring phenotype (e.g. Hawlena, Kress, Dufresne, & Schmitz, 2011; Lass & Spaak, 2003; Sheriff et al., 2009; Sheriff, Krebs, & Boonstra, 2010).

Importantly, these trait responses do not act in isolation of one another (e.g. Lind & Cresswell, 2005; Werner & Anholt, 1993). For example, Thaler, McArt, and Kaplan (2012) found that in response to predatory stink bugs, hornworm caterpillars *Manduca sexta* reduced feeding by 30%–40%; however, they concurrently enhanced their nutrient assimilation and extraction efficiency, compensating for their reduced food intake via their nutritional physiology. Similarly, Auld and Relyea (2008) showed that snails delayed reproductive onset in the presence of crayfish, however, this was offset by an extended lifetime. Further, animals may be able to have a large response in one trait without it being very costly to other traits. For example, in African ungulates, Creel, Schuette, and Christianson (2014) found that predator-induced increases in vigilance most often did not cause a reduction in foraging (but see Creel et al., 2017). Thus, understanding the suite of prey responses and their interactions (Cressler, King, & Werner, 2010; Dewitt et al., 1999) will better inform our understanding of predation risk effects at the individual level.

3.2 | Transition 2: Risk-induced trait changes alter prey fitness components and estimates of population growth rate

Adaptive prey responses to predation risk (i.e. anti-predator responses) have an overall net positive effect to the prey as compared to not responding, which is consistent with the basic tenet of plasticity, wherein trait changes reflect a balance between benefits and costs (Abrams, 2010; Lind & Cresswell, 2005; Peacor & Cressler, 2012). Yet there is a great deal of evidence and focus on the associated costs that risk-induced trait changes have on prey fitness components (given its usefulness in estimating population growth rate, Transition 2; Figure 1).

Predation risk effects on prey traits, such as foraging rate and time spent searching for optimal patches has been shown to influence prey growth rates, with implications to reproduction and survival, in many taxa, including anuran larvae (e.g. Werner & Anholt, 1996), and both marine (e.g. Trussell, 2000) and freshwater (e.g. Pangle & Peacor, 2006; Pangle et al., 2007; Peckarsky et al., 1993; Turner & Montgomery, 2003) invertebrates. For example, in experimental mescososms, Werner and Anholt (1996) found that the presence of caged dragonfly predators reduced activity and competitive ability, which was responsible for reduced individual growth rates and increased mortality probability in anuran larvae prey.

Predation risk can influence the probability of prey mortality through two broad mechanisms. First, risk effects that influence physiology and condition of prey can affect the probability of mortality (i.e. in the absence of mortality inflicted by other species; e.g. McCauley et al., 2011; Schmitz et al., 1997). For example, Macleod, Krebs, Boonstra, and Sheriff (2018) found that experimental exposure to a dog for 1 min every other day increased stress hormone levels and mortality probability in captive-held, wild snowshoe hares. Second, trait responses to predators can indirectly affect the probability of prey mortality by increasing their susceptibility to other factors, such as parasites (Hawlena, Abramsky, & Bouskila, 2010) and other predators (e.g. predator facilitation, Charnov, Gordon, & Hyatt, 1976; reviewed in Werner & Peacor, 2003). For example, Soluk and Collins (1988) showed that stonefly larvae drive mayflies from the undersurface safety of rocks and increase their susceptibility to predation by fish. Embar, Raveh, Hoffman, and Kotler (2014) showed that vipers and owls facilitate each other's hunting by limiting the refuge space and anti-predator behaviour options of gerbils.

There are also many examples from diverse taxa of risk effects on prey fecundity and the resulting number of offspring (Magnhagen, 1991; Sih, 1994). For example, in free-living song sparrows, Zanette et al. (2011) showed that playbacks of predator calls altered pre- and post-natal maternal behaviour (e.g. reduced incubation and feeding), leading to a reduction in the number of eggs laid, and the hatching and fledgling success of those eggs. In snowshoe hares, Sheriff et al. (2009) showed that increased predation risk led pregnant females to increase stress hormones, which reduced the number of offspring born, and the size and weight of those offspring. In the lecithotrophic mosquito fish (but not in the matrotrophic least killifish), Mukherjee, Heithaus, Trexler, Ray-Mukherjee, and Vaudo (2014) show that predation risk suppressed reproduction, potentially via reduced foraging and increased physiological stress, resulting in smaller clutches of larger fry.

Finally, some studies have also calculated estimates of intrinsic population growth rate from closed systems based on risk-induced changes in these fitness metrics (e.g. change in fecundity) rather than a change in the number of individuals in a population (e.g. Pangle et al., 2007). For example, Reede and Ringelberg (1995) measured the influence of juvenile perch exudate (cue of predation risk) on the number of offspring born to different clones of *Daphnia*. Females were kept individually in test tubes where they were exposed to the exudates and offspring continuously removed upon discovery. The number of babies born was used to calculate intrinsic rate of population increase. While very useful in understanding the potential of predation risk to alter population size, these types of results may not readily translate to free-living populations. As discussed in Reede and Ringelberg (1995), the greater intrinsic growth rate found in *Daphnia* exposed to fish exudates was driven by *Daphnia* having larger clutches of smaller neonates that are less likely to survive than larger neonates (although survival of offspring was not measured). As described above, most studies that investigate risk-induced

changes to prey fitness measure risk effects on certain fitness components; typically, prey condition and growth rate, probability of mortality and fecundity or number of offspring. A negative effect on a single fitness component, however, is not conclusive that predation risk has an overall negative effect on prey fitness, given potential compensatory changes in other fitness components.

3.3 | Transition 3: Risk-induced fitness changes on prey population size

Predation risk is predicted to alter prey population size by reducing prey fitness components and altering population growth rate, independently of CEs. It is this final transition (Transition 3; Figure 1) that is most important to understanding the population consequences of adaptive prey responses to predation risk, and in turn to fundamental ecological questions (e.g. species diversity, influence of species invasion) and management applications. Because of feedbacks inherent in natural systems (see Section 4 below), predicting the direction and magnitude of risk effects on population size is less straightforward than demonstrating the effects on prey traits and fitness components. For example, even within one generation of prey, Peacor and Werner (2004) found that over time, feedbacks within the system affected the direction of response; risk exposure initially reduced tadpole growth rate, yet, because of the subsequent reduction in foraging pressure, tadpole resources increased to a large enough degree that a net positive effect on tadpole growth rate was observed over a longer time-scale.

It is at this transition to population size (stage c and d; Figure 1), that empirical evidence is lacking. We performed an intensive literature search (Google Scholar from 1970 to 2017 and Web of Science from 1999 to 2017 using each of the keywords [or two words] *predation risk*, *risk effects*, *non-consumptive effects*, *non-lethal effects*, *indirect effects*, *or fear*, combined with each of *population dynamics*, *population abundance*, *or population density*; a second search was also performed in Google Scholar from 1970 to 2017 using the term '*population size NOT dynamics*, *abundance*, *density*' combined with each of *predation risk*, *risk effects*, *non-consumptive effects*, *non-lethal effects*, *indirect effects*, *or fear*). We also conducted a critical evaluation of every paper in the meta-analysis database generously provided by Evan Preisser (from Preisser et al., 2005; the database was updated to reflect studies up to 2006). When a keywords search yielded more than 1,000 papers (e.g. 'indirect effects' and 'population dynamics') we included the term 'predation'; if the search still returned more than 1,000 papers, we sorted these by relevance and limited our review to the first 1,000 papers. In total (from all our keyword searches) we identified >14,000 records (there were duplicates of papers among keyword searches; see Supporting Information Figure I. PRISMA flow chart). We then assessed each of these records by reading the title, abstract or full text and excluded all those that did not explicitly examine changes in population size over time (i.e. a change in the number of individuals within the population among generations). Papers were excluded if (a) they did not allow for changes in the population size because of survival and reproduction (e.g. adults or offspring were added or removed), (b) they did not allow recruitment to occur (e.g. ended the study prior to recruitment of offspring into the population), and (c) population size was manipulated in any manner among generations (e.g. experimentally maintained or altered population size at a particular number by removing individuals that recruited into the population). Thus, retained studies were only those that exposed a known number of individuals to a particular predation risk cue and allowed them to reproduce (and/or die if it occurred) and also allowed the subsequent offspring to grow and recruit into the population (or die) without any interference. Included studies therefore provided a measure of the number of individuals within the population over time across at least one generation. We also excluded studies that investigated the influence of predation risk on local population size (i.e. the number of individuals within a portion of habitat used by prey) due to dispersal and migration, as such studies measure changes in local population size but may not address population-level consequences due to scaling of the behavioural response experienced by the individuals exposed to risk (see Box 1 for discussion).

Our literature search identified only 10 studies that directly examined the effect of predation risk on prey population size, *none of which were performed in natural settings* (Table 1). In all of these studies, changes in prey population size was measured as changes in the number of individuals in the population across generations (over time). Thus, although many studies provide excellent evidence for risk effects on prey phenotype and fitness components (as illustrated in previous sections), the percentage of studies that examine the consequent effects of adaptive prey responses to population size is miniscule. The studies we found on population size can be divided into two broad categories: (a) those that examined the cost of risk-induced trait changes, by comparing risk exposed populations to populations with no risk exposure; and (b) those that examined the net effect (i.e. including both benefits and costs) of risk-induced trait changes, by comparing predator consumption of prey (i.e. direct killing) in populations where individuals had risk-induced trait changes versus populations where individuals did not have risk-induced trait changes.

Of the 10 studies, eight examined the influence of predation risk on prey population size, independent and isolated from CEs. Three of these studies examined risk effects on populations, allowing reproduction, mortality and recruitment to occur, but ending the experiments before the F1 generation could produce offspring (Table 1). In all three systems, predator-induced changes in reproduction and/or survival caused a reduction in population size. In 1.2 L containers in the laboratory, Bourdeau, Bach, and Peacor (2016) found that chemical cues from a predator reduced gut fullness and body condition of diaptomid copepods, which decreased fecundity and likely reduced survival, resulting in a large reduction in population size. In a closed field-system, Nelson, Matthews, and Rosenheim (2004) found that predation risk imposed by damsel bugs with surgically altered mouths reduced pea aphid population size by 30%. In 72 L tanks held outdoors, McCollum, Crowder, and McCollum (1998) found that the visual presence of predatory fish reduced reproduction and/or

survival of freshwater snails, thereby reducing the overall population size of snails by 50%.

The remaining five of these eight studies (that examined risk effects isolated from consumption) investigated risk over multiple generations and found that risk resulted in an increase, a decrease or no change in prey population size (Table 1). In single prey species studies, Hoverman and Relyea (2012) found that despite the expression of inducible anti-predator defences, predation risk did not affect freshwater snail population size over multiple generations, unlike the single generation study of McCollum et al. (1998). Boeing, Wissel, and Ramcharan (2005) found that after 10 days, risk-exposed *Daphnia* populations were smaller than control populations; however, by day 14 (end of the experiment) population size recovered and was similar between treatments. In two prey-species communities, Dawidowcz and Wielanier (2004) found that exposure to predation risk decreased multigeneration population growth and population size in *Daphnia*, but did not alter the population growth and size of the smaller sized *Ceriodaphnia reticulata*. Predation risk also altered the interaction between the two species when they were housed together. In the absence of fish kairomones *Daphnia* populations grew faster and dominated; however, in the presence of fish kairomones, *C. reticulata* outgrew and dominated the population of *Daphnia*. In multi-prey communities, Forbes and Hammill (2013) tested the effects of predation risk on protist community assemblages in either a 12:12 light:dark cycle or in continuous darkness. They found that predation risk did not alter community assemblages in the 12:12 LD regime. In continuous darkness, communities exposed to risk were different from control communities, but the magnitude and direction of changes in population size were species-specific (Forbes & Hammil, 2013). Peacor, Pangle, Schiesari, and Werner (2012) showed that the presence of fish kairomones affected zooplankton population size, negatively or positively depending on the species, in a mesocosm experiment that lasted at least three generations for all species.

We identified three studies that investigated predation risk effects on population size in the presence of CEs (Boeing et al., 2005 was included in the above findings and here; Table 1). They were able to accomplish this by comparing systems in which individuals did and did not have a phenotypic response to predation risk, and allowing consumption to occur. In these types of studies, the effect of predation risk is predicted to be qualitatively different than studies where risk effects are separated from predator consumption; rather than a negative prediction on fitness, a positive effect is predicted because the adaptive anti-predator response that is elicited is expected to increase fitness. Boeing et al. (2005) investigated both the costs and benefits of risk-induced trait changes using the *Chaoborus–Daphnia* system. They isolated *Daphnia pulex* clones that did and did not exhibit induced-trait responses and compared population size changes across three treatments: a control (no risk or predator), a risk treatment in which the predator was isolated from *Daphnia* and a predator treatment in which *Chaoborus* were freely swimming and could consume *Daphnia*. They found that induction of trait changes reduced *Daphnia* populations by 32% in the risk treatment; however, in the presence of the actual predator, those populations in which individuals had induced trait changes had 68% greater population growth than populations in which individuals were non-responsive. In a subsequent 5-week field enclosure experiment, Boeing and Ramcharan (2010) found that in the presence of freely swimming planktivorous fish predators, populations of responsive *Daphnia* clones had greater population size than non-responsive clones, which almost went extinct. van der Stap, Vos, Verschool, Helmsing, and Mooij (2007) used a similar design in which rotifers that did, and did not, express a phenotypic response to predator presence were compared (different species were used in this study, not different clones as in Boeing experiments); after many generations, populations of responding rotifers had >3-fold higher population size than non-responding rotifers.

The former eight studies, that directly assessed the effect of trait changes in the absence of consumption, illustrate that the impact of predation risk on population size can (a) be species- and context-specific, (b) have nonlinear temporal components (i.e. early reductions in population size may be compensated for over time), and (c) be reliant on the risk-induced changes in competitive ability of individual species and on community composition. The latter three studies we identified provide a great deal of insight into the net effect of risk-induced trait changes at the population level. Although implicit that adaptive induced-trait changes would provide a net benefit, evidence for this is difficult to obtain for many taxa because the relative benefit to cost of responding versus not responding must be compared. Further, the goal of many studies is to identify the cost of predation risk on prey and, thus, investigate the cost of risk-induced trait changes in environments in the absence of consumption. Clearly, the ultimate goal would be to elucidate the influence of NCEs and CEs to the net influence of free predators; i.e. given the influence of a predator on a natural population, what are the contributions of NCEs and CEs to the net effect? Whereas some studies have examined how NCEs and CEs affect demographic rates (e.g. birth rates and mortality, Pangle et al., 2007), we are unaware of studies that have disentangled these factors on the net effect of a predator on prey population size. Overall, given the surprising paucity of studies examining risk effects on population size, it is clear that much empirical work is still needed.

4 | CHALLENGES TO PREDICTING RISK EFFECTS; FROM PREY TRAITS TO POPULATION SIZE

There is much evidence that NCEs are operating in natural systems, yet key challenges in predicting how risk effects will impact prey responses and population size in natural systems remain. We still need to understand, first, the complex nature of how ecological and environmental context interacts with risk effects, prey responses and their fitness costs (Figure 1i; Transition 1 and 2), and second, the potential feedbacks within the population itself (Figure 1ii; Transition 3). Furthermore, given that consumption is rarely incorporated into

risk-effect studies, the relative strength of CEs versus NCEs and the actual benefits and costs associated with prey responses are difficult to discern. Here we discuss these limitations and challenges across the transitions between levels in the translation from induced trait changes to population size changes (Figure 1).

4.1 | Ecological and environmental context (Transition 1 and 2; Figure 1)

4.1.1 | Different phenotypic responses to the same number of predators

Predicting the response of prey to predators can be difficult because many factors influence the nature and magnitude of the response making it highly context dependent. Even at the same density of predators, prey responses may differ because of factors influencing the risk imposed by the predators. For example, abiotic factors, such as moonlight, are well known to alter small mammal responses to predation risk (e.g. Upham & Hafner, 2013), and both landscape features and refuge availability have been shown to influence the response of a number of prey (Donelan et al., 2017; Orrock et al., 2008; Thaker et al., 2011). Prey density, of conspecifics or alternate prey, can also influence the predation risk posed by the same number of predators. Overall high prey density (regardless of species) may reduce individual response to the risk of predation because higher prey density reduces the risk to each individual (Peacor, 2003; Tollrian, Duggen, Weiss, Laforsch, & Kopp, 2015). It follows that it is adaptive for prey species to use intraspecific and interspecific conspecific density to gauge the level of predation risk. Alternatively, prey species at relatively low density, compared to alternate prey species, may not respond strongly given that their realized risk of predation is low as predators focus on the more abundant prey (Fortin et al., 2015; Norrdahl & Korpimäki, 2000).

4.1.2 | Different phenotypic responses to the same level of risk

Not only do prey respond differently to the same density of predators (or predation cue), but prey may also respond differently to the same level of risk. For example, prey state can influence the response to the same level of risk, and those individuals in the 'best' condition may have the strongest response (Luttbeg & Sih, 2010; Matassa & Trussell, 2014), or those buffered during development (e.g. matrotrophic fish and not lecithotrophic fish) may have the least response (Mukherjee et al., 2014). Similarly, resource availability is well known to alter prey response, with prey in areas of high food availability predicted to respond much greater than those in low food areas, given their ability to gain excess reserves in times of safety (Sih & McCarthy, 2002). The type of predator, again even if posing the same risk, can also change prey responses. For example, sit-and-wait ambush predators may elicit different responses than cursorial, pursuit predators, even if the actual risk of death imposed by the different predators is the same (e.g. Eklöv & VanKooten, 2001; Losey & Denno, 1998; Schmitz, 2008; Thaker et al., 2011).

Whereas there are many empirical examples to support the fact that prey response is highly context-specific, there are also many theoretical examples showing times when prey should have different responses to the same number of predators or the same level of risk. For example, Abrams (1991) shows that iteroparous and semelparous prey will respond differently, and Gilliam's u/g theory (1982), which describes phenotypic responses in the face of a predation risk-energy gain trade-off predicts different responses depending on the life history stage of the organism (Gilliam & Fraser, 1987).

4.1.3 | Different costs of the same phenotypic response to predators

Context can further influence the costs and outcome of responses to predation risk; i.e. not only can context affect prey's phenotypic response, but context can influence the outcome of equivalent responses to predation risk. For example, although often assumed (and shown) that risk-induced reductions in foraging reduced growth of prey, Turner (2004) and Peacor and Werner (2004) showed that at low resource availability and/or high prey population density, risk-induced reductions in foraging can have little cost to prey because most resources were consumed regardless of foraging intensity. Additionally, risk-induced reductions in foraging rate can have a net positive effect on prey growth rate when indirect positive effects on resources are large due to nonlinearities in the relationship between resource growth rate and resource level (Peacor, 2002). Indeed, the direct negative and indirect positive effects on prey growth rate can change differentially through time, leading to predator effects on prey size changing from negative, to absent, to positive (Boeing et al., 2005; Werner & Peacor, 2006).

The presence of competitors and alternate prey can also strongly influence the cost of a prey's phenotypic response; i.e. at the same level of risk and the same level of response, the cost of the response may depend on intraspecific or interspecific competitor density. For example, using experimental mesocosms, Peacor and Werner (1997) found that predation risk of caged *Anax*, dragonfly larvae, greatly reduced the mass gain of small green frog and small bull frog larvae; effects which were exacerbated in the presence of a much better competitor, large bull frog larvae. This is due to the simple reason that a non-responding competitor continues to forage and remove resources at the same rate, leaving fewer resources for the reduced-foraging responding prey. Prey response to predation risk can also differentially influence the competitive ability of competing species, resulting in unpredictable outcomes from dyadic species interactions alone. For example, Dawidowcz and Wielanier (2004) found that fish kairomones reversed the competitive advantage of large-sized *Daphnia hyaline* over small-sized *C. reticulata*, resulting in a relatively higher population growth rate and resulting increase in population size of *C. reticulata*. In the absence of competition, fish kairomones had no effect on *C. reticulata* population size, and reduced *Daphnia* population size (see also Relyea, 2000 for example on risk-induced competition reversal).

4.2 | Compensatory population feedbacks (Transition 3; Figure 1)

Classic ecological theory illustrates a number of reasons why risk-induced changes in fitness components may not translate to affect population size in significant ways (Hutchinson, 1978; Krebs, 1994). For example, reduced reproduction may not affect population size if recruitment (juvenile survival), not fecundity (births), is limiting (Hutchinson, 1978; Krebs, 1994; Figure 2). Furthermore, risk-induced changes in reproduction or mortality, must be additive, and not simply compensatory, for those demographic effects to scale up to alter population size (Figure 2). For example, in a simplistic comparison, prey that produce many offspring with low juvenile survival (e.g. species with type III survivorship curves) may be able to suffer severe risk-induced reductions in reproduction with little effect on overall population size, assuming that the risk-induced reduction in number of offspring produced is compensatory to the low juvenile survival rate and not additive to such mortality. But this form of overcompensation in reproduction is not found in all species, and in those that produce few offspring with high juvenile survival (e.g. species with type I survivorship curves), we expect the opposite.

Changes in the magnitude of risk effects will likely also change as kill rates increase and may contribute proportionally less to population size changes as CEs become greater. Creel (2011) showed that risk effects likely increase in strength as kill rates increase, but only when kill rates are compensatory to other forms of mortality. When CEs become additive (i.e. are responsible for reduction in population size), risk effects may not change in magnitude because organisms have already responded maximally.

4.3 | Limitations of short-term experiments

Theoretical studies suggest that inferences from short-term experiments, using limited predator and prey taxa (e.g. dyadic predator–prey experiments), may overstate long-term effects on population size of free-living prey. Persson and De Roos (2003) argue that the expectation of strong long-term effects of risk at the population level is a misrepresentation of what can be inferred from short-term experiments. Using a physiologically structured model to examine risk effects in a fish system in which mortality and behaviour are size dependent, they found that although risk effects were large in short-term experiments, these effects were not important in long-term population dynamics because of strong multigenerational feedbacks from consumptive (i.e. density-mediated) effects. Luttbeg et al. (2003) reached similar conclusions from another model that incorporated size-dependent mortality and risk. Their model showed that although risk effects could be strong at certain points in the season, the longer-term effects on the population were much smaller due to behavioural compensation in foraging at different periods in the season. Abrams (2008) also found that large risk effects on prey population size may decrease over time. Abrams (2010) further argued that, due to alternate prey and apparent competition, predation risk may have a much smaller effect on population dynamics within large food webs compared to systems with one or a few prey that are typical of most empirical studies. Experiments have shown that risk effects can vary greatly when additional species are present in short-term experiments (as discussed above), but we are unaware of studies that have examined the role of system complexity on long time-scales or in natural systems.

4.4 | The lack of consumptive effects in studies of risk effects

The majority of studies examining risk effects do so in the absence of consumption from predators. These studies are specifically designed to take a detailed look at the potential costs associated with predation risk, and not the potential benefit. For example,

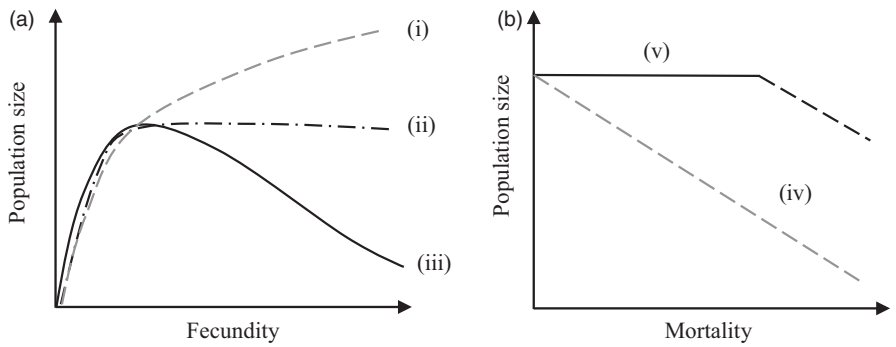

FIGURE 2 Classic ecological models illustrate conditions under which changes in fecundity and mortality will not alter population size. (a) Risk-induced decreases in fecundity may (i) reduce population size if reproductive output is directly related to recruitment (e.g. reducing the number of juveniles reduces the number of recruits), (ii) not change population size if there is a set number of juveniles that can recruit into the population, (iii) increase population size if reducing the number of juveniles reduces competition and increases juvenile or adult survival (adapted from stock-spawning models of (i) Shepherd–Cushing, 1980, (ii) Beverton–Holt, 1957, (iii) Ricker, 1954); (b) Risk-induced changes to mortality may alter population size if the effects are (iv) additive (hashed lines), but not if they are (v) compensatory (solid line; Anderson & Burnham, 1976; Nichols, Conroy, Anderson, & Burnham, 1984)

many studies have shown that risk exposure, acting via maternal physiology and behaviour, can reduce reproduction (e.g. Sheriff et al., 2009; Zanette et al., 2011). However, these studies compare reproduction in environments without consumption and thus cannot measure the associate benefit of the maternal response to risk exposure, nor the cost to her reproduction if she did not respond. Abrams (2010) shows that this approach can greatly misrepresent the influence of risk effects. In his paper, the influence of the risk effect is strongly dependent on whether consumption from the predator is included in the experimental design or not. Yet, for many systems it is difficult, if not impossible, to compare individual fitness of risk-responding and non-responding phenotypes during predator exposure (i.e. allowing consumption to occur). Potentially, future studies could compare the fitness of individuals that have been either exposed or not exposed to predation risk during temporally limited consumption trials or predator removal trials, as has been done in aquatic but not terrestrial systems. Experiments could also intensify predation risk cues to increase the perception of risk in natural systems where a known level of consumption occurs. Understanding the relative effects and strength of CEs versus NCEs on population size is an important area of need.

5 | CONCLUSIONS

There is much recognition in the need to understand the pervasive role predators play within ecosystems, and the impact of potential reintroductions or losses of these animals (Atwood et al., 2015; Estes et al., 2011; Ritchie et al., 2012). As such, it is important that we appreciate not only the CEs of predators, namely the killing of prey, but also the NCEs of predation risk (Lima, 1998). Whereas the mechanisms that underlie NCEs on prey population size have been commonly demonstrated across myriad systems, our surveys demonstrate a major disconnect in ecologists' claims of the extent of NCEs on population size and the actual evidence for this effect. Although we are gaining a better understanding of how predation risk may alter prey traits and fitness components, we still lack empirical evidence to support predictions on how predation risk may alter prey population size. We suggest three avenues for future research.

1. We need to better understand the progression from predator-induced trait changes in prey to prey population size, and the contexts in which risk-induced changes at one level may or may not translate to changes at the next.
2. Our understanding of risk-induced effects on prey populations has been generated from studies of a limited number of species (almost exclusively invertebrates and larval amphibians) across short time intervals. We suggest the need to examine such effects across a broader range of taxa over multiple generations.
3. Given the complexity of species interactions and feedback mechanisms, it is critical that more studies examine these effects in free-living systems to determine how strong population-level responses can be.

It is clear that we still fail to fully understand the ways in which risk-induced trait changes affect fitness components and population growth rate; and whether these individual-level responses scale to influence population size and dynamics. We expect that as more studies examine the predation risk–prey population size nexus, the variable and dynamic nature of this relationship will become more evident.

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AUTHORS' CONTRIBUTIONS

All authors contributed substantially to the concepts and revising of the manuscript; M.J.S. led the writing. All authors approved the final version for publication.

DATA AVAILABILITY STATEMENT

We will not be archiving data because this manuscript does not use data.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

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