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Effects of predation risk on parasite–host interactions and wildlife diseases

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Abstract

Landscapes of fear can determine the dynamics of entire ecosystems. In response to perceived predation risk, prey can show physiological, behavioral, or morphological trait changes to avoid predation. This in turn can indirectly affect other species by modifying species interactions (e.g., altered feeding), with knock-on effects, such as trophic cascades, on the wider ecosystem. While such indirect effects stemming from the fear of predation have received extensive attention for herbivore–plant and predator–prey interactions, much less is known about how they alter parasite–host interactions and wildlife diseases. In this synthesis, we present a conceptual framework for how predation risk—as perceived by organisms that serve as hosts—can affect parasite–host interactions, with implications for infectious disease dynamics. By basing our approach on recent conceptual advances with respect to predation risk effects, we aim to expand this general framework to include parasite–host interactions and diseases. We further identify pathways through which parasite–host interactions can be affected, for example, through altered parasite avoidance behavior or tolerance of hosts to infections, and discuss the wider relevance of predation risk for parasite and host populations, including heuristic projections to population-level dynamics. Finally, we highlight the current unknowns, specifically the quantitative links from individual-level processes to population dynamics and community structure, and emphasize approaches to address these knowledge gaps.

KEYWORDS
ecology of fear, nonconsumptive effects, parasitism, predation risk, trait-mediated indirect effects

INTRODUCTION

Anyone who has seen movies such as *Jaws*, *Lake Placid*, *Piranha*, or *Anaconda* knows about the visceral fear that voracious predators can induce in their potential victims. While this might seem like an unpleasant but harmless experience, science has increasingly realized that the fear of predators can have significant consequences not only

for scared prey but also for the wider community around them. In response to perceived risk of predation, the prey of many species can undergo physiological, behavioral, or morphological trait changes that reduce their predation risk (Lima, 1998; Sheriff & Thaler, 2014; Werner & Peacor, 2003). These energetically costly defenses by fearful prey can, in turn, negatively affect their fitness and abundance (Peacor et al., 2020; Werner & Peacor, 2003).

The resulting changes in prey traits can also indirectly affect other species via species interactions, with secondary knock-on effects for the community and the wider ecosystem, for example, through trophic cascades (Peacor et al., 2020). Such wide-ranging effects through a phenomenon coined the "ecology of fear" first gained attention following the reintroduction of wolves to Yellowstone National Park (Ripple & Beschta, 2004; Wirsing & Ripple, 2011). In response to wolf presence, large grazing ungulates adjusted their habitat use and foraging patterns, which led to reduced grazing pressure and subsequent increases in the recruitment of woody plants, particularly along riverbanks. This in turn led to the recovery of riparian functions and recolonization of beavers (Ripple & Beschta, 2004; but see Kauffman et al., 2013). These complex, knock-on effects stemming from perceptions of predation risk, yet extending to species interactions, are not unique to terrestrial predator–herbivore–plant systems and have also been documented in aquatic systems, for example, in sharks that prey on herbivore dugongs in marine waters, which may in turn affect seagrass meadows through altered dugong foraging (Wirsing & Ripple, 2011).

These manifold indirect effects of perceived predation risk on herbivore–plant and predator–prey interactions have received extensive attention (e.g., see reviews by Lima, 1998; Peacor et al., 2020; Sheriff & Thaler, 2014; Werner & Peacor, 2003). In contrast, much less is known about how the fear of predation can indirectly affect another common type of species interaction, namely, that between parasites and hosts. All animal species serve as hosts to parasites, ranging from microparasites such as viruses and bacteria to macroparasites such as helminths and arthropods (Goater et al., 2013). Hence, predator-induced changes in prey traits may also be relevant for their function as hosts to parasites. Specifically, some trait changes, such as altered behavior or physiology, may affect the various defense mechanisms that hosts have evolved to either avoid becoming infected or deal with infections by resistance or tolerance mechanisms (Buck et al., 2018; Råberg et al., 2009), and this in turn may decrease or increase disease risk.

Hosts that change their behavior as a response to perceived predation risk by moving to different habitats, or changing their feeding rates or activity levels, may generally be exposed to different species or numbers of parasites (Binning et al., 2017; Hoverman & Searle, 2016; Shaw & Civitello, 2021). As a case in point, tadpoles increase their swimming activity in the presence of trematode (flukes) cercariae to avoid infections; however, when predation risk cues are present, the tadpoles reduce this activity to avoid predation, which results in increased infections (Koprivnikar & Urichuk, 2017; Thiemann & Wassersug, 2000). Physiological changes such as increased stress levels of hosts in response to perceived predation risk may further increase their susceptibility to infections or affect their postinfection defenses due to immunosuppression (Shaw & Civitello, 2021; Sheldon & Verhulst, 1996).

Predation risk-induced trait changes in prey thus have the potential to trigger manifold indirect effects on parasite–host interactions. These are part of a wider range of predator effects on parasite–host interactions, including selective predation on infected hosts and free-living infective stages (Lopez & Duffy, 2021). Predator effects on parasite–host interactions resulting from actual predation (consumption) of hosts or parasites, that is, density-mediated indirect effects (Figure 1a), have been comprehensively covered in recent reviews (Johnson et al., 2010; Koprivnikar et al., 2023; Lopez & Duffy, 2021; Shaw & Civitello, 2021). In contrast, nonconsumptive effects of predators that lead to trait-mediated indirect effects on parasite–host interactions (Figure 1b) have attracted relatively little attention.

Recent reviews have also considered that parasites themselves are capable of inducing indirect effects, either through consumption, that is, infection (Figure 1c; Buck, 2019; Hatcher & Dunn, 2011), or via nonconsumptive effects if a perceived threat of infection causes changes in host traits that have repercussions for parasite–host interactions (Figure 1d; Buck et al., 2018; Buck, 2019; Buck & Ripple, 2017; Daversa et al., 2021; Koprivnikar et al., 2021; Weinstein et al., 2018). However, there remains a knowledge gap with regard to potential nonconsumptive effects of predators on parasite–host interactions (Figure 1b). In fact, one of the outstanding questions posed by Lopez and Duffy (2021) was about other mechanisms by which predators could mediate host–parasite interactions in their prey. Consequently, a comprehensive synthesis of nonconsumptive effects of predators on parasite–host interactions would serve to highlight this critical component of general predator influences and prompt more studies in this direction.

In this synthesis, we present a conceptual framework of how predation risk perceived by wildlife hosts can affect parasite–host interactions through various pathways, with implications for infectious disease dynamics. We base our approach on recent conceptual advances regarding predation-risk effects (Peacor et al., 2020) with

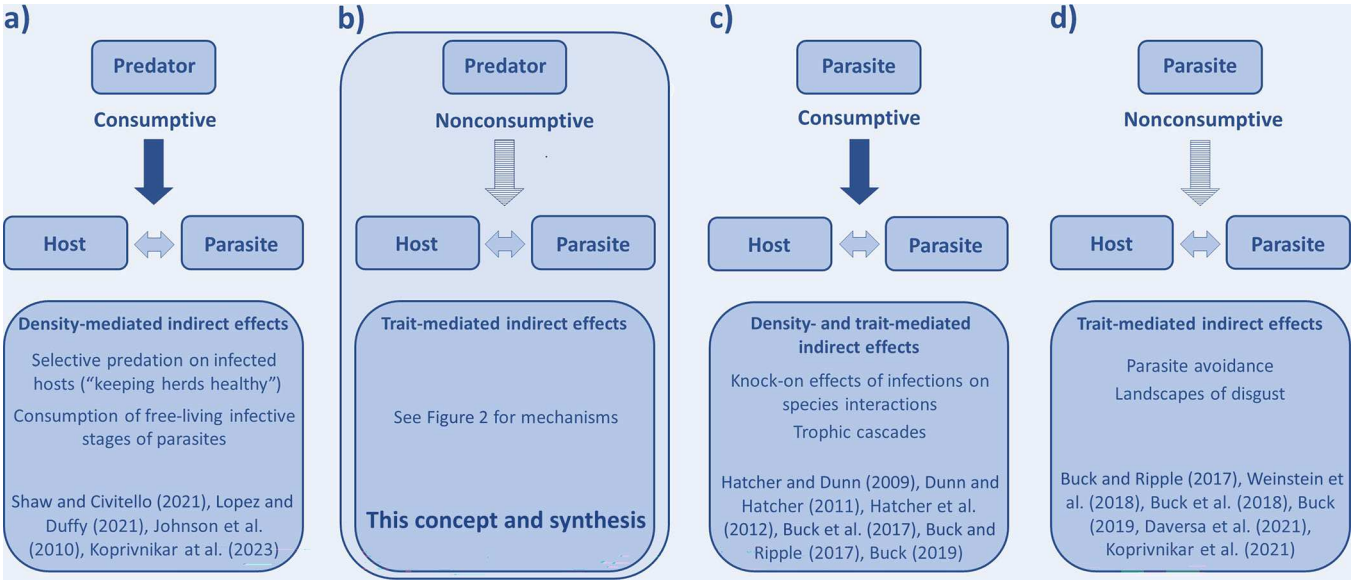

FIGURE 1 Scope and concepts of current synthesis in the context of related topics and previous reviews. While consumptive effects of predators on parasite–host interactions have been covered by several previous reviews (a), a comprehensive synthesis of nonconsumptive effects of predators on parasite–host interactions is missing to date (b). Similar to predators, parasites can also exert consumptive (c) and nonconsumptive (d) effects on hosts, with repercussions for parasite–host interactions. Boxes include examples of trait- and density-mediated effects via the different mechanisms covered by previous in-depth reviews and references therein.

the aim of expanding this general framework to include parasite–host interactions and diseases. By synthesizing key findings from the available literature, we identify critical pathways by which parasite–host interactions could be altered by predation-risk effects and discuss their wider relevance for parasite and host populations, as well as areas in need of further research. Throughout this synthesis, we use the term parasite in an ecological sense, that is, denoting a species interaction between two organisms in which the parasite gains energy and habitat at the expense of the host (Combes, 2001), including classical parasitic groups, such as helminths, and microparasites, such as viruses and bacteria that are often categorized as pathogens. By definition, parasites always have negative effects on their host, but not all parasites necessarily cause disease in the form of organismal malfunctioning with veterinary or conservation relevance. However, in these latter cases, any changes in parasite–host interactions can be expected to potentially affect the disease risk of hosts that are scared by predators.

PREDATION RISK-INDUCED CHANGES IN PREY TRAITS

Organisms can respond to a perceived risk of predation with a diversity of trait changes intended to reduce the threat of injury or death (Figure 2; for reviews see Lima, 1998; Sheriff & Thaler, 2014; Werner & Peacor, 2003). These changes can include behavioral, morphological, physiological, or life-history traits that are phenotypically flexible within individual organisms (Figure 2). Trait changes in response to predation risk can occur on different time scales, ranging from short-term responses, such as moving away from predator cues, to long-term responses, such as increasing body size or maturing earlier. For example, mollusks can quickly react to predator cues by closing their valves, retracting into their shells (Kulakovskii & Lezin, 2002; Naddafi et al., 2007; Smee & Weissburg, 2006), or by escaping into more sheltered microhabitats (Eschweiler & Christensen, 2011; Jacobsen & Stabell, 1999; Kobak & Kakareko, 2009). On longer time scales, in turn, they can grow thicker shells (Freeman & Byers, 2006). In addition, many physiological trait responses can be induced by predation risk in a broad array of organisms, for example, in the form of increased respiration or elevated glucocorticosteroid levels to support greater vigilance and escape behaviors (Hawlena & Schmitz, 2010). Finally, predation-risk responses can also include life-history shifts. For example, larval stages of amphibians and insects can escape predation in their juvenile environment by metamorphosing sooner and at smaller body sizes (Benard, 2004; Hite et al., 2018).

A variety of indirect effects can arise from these trait changes of prey in response to predation risk (Figure 3). Based on the conceptual framework and standardized terminology proposed by Peacor et al. (2020), all such predation-risk effects are based on two fundamental

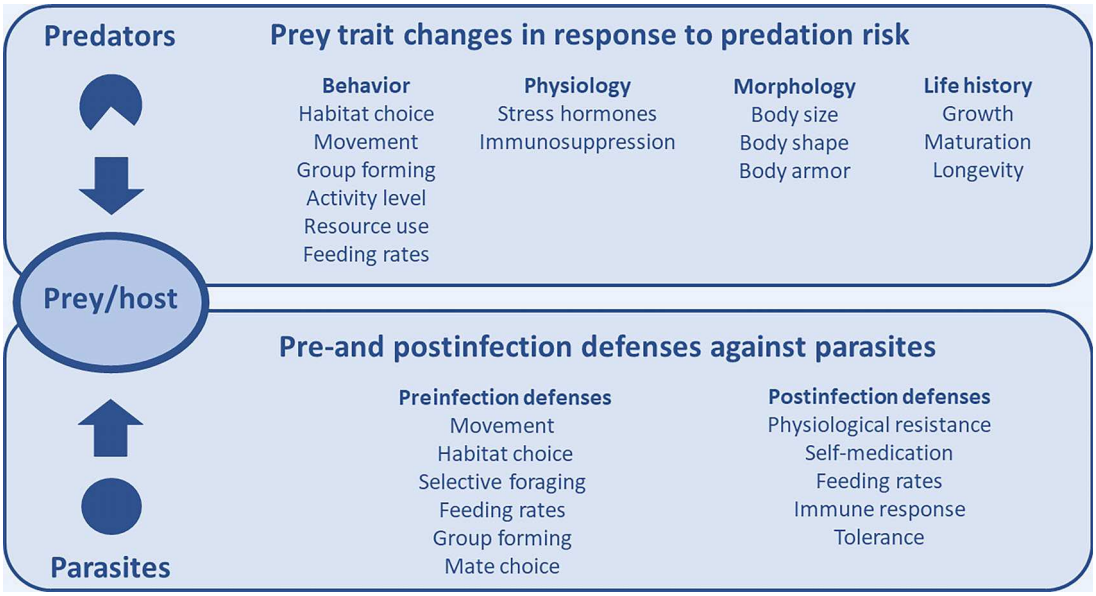

FIGURE 2 Different types of trait changes in prey in response to perceived predation risk by predators and host defenses against parasite infections. For details see text.

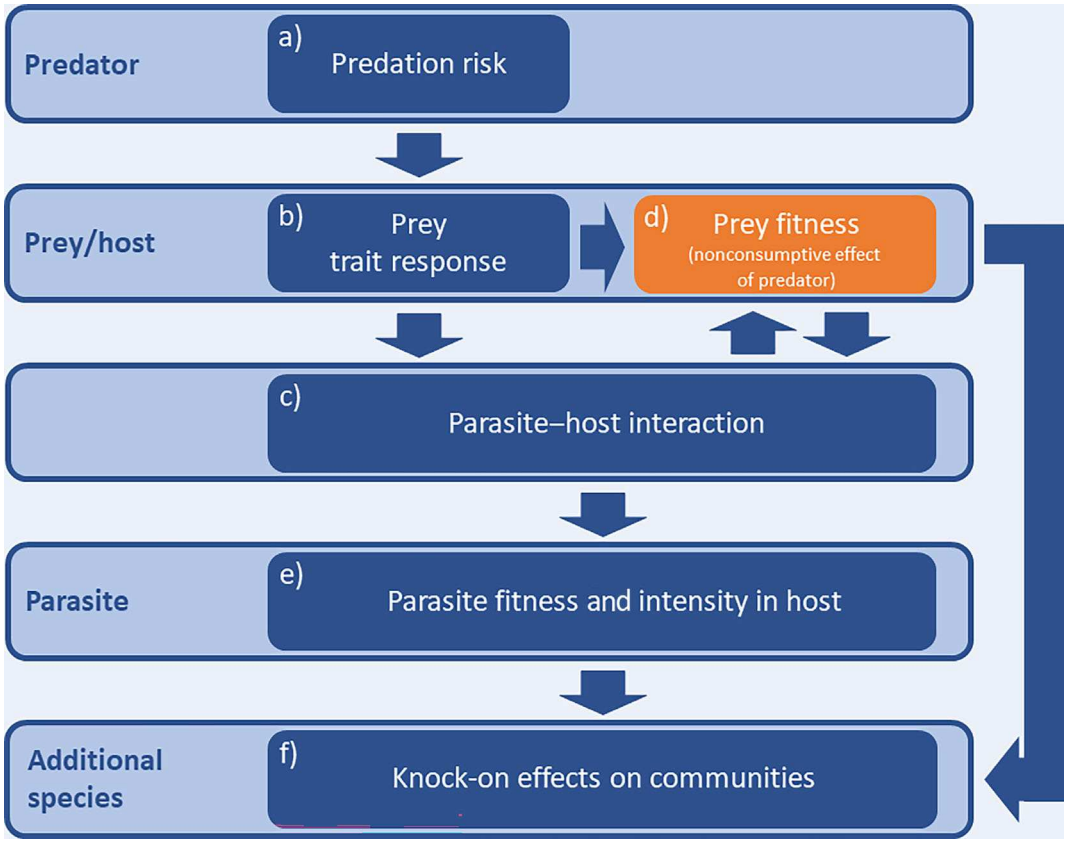

FIGURE 3 Conceptual framework illustrating how the perception of predation risk by prey/hosts can alter parasite–host interactions and have knock-on effects on the wider community (based on Peacor et al., 2020). All effects resulting from predation risk-induced trait change responses of prey (b) constitute trait-mediated indirect effects of predators. Potential changes in prey fitness (d) are considered to be nonconsumptive predator effects on prey (orange) in the strict sense (sensu Peacor et al., 2020). Note that all pathways shown act on individual prey/hosts. Population-level effects, both for parasites and hosts, may result from some of these pathways but are treated in a separate section.

factors. First, the threat of predation usually varies, in both space and time, and many organisms can detect the presence of predators via visual or olfactory cues (Figure 3a). Second, a perceived risk of predation can trigger a variety of trait changes in prey as they endeavor to avoid harm or death, as discussed previously (Figure 3b). It is important to note that these changes result from traits being flexible rather than fixed; that is, the trait changes in question are phenotypically plastic within an individual and not mean trait changes in a population (i.e., resulting from natural selection—Peacor et al., 2020).

PREDATION-RISK EFFECTS ON PARASITE–HOST INTERACTIONS

When prey that respond to predation risk also serve as hosts to parasites, we propose that this can alter parasite–host/prey interactions and wildlife diseases through pathways similar to those observed for predator–prey and herbivore–plant interactions (Figure 3c–f). This is partly because the antiparasite defenses usually exhibited by hosts may be affected by trait changes stemming from their perceived predation risk (Figure 2). Due to the typically negative fitness consequences of infections, many organisms have evolved a wide array of defenses against parasites, including preinfection and postinfection mechanisms. Preinfection mechanisms include avoidance behaviors that lower exposure to infections and can involve evasive or reduced movements to avoid encounters, spatiotemporal dodging through habitat choice and selective foraging, diel and seasonal migrations, and social behaviors such as aggregation and mate choice (see reviews by Behringer et al., 2018; Buck et al., 2018; Koprivnikar et al., 2021; Thieltges & Poulin, 2008).

Once exposed, hosts can still defend themselves through resistance mechanisms that clear parasites or prevent them from establishing successful infections. This can include behavioral and physiological components, such as grooming to remove parasites, self-medication (e.g., through consumption of medical plants) to treat infections, and the mounting of short- or long-term immune responses. Hosts can also tolerate infections in the form of various coping mechanisms (Råberg et al., 2009; Sheldon & Verhulst, 1996), including "sickness behaviors" such as lethargy that conserve host energy (Adelman & Hawley, 2017) and illness-mediated anorexia (Hite et al., 2020). Importantly, we note that many of these antiparasite defenses may be affected by general trait changes in response to predation risk. In addition, some of the general trait changes in prey may also affect the general function of prey as a host, for example, by altering the resources available to parasites after infection. Because trait changes in prey/hosts in response to predation risk may also alter parasite–host interactions and infectious disease dynamics, as well as other species interactions, these must be considered in addition to direct/consumptive effects of predators on host parasitism (reviewed by Lopez & Duffy, 2021; Shaw & Civitello, 2021). We discuss host defenses in more detail below in terms of how these may specifically be affected by trait changes in response to predation risk.

A FRAMEWORK FOR CHARACTERIZING PREDATION-RISK EFFECTS ON PARASITE–HOST INTERACTIONS

Building on the conceptual framework and standardized terminology for predation-risk effects proposed by Peacor et al. (2020), we developed an expanded version that conceptualizes and integrates different mechanisms that may drive predation-risk effects on parasite–host interactions (Figure 3c–f). These arise when predation risk-induced trait changes in prey also affect their function as a host (interaction modification sensu Peacor et al., 2020) (Figure 3c). This interaction modification can in turn affect the fitness of prey, for example, by lowering or increasing infection burden in a manner that negatively affects their survival and/or reproduction (Figure 3d). Prey fitness can also be affected by the trait changes resulting from perceived predation risk, for example, in cases where risk-induced trait responses are energetically costly (Figure 3b). Both pathways lead to nonconsumptive effects in the strict sense (sensu Peacor et al., 2020) of predators on their prey, which are basically indirect effects of predators on their prey compared to their direct consumptive effects (Figure 3d).

Predation risk-induced changes in parasite–host interactions not only affect prey/hosts but can also affect the fitness of parasites and their infection intensity in hosts. In other words, they affect a third species (the parasite) in addition to the predator and the prey (Figure 3e). Those effects can have further consequences for additional species in the community, for instance, when the parasites also infect other host species (Figure 3e). Such knock-on effects on the wider community may also result from the various predation risk-induced effects on the fitness of prey/hosts (Figure 3d), especially for species with important ecological functions such as ecosystem engineers (Figure 3e).

As outlined earlier, predation risk-induced trait changes in prey that affect their function as a host are at the foundation of the various possible trait-mediated

indirect effects of predators on parasites and diseases that we suggest here. In general, there are four crucial steps in the life cycle of parasites by which parasite–host interactions can be affected if prey that also serve as hosts show trait changes in response to the threat of predation (Figure 4). First, behavioral antipredator responses of prey, such as avoiding specific habitats or adjusting activity and feeding levels, may alter the frequency with which parasites encounter potential hosts. Second, once a host encounters parasite infective stages, predation risk-induced behavioral, physiological, or morphological trait changes in hosts (e.g., immunosuppression due to enhanced stress levels or reduced grooming) may affect parasite establishment success. Similarly, in the third step, these host trait changes may affect the persistence of infections, as well as parasite virulence and pathogenicity in hosts once infected. Finally, the amount or rate of parasite propagule production may also be affected by the same host trait changes or by life history adjustments, such as host longevity. Any changes in parasite–host interactions at one of these four crucial steps in parasite life cycles due to predation risk-induced trait changes in hosts can thus trigger the diverse indirect effects of predators on parasites, hosts, and other species outlined above.

Although research on predation-risk effects on parasite–host interactions remains in its relative infancy, the few existing studies that have investigated this suggest not only that such effects exist but that they may have important repercussions for a wide range of parasites and diseases. In the following subsections, we synthesize key findings from those studies with the aim of characterizing the various mechanisms associated with the steps highlighted in Figure 4.

Host encounter

The first step in which predation risk-induced changes in host traits can affect parasite–host interactions is the initial contact between a parasite and its host. This is a critical phase because parasite avoidance is generally considered less costly than resistance or tolerance (Gibson & Amoroso, 2022; Hart, 1990; Hart & Hart, 2018). Consequently, many predator-induced trait changes on the host side can increase or decrease the likelihood that parasites will encounter potential hosts and thus alter parasite–host interactions. For example, changes in habitat use in response to predation risk can increase host contact with infective stages of parasites. This has been observed in coastal gastropods seeking refuge in the high intertidal from predation pressure by subtidal crab and starfish, but this in turn exposes them to more trematode parasites (Byers et al., 2015). Likewise, while the negatively phototactic behavior of some *Daphnia* water flea clones in response to the presence of planktonic predators leads to lower predation, this habitat shift also leads to higher exposure to parasite spores from pond sediments, leading to elevated infection levels (Decaestecker et al., 2002).

In addition to habitat shifts, social antipredator responses, such as group forming, can also have indirect effects on host encounter rates of parasites. For example, shoaling in guppies reduces predation risk; however, at the same time, group forming leads to higher exposure of uninfected fish to conspecifics infected with monogeneans (directly transmitted ectoparasitic flatworms), thereby increasing parasite transmission and infection levels in fish (Stephenson et al., 2015; Walsman et al., 2022).

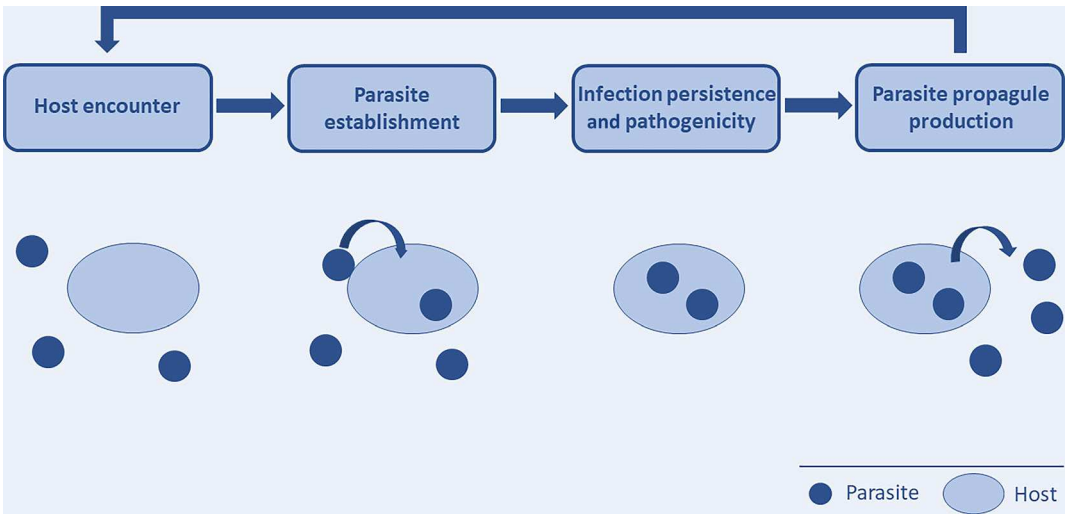

FIGURE 4 Framework for characterizing predation-risk effects on parasite–host interactions along four crucial steps in the life cycle of parasites where parasite–host interactions can be affected by predation risk-induced changes in traits of prey that also serve as host. For details see text.

Other common antipredator responses, such as seeking refuge or reducing activity levels, can also indirectly affect host encounter rates with parasites. For example, lower activity of tadpoles as an antipredator response when exposed to predator cues leads to higher exposure to trematode parasites (Koprivnikar & Urichuk, 2017; Thiemann & Wassersug, 2000). Likewise, the positioning of marine mussels within oyster beds can help protect them from crab and bird predators but lead to elevated infection from trematode parasites that emerge from snail hosts living inside the oyster beds (Goedknegt et al., 2020). At the same time, however, this antipredator behavior leads to reduced infection levels with endoparasitic copepods in mussels, probably due to oysters diluting infective stages settling from the water column into the beds, indicating that the direction and strength of predation-risk effects on host encounter rates depend on the specific parasite–host system (Goedknegt et al., 2020). Finally, morphological trait changes can affect host encounter with parasites, as observed in water fleas that increase their body size in response to predator cues. This leads to elevated feeding rates at larger body size and a higher exposure to parasite spores in the water column (Duffy et al., 2011).

Parasite establishment

Once parasites encounter a host, some of the physiological, behavioral, or morphological trait changes in response to predation-risk cues may affect the successful establishment of parasites in/on hosts. In many prey species, various stressors, including the perception of predation risk, can lead to physiological responses in the form of elevated levels of certain hormones, particularly glucocorticoids such as cortisol and corticosterone (Adamo et al., 2017; MacDougall-Shackleton et al., 2019). Those increased glucocorticoid levels can be associated with immunosuppression (Romero, 2004; Sapolsky et al., 2000), which may affect the establishment of parasites in their hosts (Adamo et al., 2017). For example, larval salamanders that are exposed to predator cues are more susceptible to virus infections, presumably resulting from glucocorticosteroid-mediated immunosuppression (Kerby et al., 2011). Similarly, sparrows exposed to predator clues not only showed lowered T-cell–mediated immune responses but also increased prevalence and intensity of *Haemoproteus* blood infections (Navarro et al., 2004), and exposure of Wistar rats to olfactorial predator clues resulted in immune suppression and higher burdens of nematode parasites (Horak et al., 2006).

Parasite establishment in hosts can also be affected by changes in host behaviors in response to predation-risk clues. For instance, in bivalves, cues of crab predators lead to closure of the valves and cessation of filtration activity (Cornelius et al., 2023). Because filtration is the main route through which bivalves are exposed to trematode infections, this antipredator response leads to a concurrent reduction of infection levels (Cornelius et al., 2023).

Infection persistence and pathogenicity

Once hosts are infected, trait changes in them in response to predation risk may affect the persistence and pathogenicity of infections through host resistance and tolerance mechanisms. Interestingly, the responses of prey to predator cues can sometimes increase immune function (Adamo et al., 2017; Duong & McCauley, 2016), with implications for infection resistance. As an example, predation cues from cannibalistic conspecific larval dragonflies cause an increased melanization response to simulated parasites in the form of injected monofilaments (Murray et al., 2020). This indicates that host trait changes in response to predation risk do not always benefit parasites; in fact, in some cases, responses to predators could have a dual benefit to prey as they also defend against parasitism. For example, predator-induced acceleration of development and early metamorphosis such as observed in amphibians (Benard, 2004; Hite et al., 2018) may also lower their exposure to aquatic parasites.

Predators can also affect host behaviors relevant for parasite persistence. For instance, female impalas decreased the time spent grooming their young when in a state of heightened alertness due to the presence of predators, which likely results in a reduced removal of ectoparasites such as ticks from calves (Blanchard et al., 2017). However, in cases where parasites manage to persist, predation risk-induced trait changes in their hosts may also affect the pathogenicity of parasites and the ability of hosts to tolerate infections. For instance, in the case of larval salamanders that are more susceptible to virus infections when exposed to predation risk cues, the same glucocorticosteroid-mediated immunosuppression mechanisms are probably also responsible for a higher mortality of infected hosts when predation-risk cues are present (Kerby et al., 2011). In another example, mosquitoes exposed to predator cues as larvae showed reduced survival when infected with a parasitic fungus as adults (Ong'wen et al., 2020). Finally, predation risk-induced effects on the pathogenicity of parasites have been observed as a result of shoaling in guppies where the increased host contact rates due to shoaling lead to an increase in multiple infections with different parasite genotypes. The resulting parasite genotype

competition, in turn, selects for higher virulence, thereby leading to nonconsumptive effects of predation on virulence evolution (Walsman et al., 2022).

Parasite propagule production

Finally, trait changes in hosts in response to predation risk may also affect the production of infective stages, for example, in cases where the stress responses of hosts lower their general condition and the availability of resources necessary for the production of infective stages, thereby lowering parasite propagule production. However, it is also possible that host trait changes in response to predation risk lead to elevated parasite propagule production. For example, water fleas show an increase in parasite spore production when exposed to predation risk cues as they increase their body size in response to predation, which leads to the availability of more resources to parasites to produce propagules (Duffy et al., 2011). Parasite propagule production can also be affected by behavioral trait changes of hosts in response to predation risk as exemplified by a higher release of trematode infective stages from infected gastropods when predation risk cues are present. This probably results from a higher activity of snails as an antipredator response, which in turn leads to increased parasite propagule release due to increased metabolic activity (Cornelius et al., 2023).

POPULATION-LEVEL CONSEQUENCES

As noted earlier, predation-risk effects on parasite–host interactions initially result from trait changes in individual prey that also serve as hosts. However, the resulting indirect effects on parasite–host interactions may also have population-level consequences for hosts and parasites. Although such consequences are theoretically likely, the question is whether these effects really matter, that is, whether trait-mediated predation-risk effects translate into significant numerical responses of host or parasite populations or in substantial changes to their regulation. For predation-risk effects on predator–prey and herbivore–plant interactions, evidence for such nonconsumptive effects of predators on their prey in the strict sense (sensu Peacor et al., 2020) is extremely limited (see review by Sheriff et al., 2020). Given the paucity of studies on predation-risk effects on parasite–host interactions, evidence is even poorer for nonconsumptive effects of predators on hosts via altered parasite–host interactions.

Whether this lack of information reflects a lack of important effects or just a lack of investigations is unclear. However, it is possible to speculate about situations where predation-risk effects should matter for host population dynamics in the context of parasitism. In general, for predation-risk effects to have population-level consequences for hosts, trait changes in individual prey need to translate into effects on population dynamics. In cases where trait changes are directly linked with the processes of individual growth, survival, or fecundity (Sheriff et al., 2020), an upstream impact on population dynamics is more likely than in cases where such links are indirect. Hence, trait changes in prey that reflect energetically costly defenses against predators (Figure 3b) are likely to affect the fitness of individuals (Figure 3d), and this in turn could lead to population-level consequences for the prey. The same holds for trait changes connected to resource use since the altered feedback from prey on their food may have population-level effects. Likewise, trait changes in prey that lead to alterations of parasite–host interactions (Figure 3c) may in turn have fitness consequences for hosts (Figure 3d). As parasite infections by definition affect their host individual's energy budget and, therefore, growth, survival, and/or fecundity (Combes, 2001), infection is likely to have fitness consequences for individual hosts and could ultimately affect host population dynamics.

However, effects at higher levels of organization will likely depend on the pathogenicity of the respective parasite and the strength of intensity-dependent pathology. In the case of virulent parasites with strong intensity-dependent pathology, predation risk-induced changes in host encounter, parasite establishment, or infection persistence (Figure 4) are likely to have strong effects on host populations. In contrast, in the case of relatively benign parasite infections with lesser pathology, predation risk-induced changes in parasite–host interactions may be of little relevance for host population dynamics. However, if long-lived parasites that are more benign keep impacting their hosts' energy budgets throughout their life, infections could still have strong impacts on host populations by affecting growth, survival, and/or fecundity.

Predation-risk effects may have relevance not only for host populations but also for the population dynamics of their parasites (Figure 3e). Any strong effects on host populations caused by predation risk-induced changes of parasite–host interactions will also have repercussions for parasite populations due to the strong dependence of parasites on their hosts. However, in cases where host populations are not affected by predation risk-induced changes to parasite–host interactions, predation-risk effects may still matter for parasite populations because changes in host encounter, parasite establishment, infection maintenance, or propagule production are all likely

to affect parasite population dynamics. For example, increased transmission stemming from predation risk-induced trait changes in hosts may significantly boost parasite population growth without significant effects on hosts. One possible scenario for such effects could be cases where prey group together in response to predation risk, which then leads to population growth for directly transmitted pathogens if these now spread more readily among hosts. In contrast, for parasites with indirect transmission, such a scenario might lead to an encounter-dilution effect for the parasites.

EFFECTS ON OTHER SPECIES

Predation risk-induced changes in parasite–host interactions may also affect other species in the wider community (Figure 3e). This could happen if the respective parasites also infect other hosts beyond the species showing trait changes due to predation risk. For instance, greater infection in one host species due to predation-risk effects may result in higher infection risk for other competent hosts due to greater general parasite population size and infectious propagule production. Such changes in infection levels may in turn alter interspecific competition between hosts, which can often be modified by differential effects of parasite infections on different species (Hatcher et al., 2006). More alarmingly, such changes in infection level could have severe consequences for zoonotic spillover of parasites, including to humans. As many hosts harbor more than one parasite species, predation risk-induced changes in parasite–host interactions involving a specific parasite species may also affect other parasites. This could have significant consequences for competitive intrahost interactions between parasites. For instance, prior infection by one parasite can increase host susceptibility to other species or strains (e.g., Halliday et al., 2020) and may also affect the composition of parasite communities within hosts, as well as both host and parasite fitness (Carpenter et al., 2021; Johnson et al., 2015).

In addition to effects resulting from changes in parasite populations, knock-on effects on the wider community (Figure 3e) may also result from the fitness effects of predation risk on individual hosts and its population-level consequences (Figure 3d). For example, if the respective host is a species with important ecological functions such as an ecosystem engineer, changes in host abundance are likely to have far-reaching effects on the wider community (Poulin et al., 1999; Pascal et al., 2020; Figure 3e). In addition, changes in host traits in response to predation risk could also affect the ecological functioning of host species in communities, with repercussions for ecosystem properties (Mouritsen & Poulin, 2005; Preston et al., 2016). While it is likely that predation risk-induced changes in parasite–host interactions may also affect other species in the wider community via these different pathways, detailed studies on such knock-on effects are lacking to date.

OUTSTANDING QUESTIONS AND FUTURE RESEARCH

Our synthesis highlights that there are several mechanisms by which predation-risk effects of predators can have consequences for parasite–host interactions. Existing studies suggest that such nonconsumptive effects of predators may have important repercussions for a wide range of parasites and diseases. However, the number of studies to date remains very limited, and more research into predation-risk effects on parasite–host interactions is needed to evaluate how common and broadly relevant they are (see Outstanding Questions Box 1). We thus call for experimental studies that explore predation-risk effects along the crucial steps in the life cycle of parasites that we present in our framework (Figure 4). Hosts that show strong predation-risk responses and that are open to experimental manipulations will be promising starting points to investigate basic research questions such as whether the known trait changes of hosts in response to predation risk affect one or more of the crucial steps in parasite–host interactions.

Even bigger research gaps exist in regard to population-level consequences and the effects on other species in the wider community of respective predator–prey/host–parasite systems (see previous two sections) but are consistent with our knowledge deficit when it comes to general predation-risk effects on predator–prey and herbivore–plant interactions (see review by Sheriff et al., 2020). As parasite infections usually affect the energy budget of infected hosts and, therefore, their growth, survival, and/or fecundity, which ultimately determine host fitness, it is quite likely that predation risk-induced alterations of parasite–host interactions can influence host population dynamics. However, this is unexplored territory and future research that integrates predation-risk effects in population models will be necessary to identify whether such population-level consequences exist.

Beyond the considerable nonconsumptive indirect effects discussed here, predators can also have direct consumptive effects on parasites (Figure 1a; Lopez & Duffy, 2021); for example, when predators preferentially feed on infected hosts, they can affect parasite infection levels in host populations (healthy herd hypothesis; Packer et al., 2003). Predators can also consume

BOX 1 OUTSTANDING QUESTIONS

While our synthesis highlights mechanisms that explain how predation-risk effects can have consequences for parasite–host interactions and disease dynamics, it also identifies a range of outstanding research questions:

1. How common are predation-risk effects on parasite–host interactions?
2. Do predation-risk effects differ among host encounter, parasite establishment, infection persistence/pathogenicity, and parasite propagule production?
3. Is there evidence for population-level consequences of changes in parasite–host interactions in response to predation risk?
4. Can predation-risk effects on parasite–host interactions result in knock-on effects on the wider community?
5. What is the relative importance of nonconsumptive effects of predators via predation-risk effects compared to the consumptive effects of predators such as predation on infected hosts and consumption of infective parasite stages?
6. Can trait changes in response to perceived infection risk ("landscape of fear") interfere with trait changes in response to predation risk with respect to the consequences for parasite–host interactions?
7. Can parasites be receivers of predation risk effects, that is, can they perceive predation risks and react with specific trait changes, potentially triggering similar effects as when hosts respond to predation risk cues?

free-living infective parasitic stages, which can affect transmission from one host to another (Johnson et al., 2010; Johnson & Thieltges, 2010; Thieltges et al., 2008). Predators thus have complex direct and indirect effects on parasite–host interactions, and studying the relative importance of predation risk effects compared to other predator effects on parasitism could yield profound insights (Lopez & Duffy, 2021; Shaw & Civitello, 2021).

Interestingly, it is not only predators that exert indirect effects on hosts through fear; parasites can also induce fear responses in potential hosts (Figure 1d). As for predation risk, a perceived threat of infection can lead to trait changes in potential hosts that in turn cause a range of indirect effects (Buck, 2019; Buck et al., 2018; Daversa et al., 2021; Koprivnikar et al., 2021). In some cases, infection risk-induced trait changes may counteract trait changes induced by predation risk. For instance, in the tadpole example discussed earlier, individuals increase their swimming activity in the presence of trematode cercariae to avoid infections and reduce this activity in the presence of predators (Koprivnikar & Urichuk, 2017; Thiemann & Wassersug, 2000). Currently we lack a comprehensive understanding of the relative importance of trait changes in response to both predation and infection risk for parasite–host interactions.

Finally, it is currently not known whether parasites can also be receivers of predation-risk effects considering that many free-living infective stages of parasites can be consumed by predators (Johnson et al., 2010; Johnson & Thieltges, 2010; Thieltges et al., 2008). If they can perceive predation risk and react with specific trait changes, it could potentially trigger similar indirect effects as discussed earlier, especially if some parasites have evolved antipredator behaviors, and this interferes with their transmission success. Since many free-living infective stages of parasites can perceive chemical cues which they use for host finding and seeking (Chaisson & Hallem, 2012; Haas, 2003), it is possible that certain chemical clues released by predators can be perceived by parasites and trigger specific antipredator strategies. However, whether such behavioral antipredator responses in parasites exist remains to be studied.

CONCLUSIONS

Our synthesis indicates that predation risk perceived by organisms that serve as hosts to parasites could have far-reaching indirect effects on parasite–host interactions, with implications for infectious disease dynamics and natural communities as a whole. To date, studies on predation-risk effects have mainly focused on how the threat of predation can affect predator–prey and herbivore–plant interactions; here we extend this general framework by including parasite–host interactions to widen the scope of potential nonconsumptive and trait-mediated indirect effects exerted by predators on prey. We look forward to future studies that will help to integrate the full range of predation-risk effects and evaluate their relative relevance compared to direct (consumptive) predator effects.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

No data were used in this synthesis.

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