**Broadening the ecology of fear: non-lethal effects arise from diverse responses to predation and parasitism**

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**Abstract**

Research on the ‘ecology of fear’ posits that defensive prey responses to avoid predation can cause non-lethal effects across ecological scales. Parasites also elicit defensive responses in hosts with associated non-lethal effects, which raises the longstanding, yet unresolved question of how non-lethal effects of parasites compare with those of predators. We developed a framework for systematically answering this question for all types of predator and parasite systems~~.~~ Our framework predicts that trait responses should be strongest toward predators, parasitoids and parasitic castrators, but more diverse and perhaps more frequent for parasites than for predators. In a case study of larval amphibians, whose trait responses to both predators and parasites have been relatively well-studied, existing data indicate that individuals generally respond more strongly and proactively to short-term predation risks than to parasitism. Apart from studies using amphibians, there have been few direct comparisons of responses to predation and parasitism, and none have incorporated responses to micropredators, parasitoids, or parasitic castrators, or examined their long-term consequences. Addressing these and other data gaps highlighted by our framework can advance the field toward understanding how non-lethal effects impact prey/host population dynamics and shape food webs that contain multiple predator and parasite species.

**Introduction**

 *“I'm not afraid of death; I just don't want to be there when it happens.”*

*-Woody Allen*

 Whether we call it fear or good sense, efforts to avoid death lead animals to forgo foraging~~,~~ reduce activity levels, seek refuges, and exhibit other costly responses to predators [1,2]. Parasites can have similar influences. To reduce infection risk, hosts may avoid infected conspecifics [3–5], defend against infectious propagule attack [6], or avoid risky areas, such as faeces representing a hot spot of undetectable nematode eggs [7–9]. Basic emotions like ‘disgust’ [9–12] and the age-old cliché “avoid like the plague” suggest that parasite avoidance is interwoven in our own history as much as is our fear of predators.

Fear exemplifies trait responses - adaptive morphological, physiological, and behavioural changes - elicited by threats from predators and parasites. Trait responses to either predators or parasites can have trade-offs that trigger non-lethal effects, including reduced individual fitness and ‘trait-mediated indirect effects’ [13], like trophic cascades [14–16] that shape communities [17]. Wolves, for example, frighten elk away from open foraging grounds into sheltered habitats with less nutritious vegetation, which then reduces elk birth rates [18] and alters vegetation structure [19]. By eliciting trait responses, predators and parasites impact prey/hosts, and wider communities, even without consuming them.

Trait responses to parasites, unlike most of those to predators, are not confined to proactive measures to reduce contact prior to attack. Because parasitism is not immediately lethal, hosts can respond after a successful attack by a parasite [20–22]. Immune responses are one of myriad host responses made after infection that can have non-lethal effects [23]. Further, post-infection trait responses elicited by parasites can last a long time, leading some to hypothesize that parasites could cause stronger cumulative non-lethal effects than predators [16,24]. Nevertheless, how non-lethal effects accumulate across different predator-prey and host-parasite interactions remain uncertain, in no small part due to the lack of a formal framework for drawing such comparisons. Existing frameworks, which focus either on specific systems or specific types of trait responses, do not accommodate the diversity of predatory and parasitic consumers and trait responses that they elicit in prey and hosts. This not only stymies research. With the yet incomplete understanding of how predators and parasites jointly influence populations and communities, popular wildlife conservation interventions, such as re-introductions and translocations, may have unanticipated consequences, as may ongoing disease pandemics.

Here, we compare trait responses to predation and parasitism, considering how their frequency, strength, and diversity (i.e. number of distinct types)may drive differences in how non-lethal effects accrue in prey and hosts. Building on recent conceptual developments [22,25], our goal is to establish a quantitative foundation for estimating non-lethal effects in ecosystems containing multiple predator and parasite species. We draw from consumer-resource theory to construct a general framework for studying prey and host trait responses to all types of predatory and parasitic consumers, including predators, micro-predators, parasitoids, parasitic castrators, typical parasites, and pathogens [26]. Unlike conceptual frameworks for the ecology of ‘fear’ and ‘disgust’, we deconstruct interactions into sequential phases to consider trait responses before, during, and after an attack, allowing us to compare and contrast the complete diversity of trait responses to predators and parasites. We use this framework to form specific predictions regarding how trait responses and associated non-lethal effects should manifest from interactions with different types of predators and parasites. We then use the framework to guide a systematic review of the comparative literature on trait responses that assessed the state of available information on the topic. We also analysed comparative data on larval amphibians, the most common animal group used by the reviewed studies~~,~~ as a case study in quantitatively comparing trait responses to predators and parasites. We conclude by highlighting unresolved questions in the field and how to address them.

**A general trait-response framework for examining non-lethal effects**

*Definitions and terminology*

We define prey/host trait responses broadly as changes in any trait (e.g., morphological, physiological, immunological or behavioural) to defend against predation or parasitism. We focus on prey/host adaptive, inducible responses. However, our framework (described below) can also consider maladaptive trait responses, such as those occurring from parasite manipulation [27], and constitutive defence adaptations. We use ‘non-lethal effects’ as a general term to describe the direct and indirect consequences of from prey/host trait responses to predators and parasites [13,28]). As we demonstrate below, terms developed for predator-prey systems, including ‘risk effects’ and ‘non-consumptive effects’ [13], are insufficient for describing the diversity of trait responses and associated effects possible from a general consumer-resource perspective. For example, not all trait responses are based on risk; many are actually based on consumption [16,22].

*Existing trait response frameworks*

The ecology of fear in predator-prey systems provides a strong, yet incomplete foundation for examining trait responses and their impact on populations and communities. Fear denotes a particular trait response to the risk of predation, before a predator attacks [13]. Prey movement away from foraging habitats when predators are nearby is a well-studied fear response [1,14,29]. However, as Lima and Dill (1990) pointed out in their seminal framework for behavioural decision-making in predator-prey interactions [1], prey may also exhibit defensive trait responses during predator attack and even after being captured, phases not covered within the standard domain of the ‘ecology of fear’. Systematic examination of non-lethal effects must go beyond fear to consider multiple trait responses made throughout interactions. This becomes especially obvious when incorporating responses to parasites.

In stark contrast to most predation, host responses after parasite attack can continue as parasites feed on individuals (i.e. during infection). The same is true for prey of micropredators (e.g. mosquitoes, ticks, leeches), which by definition attack and feed on multiple prey without necessarily killing them [26]. Surviving while being fed on by parasites or micropredators opens up a broad range of responses that slow or stop feeding, or otherwise minimize its impact. Immune responses are a clear example of host responses made during and after parasite feeding that do not occur in predator-prey systems. Immune responses and other trait responses during parasite and micropredator feeding also cause non-lethal effects to individuals and broader ecosystems [16,22]. Recognizing that post-attack trait responses can generate substantial non-lethal effects permits a more comprehensive assessment of how non-lethal effects drive population, community, and ecosystem processes.

*A general trait response framework*

The breadth of trait responses exhibited across different phases of predator-prey and host-parasite interactions are captured in our proposed framework (Fig. 1). The framework is informed by a general consumer-resource model that breaks down interactions as a sequence through which individuals transition among discrete sequential states [30] (Fig. S1). Predators and parasites transition through three states – questing (pre-contact searching for prey/hosts), attacking (attempting to subdue prey/hosts), consuming (actively ingesting prey/hosts; Fig. S1). At the same time, prey/hosts transition through four states – susceptible (not contacting predator/parasite), exposed (being attacked), consumed (being eaten), resistant (i.e. invulnerable to attack; Fig. S1). Individuals transition between those states following sequential biological processes: contact, attack failure and success, and feeding. The Lafferty et al. [30] model effectively characterizes the dynamics of all types of host-parasite, predator-prey, and other consumer-resource systems [30]. Thus, although this review focuses on animals and their consumers, our framework applies to all resource taxa—e.g., plants, animals, fungi, and bacteria--and their consumers. It can also include what might be considered to be “non-consumptive” parasites, like brood parasites.

Trait responses in our framework take three forms based on their function in defence - **avoid contact, resist attack,** or **resist consumption** (Fig. 1). *Susceptible* prey/hosts may **avoid contact** with *questing* consumers before an attack. Effective avoidance reduces the rate that *questing* predators and parasites transition to *attacking*, and prey/hosts benefit from not transitioning from *susceptible* to *exposed* states (Table S3). Prey/hosts that become *exposed* may **resist attack** to increase the probability that attacks fail. Resisting-attack includes “fight or flight” responses, like hares sprinting to burrows when being chased by lynx, or tadpoles rapidly and erratically swimming when being attacked by trematode cercariae [6]. Finally, prey/hosts that are being *consumed* (i.e. being eaten or infected) and remain alive may **resist consumption** through resistance [20] and tolerance [21] mechanisms. Resistance mechanisms shorten or slow predator and parasite feeding rates, exemplified by behaviours like social grooming by primates [31] and adaptive immune responses to parasitism [23] . Tolerance mechanisms lessen the damage from feeding without affecting feeding rates. Increasing tissue repair and protecting high-risk areas of the body from parasite feeding, as tadpoles do in response to trematodes [6], exemplify tolerance mechanisms for resisting consumption.

*Factors shaping the strength, duration, and frequency of trait responses*

With the full range of trait responses classified and integrated into a consumer-resource dynamics model, we can now consider the conditions that determine which responses predators and parasites are likely to elicit, and how strong and frequent they are likely to be. The extent to which prey/hosts avoid and resist consumers depends first and foremost on their basic physical and sensory abilities [32]. For instance, tadpoles cannot physically leave ponds when predators are present. They may, however, reduce activity to avoid contact [33]. Individuals must also be able to detect consumer threats to respond to them. Prey/hosts use both visual and non-visual cues to detect predation and parasitism risk, making sensory limitations (e.g. sight, hearing, and smell) potential constraints on trait responses [34,35]. Impediments to either risk detection or the ability to act on detected risk should reduce the frequency of, or even preclude, induced trait responses, whether behavioural, morphological, or physiological.

Even when prey/hosts have the physical and sensory capacity to mount responses, trade-offs may mediate the frequency and strength of trait responses [32]. Whether via reductions in foraging, reproduction, or energy levels, fitness-related costs of exhibiting trait responses compete with the benefits of responses (i.e. avoiding the costs of not responding), making the frequency and strength that individuals exhibit trait responses a matter of economics [32]. Perhaps less recognized is the possibility that trade-offs may change over different phases of any given interaction. The frequency, duration, and strength of trait responses will, interestingly, depend their relative costs and benefits compared to available trait responses at other phases of the interaction. Exemplified by certain host-parasite interactions, avoiding contact may be more costly than becoming infected, potentially driving stronger and more frequent resistance responses after becoming infected. The relative costs and benefits of different trait responses are accounted for in our framework through functions that link responses to phase-specific mortality and fecundity rates, which expresses response costs. Those costs are balanced by benefits of responses, expressed through response impacts on state transition rates, as described in the above section.

*Predicting trait responses and non-lethal effects across different predators and parasites*

The frequency, and strength and diversity of trait responses in prey/hosts will also depend on traits of the predators and parasites. Predators and parasites have distinct ‘consumer strategies’ [26] that comprise traits for attacking, feeding on and impacting prey/hosts [26,30]. We distinguish the following strategies: Predator, micropredator, parasitoid, parasitic castrator, typical parasite, pathogen [26]. Incorporating differences in consumer strategies into our framework leads to hypotheses and predictions for how trait responses and their non-lethal effects vary among different types of predators and parasites:

Hypothesis 1: The magnitude and frequency of trait responses scale with the fitness consequences of predator/parasite feeding. One key distinction among consumer strategies involves the fitness consequences of predation and parasitism on individual prey/hosts. Predators and most parasitoids eliminate future reproductive success of their prey or hosts by killing them, and parasitic castrators do so by blocking host reproduction. In contrast, feeding by most other types of parasites and micropredators is not so deadly and does not completely eliminate future fitness gains.

Intuitively, prey/hosts should generally exhibit stronger trait responses against predators or parasites whose feeding imposes more severe damage to fitness. This leads to prediction that consumers that eliminate prey/host fitness after successful attack - predators, parasitoids, and parasitic castrators - should generally elicit the strongest trait responses of any consumers, resulting in strong non-lethal effects from a given response. However, strong trait responses and non-lethal effects may also emerge with micropredators and parasites that *do* have strong negative fitness impacts, such as highly virulent pathogens, or the micropredators that vector them. However, trait responses to non-vectoring micropredators and less damaging parasites should be relatively weaker, with each response having weaker associated non-lethal effects. For example, we would expect amphibians to avoid breeding ponds containing fish predators and infective stages of highly virulent viruses (e.g. *ranavirus*) more than ponds containing pathogen-free leaches and infective stages of less virulent fungal parasites (e.g *Batrachochytrium dendrobatidis*). These predictions highlight that differences in the fitness consequences of being consumed should drive variation in non-lethal effects not only between predators and parasites, but also between different types of parasites, with some parasites being more similar to predators than to other types of parasites.

Hypothesis 2: The frequency of trait responses scales with the frequency of interactions. Predators and parasites differ in how frequently they pursue and attack prey/hosts. While the magnitude of the trait response should scale with consequence of consumption, the frequency of trait responses should scale directly with interaction frequency. This hypotheses indicates that parasites and micropredators might provoke lower-magnitude responses, but these responses could occur at higher frequencies. For instance, many animals spend more time swatting at biting insects than hiding from predators [36]. Hence, it is entirely possible that high-frequency low-level responses could have cumulative non-lethal effects as high or higher than those arising from low-frequency, strong responses.

Hypothesis 3: The timing of death determines the diversity of trait responses available to prey and hosts. We can also consider differences in the timing of prey and host death, or ‘reproductive death’, during interactions. Predators usually kill prey before or shortly after commencing to feed. This is not the case for most parasites and their hosts. In fact, keeping hosts alive during consumption can be critical to the persistence of certain parasites [40,41]. Even parasitoids and castrators can have a substantial amount of feeding time before hosts are killed or castrated. Recognizing basic differences in the timing of prey and host mortality leads to two clear predictions for how non-lethal effects of predators and parasites differ. First, prey responses to predators will be constrained to avoiding contact and resisting attack (Fig. 1b). Second, parasites and micropredators will elicit defensive trait responses at all interaction phases (Fig. 1c-d), meaning that hosts generally have a broader toolkit for defending against parasites than prey have for defending against predators. This suggests non-lethal effects of parasites arise from a more diversity in response types than those arising from predators.

Integrating the above hypotheses leads to the predictions that (1) predators, parasitic castrators, and parasitoids should provoke the strongest individual responses that are confined to avoiding contact and resisting attack (because of their shared negation of prey/host fitness upon successful attack and consumption), and (2) all types of parasites should generally provoke more frequent and diverse responses than do predators (because of their prolonged feeding stage on the live host).Exceptions may exist, such as some parasitoids that paralyze hosts during the initial attack, which can make hosts incapable of resisting consumption [39].Also, as discussed above, constraints to detection may alter expected differences in how prey/hosts exhibit trait responses to different predators and parasites. For instance, to the extent that visual detection is important to elicit trait responses, animals should avoid contact with predators more than contact with micropredators and parasites, because the generally larger size of predators makes them easier to see [26]. On average, however, our framework indicates that non-lethal effects will be strongest not solely from consumers that impose the highest risk of death, but rather from those that impose such strong costs while keeping the consumed host or prey alive.

**Applying the trait response framework**

We used the above framework carry out (a) a systematic review of the literature to compare trait responses to predation and parasitism and (b) a case study of trait responses to predators and parasites in larval amphibians (tadpoles). The two exercises serve two purposes. First, they provide a demonstration of how our framework can be applied to gain more thorough understanding of trait responses and their non-lethal effects. Second, they provide an assessment of the breadth of comparative data on the topic and preliminary synthesis the available information to explore general trait response patterns.

*Studies to measure trait responses to predation and parasitism*

* We compiled studies that directly compared trait responses to both predation and parasitism risk in a single prey/host species (see supporting material for details of literature search). ~~The~~ Almost all such studies measured trait responses in larval frogs (i.e. ‘tadpoles’, N = 106 entries across 13 studies, Table S1), perhaps because they are very tractable experimentally. Behavioural traits were most common, with activity level being the most reported trait (Table S1, Table S2). We did not find studies that measured immunological trait responses, likely because this is distinct to host-parasite systems. The studies spanned the following consumer strategies: solitary predators, trophically transmitted parasites, typical parasites, and pathogens (Table S1); in no cases were responses to parasitoids, parasitic castrators, micropredators, or social predators considered. Predator-induced trait responses were only measured during *questing* predator states, representing avoidance of contact, whereas measurements of parasite-induced responses included all three states: *questing* (N=9), *attacking* (N=5), and *consuming* (N=24). The limited data we found constrained our ability to make statistical comparisons, although some general patterns did emerge.

*A case study in tadpoles*

Despite the limited breadth of the comparative studies on trait responses to predators and parasites, there was sufficient data on predator- and parasite-induced trait responses in tadpoles, the most well-studied animal group in the reviewed studies, to quantitatively compare trait responses to predators and parasites. Analysis of the data (see the detailed methods in the supplementary material) showed that the magnitude and direction of tadpole responses to predators (Fig. 2a), parasites (Fig. 2b), and their combination (Fig. 2c) varied from study to study. However, on average and across all consumer and resource states, predator-induced trait responses were stronger in magnitude than parasite-induced trait responses (Fig. 3a, Table S4). These patterns were evident after controlling for consumer *state* (comparing questing predators to questing parasites) (Fig. 3b), though they were weaker (Table S4), likely owing to lower power of the data subset. Distinguishing between trait response types (avoid contact, resist attack, resist consumption) revealed that tadpoles did respond to parasites, but only by resisting parasites after infection and at lower magnitudes than their responses to avoid predator contact (Fig. 3b, Table S4). Tadpoles also responded to the simultaneous presence of predators and parasites on average, and at similar magnitudes to their responses to predators alone (Fig. 3a). The strong tadpole responses to predators primarily represented reductions in activity levels (Fig. S7a, Table S4), and they were most evident when measuring group-level responses as opposed to individual-level responses (Fig. S7b, Table S4). Across the host-parasite interactions studied, responses did not differ between pathogens and trophically-transmitted parasites, the two parasite strategies investigated (Table S3). None of the studies measured response frequency in nature and few considered long-term effects, both of which we expect might be higher in response to parasites than to predators.

**Discussion**

Our framework proposes that non-lethal effects from predation and parasitism are a function of diverse and potentially interacting defensive responses by prey and hosts, exhibited at different phases of consumer-resource interactions. Yet, current comparative data comprise brief snapshots of interactions that do not fully capture the temporal dynamics of trait responses, which precludes reliable comparisons of non-lethal effects. Tracking individuals through all interaction phases could provide insight into how non-lethal effects accrue from multiple trait responses (Box 1). Such longitudinal data could also detect interactive effects between trait responses. For example, hosts that invest heavily in immune defences may exhibit weaker avoidance of contact with parasites, particularly if avoidance conflicts with feeding, reproducing, and other fitness-related activities. The growing literature on ‘non-consumptive effects’ of parasites [22,40] focuses on contact avoidance, yet this misses several other ways that hosts can respond to parasitism. Taking a broader view of how prey/hosts can defensively respond helps us compare and contrast the diverse ways that predation and parasitism shape behavior, ecology, and evolution.

Trait response data on tadpoles, although limited, underscore the importance of distinguishing the timing of trait responses. Pooling all trait response measurements suggested that parasites generally did not elicit changes in tadpole activity levels as did predators. However, accounting for response timing revealed substantial changes in activity levels that were confined to post-infection periods of interaction. Adult amphibians show similar tendencies to resist infections rather than avoid them [41]. Resisting consumption appears to be primary form of trait response to parasitism in this group of animals, whose role in non-lethal effects can most fully be estimated by treating the responses as separate from those made earlier in interactions.

The post-infection resistance responses apparent in tadpoles are particularly notable considering that immune responses were not factored in. Immune responses are a very common form of resisting parasitism that can be exhibited for prolonged periods. The combined non-lethal effects of tadpole parasites were therefore likely much stronger than the analysed data suggest. Additionally, non-lethal effects of parasitism could have occurred from host phenotypic changes caused by parasite manipulation [27,42], and even directly from parasite feeding independent of defensive responses. For instance, general energy drain or direct tissue damage caused by parasite infection can substantially impact host performance [43,44]. Together, the diverse (and potentially frequent) non-lethal effects of parasites might rival predator avoidance in tadpoles.

Accounting for distinct predator and parasite strategies led to defensive trait-response predictions that did not align with the predator versus parasite dichotomy. Pinpointing the sources of variation in non-lethal effects therefore demands a functional approach rather than a taxonomic approach to distinguishing predators and parasites [26]. Parasitoids, and parasitic castrators in particular, could offer rewarding insights because they share different functional characteristics with either predators or other parasites. Avoidance and resistance responses to parasitoids are well-documented [39], but how their frequency and strength compare with responses to predators and less debilitating parasites is unknown. The latter can also be said for parasitic castrators. Given the high fitness consequences of infection from parasitoids and castrators, together with infections that are not immediately lethal, their combined non-lethal effects may very well be the strongest of all predator and parasite functional groups.

Regardless of how individuals respond to predators and parasites alone, risks of predation and parasitism in real ecosystems rarely occur in isolation. Future research could apply our framework to investigate the additive and interactive non-lethal effects of simultaneous exposure to predators and parasites. Our analysis of the tadpole data suggests that responses to simultaneous exposure are non-additive, perhaps owing to the prioritization of responses to the more severe threat (Fig. S5). Although not a focus of this review, evidence for increasing predation of parasitized prey [45–47], and increasing parasitism in predator-rich environments [48], provide further indication that predators and parasites interact to impose non-lethal effects. Fewer studies have considered single responses that defend against both predators and parasites. Nevertheless, there were several cases where tadpole responses to predators and parasites responses were in the same direction (i.e. a reduction in the trait expression). Trait responses that effectively deter both predators and parasites may mitigate the non-lethal effects incurred from the essential task of defending oneself against being eaten.

**Conclusion**

Whether through fear or through infection, predatory and parasitic consumers elicit defensive trait responses in their resources that give rise to non-lethal effects on individuals, with potential consequences for communities and ecosystems. A general consumer-resource model helped us to develop a framework for systematically comparing trait responses to various types of predators and parasites. Different types of predators and parasites should elicit different trait responses, and therefore, have different non-lethal effects, given differences in consumer strategies that influence when and how strongly they impact prey and hosts. However, many predator and parasite strategies have not yet been tested comparatively. Expanding research of non-lethal effects to regularly consider different predator and parasite strategies sets the foundation for exploring how non-lethal effects manifest in the multi-dimensional food webs found in real ecosystems.

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**References**

1. Lima SL, Dill LM. 1990 Behavioral decisions made under the risk of predation: a review and prospectus. *Canadian Journal of Zoology* **68**, 619–640.

2. Dröge E, Creel S, Becker MS, M’soka J. 2017 Risky times and risky places interact to affect prey behaviour. *Nature Ecology & Evolution* **1**, 1123–1128. (doi:10.1038/s41559-017-0220-9)

3. Milinski M, Bakker TCM. 1990 Female sticklebacks use male coloration in mate choice and hence avoid parasitized males. *Nature* **344**, 330.

4. Kavaliers M, Colwell DD, Braun WJ, Choleris E. 2003 Brief exposure to the odour of a parasitized male alters the subsequent mate odour responses of female mice. *Animal Behaviour* **65**, 59–68. (doi:10.1006/anbe.2002.2043)

5. Behringer DC, Butler MJ, Shields JD. 2006 Avoidance of disease by social lobsters. *Nature* **441**, 421–421. (doi:10.1038/441421a)

6. Sears BF, Snyder PW, Rohr JR. 2013 Infection deflection: hosts control parasite location with behaviour to improve tolerance. *Proceedings of the Royal Society B: Biological Sciences* **280**, 20130759–20130759. (doi:10.1098/rspb.2013.0759)

7. Hart BL. 1994 Behavioural defense against parasites: interaction with parasite invasiveness. *Parasitology* **109**, S139–S151.

8. Curtis VA. 2014 Infection-avoidance behaviour in humans and other animals. *Trends in Immunology* **35**, 457–464. (doi:10.1016/j.it.2014.08.006)

9. Weinstein SB, Buck JC, Young HS. 2018 A landscape of disgust. *Science* **359**, 1213–1214. (doi:10.1126/science.aas8694)

10. Curtis V, de Barra M. 2018 The structure and function of pathogen disgust. *Philosophical Transactions of the Royal Society B: Biological Sciences* **373**, 20170208. (doi:10.1098/rstb.2017.0208)

11. Dougherty ER, Seidel DP, Carlson CJ, Spiegel O, Getz WM. 2018 Going through the motions: incorporating movement analyses into disease research. *Ecology Letters* **21**, 588–604. (doi:10.1111/ele.12917)

12. Tybur JM, Çınar Ç, Karinen AK, Perone P. 2018 Why do people vary in disgust? *Philosophical Transactions of the Royal Society B: Biological Sciences* **373**, 20170204. (doi:10.1098/rstb.2017.0204)

13. Peacor SD, Barton BT, Kimbro DL, Sih A, Sheriff MJ. 2020 A framework and standardized terminology to facilitate the study of predation-risk effects. *Ecology* **101**, e03152. (doi:https://doi.org/10.1002/ecy.3152)

14. Brown JS. 1998 The ecology of fear: optimal foraging, game theory, and trophic interactions. *IFAC Proceedings Volumes* **31**, 31. (doi:10.1016/S1474-6670(17)38332-5)

15. Ritchie EG, Johnson CN. 2009 Predator interactions, mesopredator release and biodiversity conservation. *Ecology Letters* **12**, 982–998. (doi:10.1111/j.1461-0248.2009.01347.x)

16. Buck JC, Ripple WJ. 2017 Infectious agents trigger trophic cascades. *Trends in Ecology & Evolution* **32**, 681–694. (doi:10.1016/j.tree.2017.06.009)

17. Pringle RM *et al.* 2019 Predator-induced collapse of niche structure and species coexistence. *Nature* **570**, 58–64. (doi:10.1038/s41586-019-1264-6)

18. Creel S, Christianson D, Liley S, Winnie JA. 2007 Predation risk affects reproductive physiology and demography of Elk. *Science* **315**, 960–960. (doi:10.1126/science.1135918)

19. Fortin D, Beyer HL, Boyce MS, Smith DW, Duchesne T, Mao JS. 2005 Wolves influence elk movements: behavior shapes a trophic cascade in Yellowstone National Park. *Ecology* **86**, 1320–1330.

20. Rigby MC, Hechinger RF, Stevens L. 2002 Why should parasite resistance be costly? *Trends in parasitology* **18**, 116–120.

21. Raberg L, Graham AL, Read AF. 2009 Decomposing health: tolerance and resistance to parasites in animals. *Philosophical Transactions of the Royal Society B: Biological Sciences* **364**, 37–49. (doi:10.1098/rstb.2008.0184)

22. Buck JC. 2019 Indirect effects explain the role of parasites in ecosystems. *Trends in Parasitology* (doi:10.1016/j.pt.2019.07.007)

23. Hawley DM, Altizer SM. 2011 Disease ecology meets ecological immunology: understanding the links between organismal immunity and infection dynamics in natural populations: Disease ecology meets ecological immunology. *Functional Ecology* **25**, 48–60. (doi:10.1111/j.1365-2435.2010.01753.x)

24. Rohr JR, Swan A, Raffel TR, Hudson PJ. 2009 Parasites, info-disruption, and the ecology of fear. *Oecologia* **159**, 447–454. (doi:10.1007/s00442-008-1208-6)

25. Doherty J-F, Ruehle B. 2020 An integrated landscape of fear and disgust: The evolution of avoidance behaviors amidst a myriad of natural enemies. *Front. Ecol. Evol.* **8**, 564343. (doi:10.3389/fevo.2020.564343)

26. Lafferty KD, Kuris AM. 2002 Trophic strategies, animal diversity and body size. *Trends in Ecology & Evolution* **17**, 507–513.

27. Lafferty KD, Shaw JC. 2013 Comparing mechanisms of host manipulation across host and parasite taxa. *Journal of Experimental Biology* **216**, 56–66. (doi:10.1242/jeb.073668)

28. Abrams P. 2007 Defining and measuring the impact of dynamic traits on interspecific interactions. *Ecology* **88**, 25555–2562.

29. Creel S, Winnie J, Maxwell B, Hamlin K, Creel M. 2005 Elk alter habitat selectioin as an antipredator response to wolves. *Ecology* **86**, 3387–3397. (doi:10.1890/05-0032)

30. Lafferty KD, DeLeo G, Briggs CJ, Dobson AP, Gross T, Kuris AM. 2015 A general consumer-resource population model. *Science* **349**, 854–857. (doi:10.1126/science.aaa6224)

31. Hart BL, Hart LA. 2018 How mammals stay healthy in nature: the evolution of behaviours to avoid parasites and pathogens. *Philosophical Transactions of the Royal Society B: Biological Sciences* **373**, 20170205. (doi:10.1098/rstb.2017.0205)

32. Ydenberg RC, Dill LM. 1986 The economics of fleeing from predators. *Advances in the Study of Behavior* **16**, 229–249.

33. Hossie T, Landolt K, Murray DL. 2017 Determinants and co-expression of anti-predator responses in amphibian tadpoles: a meta-analysis. *Oikos* **126**. (doi:10.1111/oik.03305)

34. Kats LB, Dill LM. 1998 The scent of death: Chemosensory assessment of predation risk by prey animals. *Écoscience* **5**, 361–394. (doi:10.1080/11956860.1998.11682468)

35. Behringer DC, Karvonen A, Bojko J. 2018 Parasite avoidance behaviours in aquatic environments. *Phil. Trans. R. Soc. B* **373**, 20170202. (doi:10.1098/rstb.2017.0202)

36. Hart BL. 1992 Behavioral Adaptations to Parasites: An Ethological Approach. *The Journal of Parasitology* **78**, 256. (doi:10.2307/3283472)

37. Anderson RM, May RM. 1981 The population dynamics of microparasites and their invertebrate hosts. *Proceedings of the Royal Society B-Biological Sciences* **291**, 452–491.

38. Jensen KH, Little T, Skorping A, Ebert D. 2006 Empirical Support for Optimal Virulence in a Castrating Parasite. *PLoS Biol* **4**, e197. (doi:10.1371/journal.pbio.0040197)

39. Abram PK, Brodeur J, Urbaneja A, Tena A. 2019 Nonreproductive effects of insect parasitoids on their hosts. *Annu. Rev. Entomol.* **64**, 259–276. (doi:10.1146/annurev-ento-011118-111753)

40. Buck JC, Weinstein SB. 2020 The ecological consequences of a pandemic. *Biol. Lett.* **16**, 20200641. (doi:10.1098/rsbl.2020.0641)

41. Daversa DR, Manica A, Bosch J, Jolles JW, Garner TWJ. 2018 Routine habitat switching alters the likelihood and persistence of infection with a pathogenic parasite. *Functional Ecology* **32**, 1262–1270. (doi:10.1111/1365-2435.13038)

42. Poulin R, Brodeur J, Moore J. 1994 Parasite manipulation of host behaviour: Should hosts always lose? *Oikos* , 479–484.

43. Munger JC, Karasov WH. 1989 Sublethal parasites and host energy budgets: tapeworm infection in white-footed mice. *Ecology* **70**, 904–921. (doi:10.2307/1941358)

44. Delahay RJ, Speakman JR, Moss R. 1995 The energetic consequences of parasitism: effects of a developing infection of *Trichostrongylus tenuis* (Nematoda) on red grouse (*Lagopus lagopus scoticus*) energy balance, body weight and condition. *Parasitology* **110**, 473. (doi:10.1017/S0031182000064817)

45. Lafferty KD, Morris AK. 1996 Altered behavior of parasitized killifish Increases susceptibility to predation by bird final hosts. *Ecology* **77**, 1390–1397. (doi:10.2307/2265536)

46. Johnson PTJ, Stanton DE, Preu ER, Forshay KJ, Carpenter SR. 2006 Dining on disease: how interactions between infection and environment affect predation risk. *Ecology* **87**, 1973–1980. (doi:10.1890/0012-9658(2006)87[1973:DODHIB]2.0.CO;2)

47. Rae J, Murray D. 2019 Pathogen vs. predator: ranavirus exposure dampens tadpole responses to perceived predation risk. *Oecologia* **191**, 325–334. (doi:10.1007/s00442-019-04501-1)

48. Stephenson JF, Van Oosterhout C, Mohammed RS, Cable J. 2015 Parasites of Trinidadian guppies: evidence for sex-and age-specific trait-mediated indirect effects of predators. *Ecology* **96**, 489–498.

**Figures**

**Fig. 1. A general trait response framework and predictions.** **(a)** Resources can mount three sequential responses to consumers, each with distinct effects on the interaction: avoid contact, resist attack, resist consumption. Examples of each type of response are listed in italicized text. Responses may be constrained by physical and sensory limitations, as well as trade-offs against other fitness-related activities (e.g. feeding and reproducing). The general framework can be tailored to specific types of consumer-resource interactions, such as interactions between field mice and **(b)** owl predators, **(c)** biting flies and infective nematodes (red worms in faeces), or **(d)** between caterpillars and parasitoid wasps. The lack of a consumption stage in **(b)** illustrates that prey rarely respond during consumption by predators.

**Fig. 2. Forest plots of effect sizes used in the meta-analysis.** The distribution of effect sizes for responses elicited by the presence of **(a)** predators, **(b)** parasites, and **(c)** their combined presence resulting from resource adjustments in activity (blue), space use (red), and morphological/physiological traits (grey). Error bars denote the 95% confidence intervals.

**Fig. 3. Relative magnitude of responses to predation vs. parasitism. (a)** The estimated mean magnitude of trait responses to predation cues (blue), parasitism cues (red), and both cues (grey). **(b)** Mean trait responses to predators (blue) and parasites (red) when distinguishing by the type of trait responses, as defined in our framework. Only avoidance responses to questing predators were found in our literature review, likely owing the low probability of surviving attack or consumption by predators. Responses to the combined presence of predators and parasites are not shown in **(b)** because only one study with this treatment had predators and parasites in the same state. Lines denote the mean response magnitudes, boxes denote the standard error of the mean, and error bars denote the 95% confidence intervals.

Fig. 1



Fig. 2



Fig. 3



**Box 1. Future directions for research of non-lethal effects**

Comparative data on trait responses to predators and parasites are limited. Multiple research avenues can address these limitations to produce more comprehensive estimates of non-lethal effects in multi-trophic ecosystems:

1. **Comparative experiments of trait responses that cover a broader range of consumer-resource systems** can determine how generalizable the patterns across systems, and opens avenues to consider factors like consumer and resource phylogenies on response magnitudes. Incorporating treatments of both consumer threats minimizes between-study biases that arise through inherent discrepancies in environmental conditions and protocol execution of independent studies. The studies used in our case study provide examples for comparative experimental designs that future research can apply.

**2. Comparative studies that include responses to parasitic castrators, parasitoids and micro-predators** are needed to test the mechanisms, such as detection and fitness consequences, driving trait responses to predator and parasite threats (see discussion). Comparisons of trait responses to different consumer types are currently limited to typical and trophically-transmitted parasites, pathogens, and solitary predators – only three of the 10 consumer strategies found in natural ecosystems [26,30].

**3.** **Immunological responses to parasites**, although one of the most common forms of anti-parasite defence, were not factored into the comparative literature and likely obscured the results of our tadpole case study. Future research may factor in immunological responses of hosts when assessing how the magnitude of TMEs arising parasitism reflect compares with those of predation.

**4. Longitudinal data on prey and host responses** are needed to determine both the frequency and range of trait responses that individuals may exhibit throughout the course of interactions with consumers. For example, since hosts can resist parasitism during the consumption phase, an open question is whether individual hosts exhibit both avoidance and resistance responses when encountering parasites or if they choose one or the other. The strength of parasite-based TMEs is a function of the cumulative strength of responses made to all consumer states, but the studies reviewed here provided only snapshots of responses to single consumer states. With the types of potential trait responses now identified in our framework, future research can determine the range and frequency of responses exhibited by resources during non-lethal interactions to develop more comprehensive estimates of TMEs.

5. Our focus on trait responses lays a foundation for **scaling non-lethal effects to individual fitness and community function**. Future work can extend the length of prey and host monitoring to link trait responses to individual fitness. Mesocosm and field experiments mirroring the experimental designs of the studies used here can introduce primary producers and other species in food webs into the picture, allowing associations between response magnitudes to predators and parasites and trophic flows and cascades to be quantified.

1. Lima SL, Dill LM. 1990 Behavioral decisions made under the risk of predation: a review and prospectus. *Canadian Journal of Zoology* **68**, 619–640.

2. Dröge E, Creel S, Becker MS, M’soka J. 2017 Risky times and risky places interact to affect prey behaviour. *Nature Ecology & Evolution* **1**, 1123–1128. (doi:10.1038/s41559-017-0220-9)

3. Milinski M, Bakker TCM. 1990 Female sticklebacks use male coloration in mate choice and hence avoid parasitized males. *Nature* **344**, 330.

4. Kavaliers M, Colwell DD, Braun WJ, Choleris E. 2003 Brief exposure to the odour of a parasitized male alters the subsequent mate odour responses of female mice. *Animal Behaviour* **65**, 59–68. (doi:10.1006/anbe.2002.2043)

5. Behringer DC, Butler MJ, Shields JD. 2006 Avoidance of disease by social lobsters. *Nature* **441**, 421–421. (doi:10.1038/441421a)

6. Sears BF, Snyder PW, Rohr JR. 2013 Infection deflection: hosts control parasite location with behaviour to improve tolerance. *Proceedings of the Royal Society B: Biological Sciences* **280**, 20130759–20130759. (doi:10.1098/rspb.2013.0759)

7. Hart BL. 1994 Behavioural defense against parasites: interaction with parasite invasiveness. *Parasitology* **109**, S139–S151.

8. Curtis VA. 2014 Infection-avoidance behaviour in humans and other animals. *Trends in Immunology* **35**, 457–464. (doi:10.1016/j.it.2014.08.006)

9. Weinstein SB, Buck JC, Young HS. 2018 A landscape of disgust. *Science* **359**, 1213–1214. (doi:10.1126/science.aas8694)

10. Curtis V, de Barra M. 2018 The structure and function of pathogen disgust. *Philosophical Transactions of the Royal Society B: Biological Sciences* **373**, 20170208. (doi:10.1098/rstb.2017.0208)

11. Dougherty ER, Seidel DP, Carlson CJ, Spiegel O, Getz WM. 2018 Going through the motions: incorporating movement analyses into disease research. *Ecology Letters* **21**, 588–604. (doi:10.1111/ele.12917)

12. Tybur JM, Çınar Ç, Karinen AK, Perone P. 2018 Why do people vary in disgust? *Philosophical Transactions of the Royal Society B: Biological Sciences* **373**, 20170204. (doi:10.1098/rstb.2017.0204)

13. Peacor SD, Barton BT, Kimbro DL, Sih A, Sheriff MJ. 2020 A framework and standardized terminology to facilitate the study of predation-risk effects. *Ecology* **101**, e03152. (doi:https://doi.org/10.1002/ecy.3152)

14. Brown JS. 1998 The ecology of fear: optimal foraging, game theory, and trophic interactions. *IFAC Proceedings Volumes* **31**, 31. (doi:10.1016/S1474-6670(17)38332-5)

15. Ritchie EG, Johnson CN. 2009 Predator interactions, mesopredator release and biodiversity conservation. *Ecology Letters* **12**, 982–998. (doi:10.1111/j.1461-0248.2009.01347.x)

16. Buck JC, Ripple WJ. 2017 Infectious agents trigger trophic cascades. *Trends in Ecology & Evolution* **32**, 681–694. (doi:10.1016/j.tree.2017.06.009)

17. Pringle RM *et al.* 2019 Predator-induced collapse of niche structure and species coexistence. *Nature* **570**, 58–64. (doi:10.1038/s41586-019-1264-6)

18. Creel S, Christianson D, Liley S, Winnie JA. 2007 Predation risk affects reproductive physiology and demography of Elk. *Science* **315**, 960–960. (doi:10.1126/science.1135918)

19. Fortin D, Beyer HL, Boyce MS, Smith DW, Duchesne T, Mao JS. 2005 Wolves influence elk movements: behavior shapes a trophic cascade in Yellowstone National Park. *Ecology* **86**, 1320–1330.

20. Rigby MC, Hechinger RF, Stevens L. 2002 Why should parasite resistance be costly? *Trends in parasitology* **18**, 116–120.

21. Raberg L, Graham AL, Read AF. 2009 Decomposing health: tolerance and resistance to parasites in animals. *Philosophical Transactions of the Royal Society B: Biological Sciences* **364**, 37–49. (doi:10.1098/rstb.2008.0184)

22. Buck JC. 2019 Indirect effects explain the role of parasites in ecosystems. *Trends in Parasitology* (doi:10.1016/j.pt.2019.07.007)

23. Hawley DM, Altizer SM. 2011 Disease ecology meets ecological immunology: understanding the links between organismal immunity and infection dynamics in natural populations: Disease ecology meets ecological immunology. *Functional Ecology* **25**, 48–60. (doi:10.1111/j.1365-2435.2010.01753.x)

24. Rohr JR, Swan A, Raffel TR, Hudson PJ. 2009 Parasites, info-disruption, and the ecology of fear. *Oecologia* **159**, 447–454. (doi:10.1007/s00442-008-1208-6)

25. Doherty J-F, Ruehle B. 2020 An integrated landscape of fear and disgust: The evolution of avoidance behaviors amidst a myriad of natural enemies. *Front. Ecol. Evol.* **8**, 564343. (doi:10.3389/fevo.2020.564343)

26. Lafferty KD, Kuris AM. 2002 Trophic strategies, animal diversity and body size. *Trends in Ecology & Evolution* **17**, 507–513.

27. Lafferty KD, Shaw JC. 2013 Comparing mechanisms of host manipulation across host and parasite taxa. *Journal of Experimental Biology* **216**, 56–66. (doi:10.1242/jeb.073668)

28. Abrams P. 2007 Defining and measuring the impact of dynamic traits on interspecific interactions. *Ecology* **88**, 25555–2562.

29. Creel S, Winnie J, Maxwell B, Hamlin K, Creel M. 2005 Elk alter habitat selectioin as an antipredator response to wolves. *Ecology* **86**, 3387–3397. (doi:10.1890/05-0032)

30. Lafferty KD, DeLeo G, Briggs CJ, Dobson AP, Gross T, Kuris AM. 2015 A general consumer-resource population model. *Science* **349**, 854–857. (doi:10.1126/science.aaa6224)

31. Hart BL, Hart LA. 2018 How mammals stay healthy in nature: the evolution of behaviours to avoid parasites and pathogens. *Philosophical Transactions of the Royal Society B: Biological Sciences* **373**, 20170205. (doi:10.1098/rstb.2017.0205)

32. Ydenberg RC, Dill LM. 1986 The economics of fleeing from predators. *Advances in the Study of Behavior* **16**, 229–249.

33. Hossie T, Landolt K, Murray DL. 2017 Determinants and co-expression of anti-predator responses in amphibian tadpoles: a meta-analysis. *Oikos* **126**. (doi:10.1111/oik.03305)

34. Kats LB, Dill LM. 1998 The scent of death: Chemosensory assessment of predation risk by prey animals. *Écoscience* **5**, 361–394. (doi:10.1080/11956860.1998.11682468)

35. Behringer DC, Karvonen A, Bojko J. 2018 Parasite avoidance behaviours in aquatic environments. *Phil. Trans. R. Soc. B* **373**, 20170202. (doi:10.1098/rstb.2017.0202)

36. Hart BL. 1992 Behavioral Adaptations to Parasites: An Ethological Approach. *The Journal of Parasitology* **78**, 256. (doi:10.2307/3283472)

37. Anderson RM, May RM. 1981 The population dynamics of microparasites and their invertebrate hosts. *Proceedings of the Royal Society B-Biological Sciences* **291**, 452–491.

38. Jensen KH, Little T, Skorping A, Ebert D. 2006 Empirical Support for Optimal Virulence in a Castrating Parasite. *PLoS Biol* **4**, e197. (doi:10.1371/journal.pbio.0040197)

39. Abram PK, Brodeur J, Urbaneja A, Tena A. 2019 Nonreproductive effects of insect parasitoids on their hosts. *Annu. Rev. Entomol.* **64**, 259–276. (doi:10.1146/annurev-ento-011118-111753)

40. Buck JC, Weinstein SB. 2020 The ecological consequences of a pandemic. *Biol. Lett.* **16**, 20200641. (doi:10.1098/rsbl.2020.0641)

41. Daversa DR, Manica A, Bosch J, Jolles JW, Garner TWJ. 2018 Routine habitat switching alters the likelihood and persistence of infection with a pathogenic parasite. *Functional Ecology* **32**, 1262–1270. (doi:10.1111/1365-2435.13038)

42. Poulin R, Brodeur J, Moore J. 1994 Parasite manipulation of host behaviour: Should hosts always lose? *Oikos* , 479–484.

43. Munger JC, Karasov WH. 1989 Sublethal parasites and host energy budgets: tapeworm infection in white-footed mice. *Ecology* **70**, 904–921. (doi:10.2307/1941358)

44. Delahay RJ, Speakman JR, Moss R. 1995 The energetic consequences of parasitism: effects of a developing infection of *Trichostrongylus tenuis* (Nematoda) on red grouse (*Lagopus lagopus scoticus*) energy balance, body weight and condition. *Parasitology* **110**, 473. (doi:10.1017/S0031182000064817)

45. Lafferty KD, Morris AK. 1996 Altered behavior of parasitized killifish Increases susceptibility to predation by bird final hosts. *Ecology* **77**, 1390–1397. (doi:10.2307/2265536)

46. Johnson PTJ, Stanton DE, Preu ER, Forshay KJ, Carpenter SR. 2006 Dining on disease: how interactions between infection and environment affect predation risk. *Ecology* **87**, 1973–1980. (doi:10.1890/0012-9658(2006)87[1973:DODHIB]2.0.CO;2)

47. Rae J, Murray D. 2019 Pathogen vs. predator: ranavirus exposure dampens tadpole responses to perceived predation risk. *Oecologia* **191**, 325–334. (doi:10.1007/s00442-019-04501-1)

48. Stephenson JF, Van Oosterhout C, Mohammed RS, Cable J. 2015 Parasites of Trinidadian guppies: evidence for sex-and age-specific trait-mediated indirect effects of predators. *Ecology* **96**, 489–498.