Review

# Parasites as predators: unifying natural enemy ecology

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**Parasitism and predation have long been considered analogous interactions. Yet by and large, ecologists continue to study parasite–host and predator–prey ecology separately. Here we discuss strengths and shortcomings of the parasite-as-predator analogy and its potential to provide new insights into both fields. Developments in predator–prey ecology, such as temporal risk allocation and associational resistance, can drive new hypotheses for parasite–host systems. Concepts developed in parasite–host ecology, such as threshold host densities and phylodynamics, might provide new ideas for predator–prey ecology. Topics such as trait-mediated indirect effects and enemy-mediated facilitation provide opportunities for the two fields to work together. We suggest that greater unification of predator–prey and parasite–host ecology would foster advances in both fields.**

## Introduction

Since Anderson and May's seminal models of parasite–host dynamics [1], the analogy of parasites as predators has provided fundamental insights for the emerging field of parasite–host ecology [2]. Anderson and May drew upon the rich predator–prey literature to derive basic models of parasite–host dynamics [1]. These models have been useful for understanding many parasite–host systems [3–5], attesting to the value of predator–prey theory as a source of insights into parasite–host ecology. Before then, parasitology and ecology were treated largely as separate areas of inquiry, with parasitologists emphasizing description of species and life cycles and ecologists emphasizing organismal interactions at macroscopic scales. In the last three decades, however, the field of parasite–host ecology has grown in prominence, recently approaching the number of predator–prey publications in 13 top ecological journals (Figure 1). In spite of increased usage of the parasite-as-predator analogy (Box 1) and of the more general term 'natural enemy' [6–10], the two fields remain largely separate. In cases where the parasite-as-predator analogy is valid, predator–prey ecology should have much to offer the nascent field of parasite–host ecology. Likewise, parasite–host ecology should also have concepts to offer predator–prey ecology.

In this paper, we evaluate the parasite-as-predator analogy by discussing the characteristics of parasites that make them similar to and different from predators. We also evaluate the implications of these differences, and we discuss whether and how key concepts developed for predation have been used in the development of hypotheses for parasitism. We then explore new ways that the analogy could be used to derive hypotheses for both fields. We conclude by summarizing the implications and limitations of the parasite-as-predator analogy and calling for greater integration of predator–prey and parasite–host ecology into the more general framework of natural enemy ecology.

## Are parasites predators?

Parasitism is sometimes conceptualized as a special case of predation [1,2], but not all parasites fit the classic definition of a predator. Classically, predation is defined as an

### Glossary

**Acquired immune system**: the portion of the vertebrate immune system which responds to novel parasites via directed cell mutation followed by selection for responsiveness to parasite molecules.
**Amensalism**: interaction in which one organism causes a negative effect on another without being positively or negatively affected in return.
**Aposematism**: evolution of distinctive warning signals by resistant (often toxic) prey species to discourage predation.
**Apparent competition**: indirect negative effect of one prey or host species on another, mediated by a shared natural enemy.
**Associational resistance**: interaction in which one prey or host species benefits from associating with another prey or host species that has superior defenses against natural enemies.
**Batesian mimicry**: evolution of a susceptible prey species to resemble a resistant prey species to discourage predation.
**Competition**: negative interaction between two species, usually mediated by a limited shared resource.
**Constitutive**: formed or expressed without regard to environmental conditions.
**Density-mediated indirect effects**: effects of one species (or abiotic factor) on another mediated by effects on the population density of a third species.
**Dilution effect**: lower parasitism due to higher host diversity, usually referring to reduced parasite transmission to a susceptible host caused by wasted infection attempts on a resistant host.
**Immune system/immunity**: nonbehavioral defensive mechanisms used by hosts against parasites.
**Inducible**: capable of being formed, activated or expressed in response to a stimulus.
**Interaction web**: description of interactions between species in an ecosystem, including both trophic and nontrophic relationships.
**Keystone species**: a predator which promotes diversity by targeting competitively superior prey.
**Natural enemy**: organism that obtains resources from and exerts a direct negative effect on another organism.
**Parasite**: a symbiont that causes harm to another organism, the host, which the parasite utilizes as habitat.
**Phylodynamics**: the study of interacting epidemiological and evolutionary processes that drive spatiotemporal and phylogenetic patterns of disease dynamics.
**Predator**: an organism that consumes another organism, the prey.
**Symbiosis**: interaction in which one organism lives with, in or on the body of another.
**Threshold host density**: minimum population density of hosts necessary to maintain the cycle of parasite transmission.
**Trait-mediated indirect effects**: effects of one species (or abiotic factor) on another species mediated by effects on traits of a third species.
**Trophic level**: position of an organism in a food chain.

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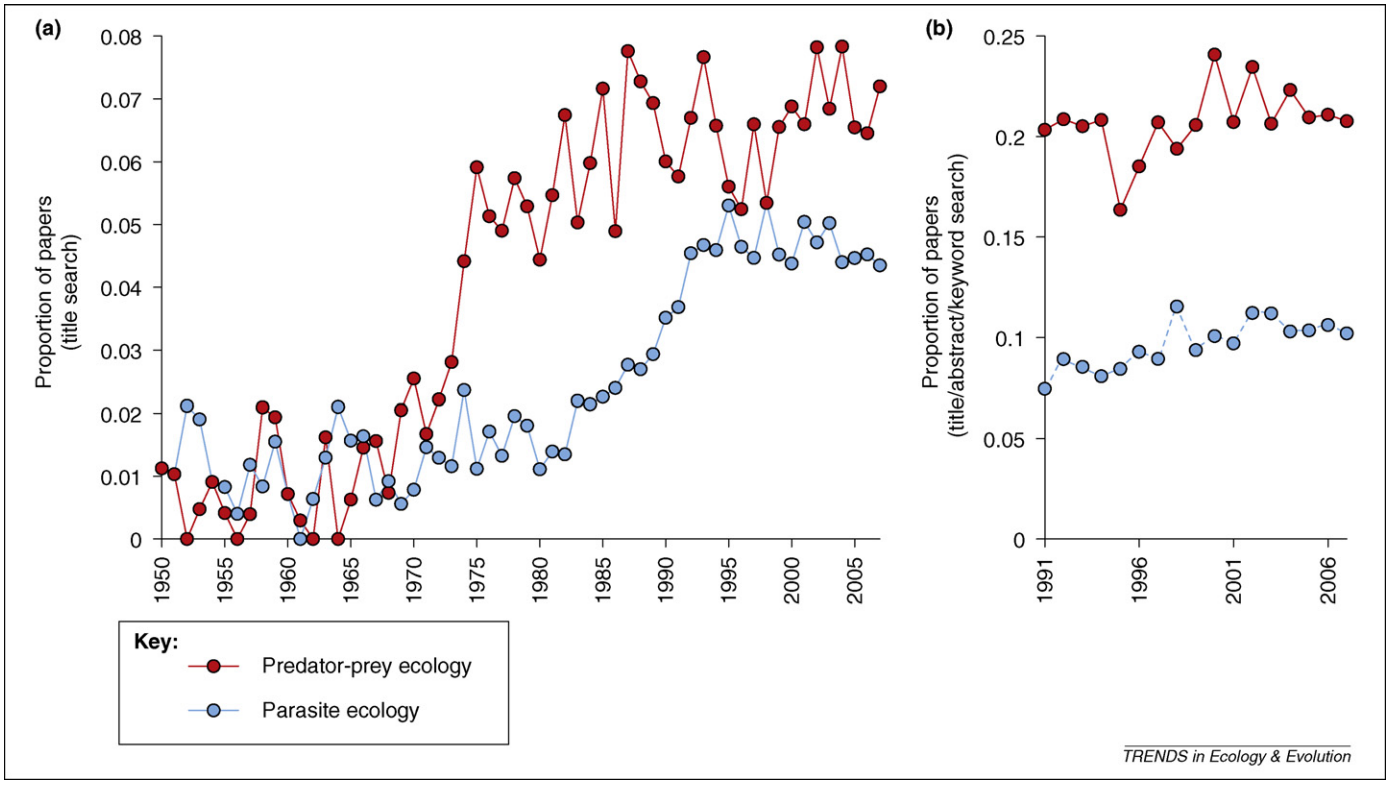


**Figure 1**. Historical trends in predator–prey and parasite–host ecology. **(a)** Disease ecology emerged as an important subdiscipline of ecology in the 1980 s and came to prominence in the 1990 s, nearly 20 years after predator–prey ecology. Data are proportions of papers published in selected ecological journals since 1950 whose titles included search terms related to disease ecology (disease* or parasit* or pathogen*) or predator–prey ecology (predat* or prey), based on ISI Web of Science searches (journals: *Trends in Ecology and Evolution*, *Ecology*, *Ecological Applications*, *Ecological Monographs*, *Journal of Ecology*, *Journal of Animal Ecology*, *Journal of Applied Ecology*, *Functional Ecology*, *Ecology Letters*, *Oecologia*, *Oikos*, *Annual Review of Ecology and Systematics* and *American Naturalist*). **(b)** A significant increase in parasite-related publications occurred from 1991 to 2007 ($P < 0.001$) with no change in predator–prey publications ($P = 0.141$; proportions arcsine transformed). Indeed, the rate of increase in parasite-related publications was more than twice that of predator–prey publications (standardized slope coefficient: 0.373 versus 0.774). These findings were based on a search of titles, abstracts and keywords which provided more precise data (abstracts and keywords before 1991 could not be searched reliably owing to changes in the search engine).

interaction in which one organism consumes either all or part of another living organism (the prey), causing a direct negative effect on the prey [11]. Parasitism is a symbiosis in which one organism, the parasite, causes harm to another, the host, which the parasite utilizes as habitat and depends on for resource acquisition [12]. Various characteristics have been proposed to separate the two interaction types, including differences in body size (predators > prey; parasites < hosts [2]), the durability of the interaction (longer interactions between parasites and hosts [13]) and the number of victims attacked throughout a life-history stage (predators attack multiple prey; parasites utilize a single host [14]). Although useful for choosing models to describe population dynamics [14], these distinc-

## Box 1. Historical use of the parasite-as-predator analogy

The parasite-as-predator analogy has provided useful insights into the emerging field of disease ecology. In addition to motivating the models of Anderson and May [1], the analogy has been used to develop theory for how parasites interact with each other within hosts [72] and for how parasites influence species invasions into new habitats [73]. Similar to predators, parasites can mediate apparent competition between alternative hosts, sometimes leading to exclusion of one host by the other [8]. Additionally, due to tradeoffs between competitive ability and antiparasite defenses in hosts, parasites can act as keystone species, promoting host species diversity by selectively targeting competitively superior host species [39].

Many concepts developed in the study of antipredator behaviors, such as optimal levels of defense under different levels of predation and resource abundance, tradeoffs between antipredator defense and reproduction, and gregarious social behavior under the risk of predation [17], have also proven relevant in parasite–host systems [74]. Antiparasite defenses are costly to hosts and trade off with reproductive effort, and host gregariousness can reduce the per-capita attack rate of searching parasites [74].

Another predator–prey concept which has been incorporated into the parasite ecology literature is intraguild predation, in which a top predator ('intraguild predator') consumes both a prey species ('intraguild prey') and the intraguild prey's resource [12]. Similar models have been used to describe parasite–host systems in which one parasite (analogous to the intraguild predator) can infect hosts already infected with another parasite (analogous to the intraguild prey), thereby killing both the host and the initial parasite [12].

Some aspects of predator–prey ecology have had more delayed integration into parasite–host ecology than others, such as the assimilation of parasites into food webs [15]. Food webs are composed of consumer–resource interactions and until recently were almost entirely restricted to predators and prey [15]. Parasite ecologists have started incorporating parasites into food webs by placing parasites on the trophic level above their hosts, treating host–parasite relationships as trophic interactions regardless of whether a given parasite truly consumes host tissue [15]. This approach has been successful for predicting food web properties, such as the efficiency of energy transfer between trophic levels [15], although use of more general interaction web terminology might be more appropriate for parasites that do not actually feed upon their hosts [75].

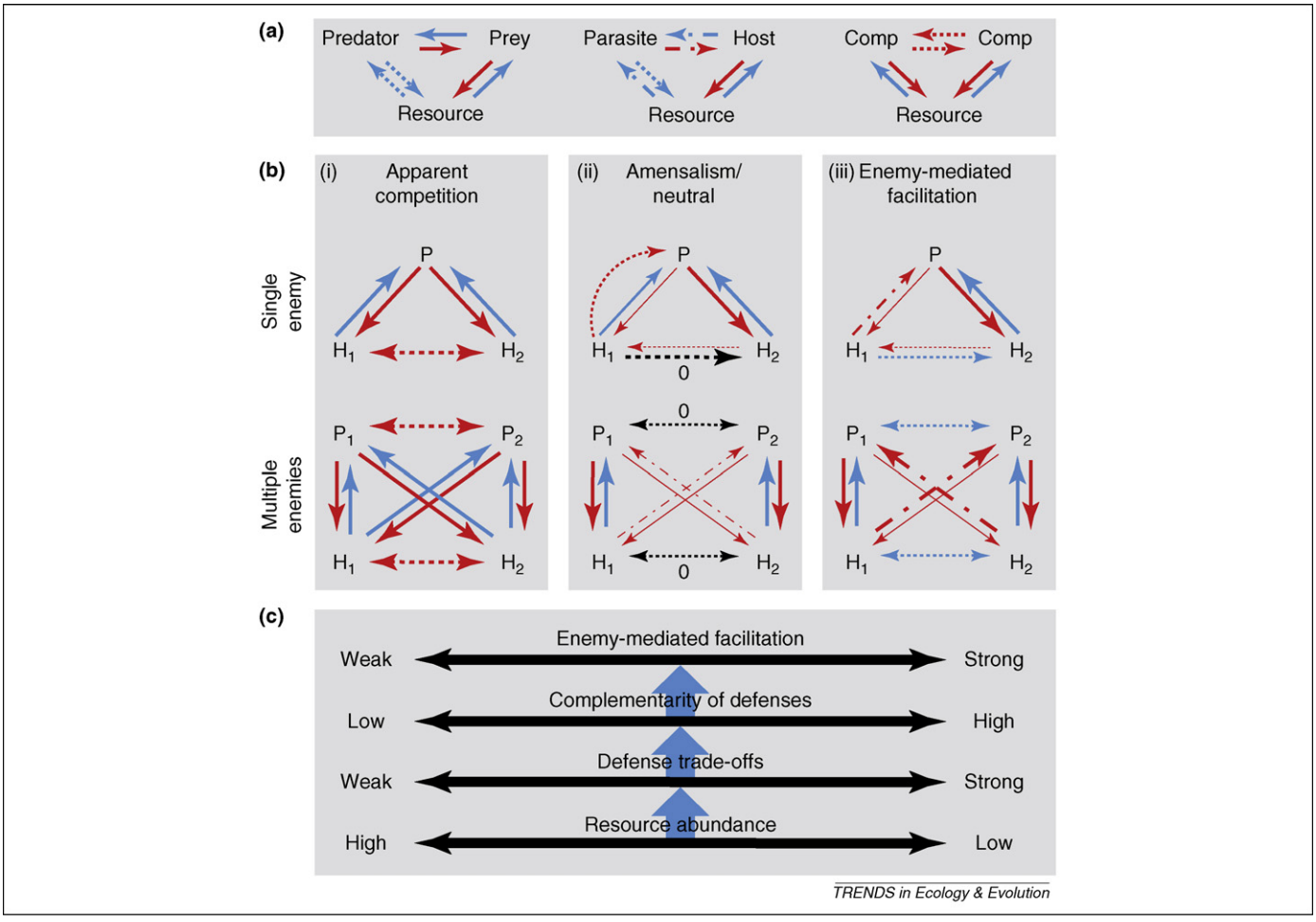


**Figure 2**. Flow diagrams for different types of community-level interactions. **(a)** Comparison of resource use in predator–prey, parasite–host and competitive interactions. Arrows indicate positive (blue), negative (red) or neutral (0) effects. As with a predator, a parasite benefits from its host and is expected to influence resources positively via negative effects on the host. Solid arrows indicate trophic interactions (direct consumption) and dashed arrows indicate nontrophic interactions (indirect effects). Dotted and dashed arrows indicate interactions which might or might not be direct, depending on whether a parasite consumes host tissue. **(b)** Interaction diagrams of apparent competition and enemy-mediated facilitation between prey or host species (H) mediated by natural enemies (P). The first row indicates the gradient from apparent competition to indirect amensalism to unidirectional facilitation via a single natural enemy. The curved dashed arrow indicates the indirect costs to the enemy of attacking the resistant prey or host, which in this case balances the benefit of attacking this prey or host for a neutral net effect. The net effect of this interaction is indicated in subsequent diagrams using a dotted and dashed arrow. The second row indicates the gradient from bidirectional apparent competition to enemy-mediated facilitation via two shared natural enemies. **(c)** Proposed effect of resource abundance on the strength and frequency of enemy-mediated facilitation in communities. Limited resource abundance might increase tradeoffs in the abilities of prey or hosts to defend against different natural enemies, leading to greater specialization of prey or hosts to defend against only one or a few natural enemies. If different prey or hosts specialize to defend against different natural enemies, prey or hosts might rely more heavily on facilitative interactions with complementary prey or host species to defend against natural enemies. Upward pointing arrows indicate proposed causal relationships between gradients.

tions can be problematic; for example, some organisms, such as many caterpillars, consume plant tissue but feed on a single individual. Generally classified as predators, these insect herbivores would instead be considered parasites by the latter definition. We therefore prefer to recognize predators and parasites as overlapping classes of natural enemies, distinguished by consumption of prey tissue or symbiosis with a host organism, respectively.

By these criteria, parasites that consume host tissue (e.g. mosquitoes) should also be considered predators, and predators that live in or on the bodies of their prey (e.g. leaf-boring insects) should also be considered parasites. However, many parasites do not consume host tissue at all, such as tapeworms which absorb nutrients from the host's last meal [12]. Such parasites are in a sense more like competitors for host resources than predators (Figure 2a). Indeed, they might be considered on the same trophic level as their hosts (Box 2), despite the frequent characterization of parasitism as a type of trophic interaction [14,15]. Unlike classic competitors, however, parasites rely entirely on hosts for access to resources. This characteristic is similar to predators, which rely on prey for access to energy and nutrients. Because of this reliance, the directions of positive and negative interactions among a parasite, its host and their shared resources are more similar to predation than to competition (Figure 2a), even for parasites that do not consume host tissue. These similar interactions are the foundation of the parasite-as-predator analogy, but care should be taken when extending the analogy to other aspects of parasite–host or predator–prey biology (Box 2).

## Using predator–prey ecology to generate new hypotheses for parasite–host ecology

Despite the successful application of the parasite-as-predator analogy to many questions in parasite–host systems

**Box 2. How do parasites differ from predators?**

Although predation and parasitism share much in common, the definitions of parasites as symbionts with their hosts and predators as consumers of prey tissue lead to some fundamentally distinct differences between the two interaction types. Ignoring these differences could lead to inappropriate usage of the parasite-as-predator analogy.

**Parasites as symbionts**

**Relationship intimacy**
Because parasites utilize their hosts as habitat, parasite–host interactions are more physically intimate than interactions of free-living predators with prey. Such physical intimacy promotes evolution of specific defensive mechanisms (e.g. cellular- and molecular-level defenses) that are unavailable to predators. Likewise, parasites can exploit hosts by hijacking host genetic replication mechanisms (e.g. viruses), opportunities unlikely to be exploited by predators.

**Relationship duration**
Predation events are often immediately fatal for prey, whereas host–parasite relationships are relatively long in duration [13]. Furthermore, relationship duration is integral to parasite fitness. Long-duration infections provide more opportunities to transmit infectious stages to new hosts but trade off with the rate of transmission, because both are thought to depend upon the level of parasite virulence [76]. This tradeoff leads to the evolution of intermediate virulence [76], a concept irrelevant to predators which should always maximize lethality. Because parasites sometimes evolve reduced virulence, parasites are more likely to evolve into mutualists with their hosts than predators are with their prey.

**Generation time**
Parasites are typically smaller and have shorter generation times than hosts, allowing parasites to use hosts as habitat and giving parasites an advantage in an evolutionary arms race. This advantage might explain the need for acquired immune responses in vertebrates, which provides a way for cells replicating within the host body to 'evolve' new immune defenses at a rate similar to or exceeding rates of parasite evolution [21]. The situation is reversed for predators, which generally have longer generation times than their prey. Predators should therefore develop advanced learning mechanisms analogous to acquired immunity, allowing them to respond to rapid changes in prey defenses.

**Predators consume prey tissue**

**Trophic level**
Predators and prey occupy different trophic levels, whereas parasites and hosts sometimes exist on the same resource plane if they compete for host resources rather than consume host tissue. The more the parasite's nutrients have been processed by the host, the higher the parasite's trophic level relative to the host. Nonpredatory parasites should thus exhibit less accumulation of toxins and stable isotopes than predators [15].

(Box 1), important concepts remain that can provide new and potentially useful hypotheses.

### *Temporal variation in risk: inducible defenses, risk anticipation and risk allocation*

Prey often utilize inducible rather than constitutive defenses, giving them flexibility to counter predators solely when threats occur [16]. These inducible defenses have important effects on predator–prey dynamics, tending to stabilize cyclic fluctuations in predator and prey populations [16]. Inducible defenses can also have substantial effects on cyclic parasite–host dynamics. For example, acquired immunity (an inducible immune defense) following mass vaccination can lengthen epidemic cycles [3].

Prey can sometimes anticipate and prepare for predictable periods of elevated predation risk, responding to diurnal, seasonal and monthly patterns of predator activity with preemptive adjustments of antipredator defenses [17]. Parasite infection risk can also follow predictable seasonal or diurnal cycles [18,19], as can changes in host immune responses [20,21], but most studies have assumed that such seasonal changes in immunity are driven by seasonal breeding or environmental variables (e.g. temperature) [20,21]. Researchers should consider that these changes might sometimes reflect preemptive responses to predictable changing infection risk.

When prey are unable to anticipate changes in predation risk, induction or reduction of antipredator defenses sometimes lags behind these changes, reducing the efficacy of inducible defenses [17]. Inducible host responses probably also lag behind unpredictable changes in infection risk, based on observed lags in immune changes following fluctuations in temperature or other environmental factors [20]. This might lead to periods of suboptimal antiparasite defenses [20]. The fitness consequences of these lags in response to changes in infection risk remain relatively unexplored.

The magnitude of inducible defenses against predators can also be influenced by temporal variation in the risk of predation, as postulated by the risk allocation hypothesis [22]. This hypothesis proposes that prey should respond more strongly to predation risk if this risk is brief and infrequent, because in this case prey can make up for large opportunity costs (e.g. reduced foraging activity) of strong antipredator responses during the long low-risk periods. If the risk of predation is more frequent or prolonged, however, prey should be forced to forage even during high-risk periods, so their antipredator responses should be weaker, as observed for several predator–prey systems (e.g. cichlid fish exposed to predation cues at different frequencies [23]).

Hosts might also invest in antiparasite responses according to the risk allocation hypothesis if (i) there is a tradeoff between antiparasite responses and other important activities, such as reproduction or foraging (documented for various hosts [21]), and (ii) hosts reliably detect and respond to temporal variation in infection risk (e.g. desert locusts [24]). If hosts adhere to the risk allocation hypothesis, it would have important implications for the interpretation of experimental infection studies. Hosts used in infection studies are frequently maintained in parasite-free environments followed by a pulse of very high exposure. According to the risk allocation hypothesis, this practice could lead to elevated antiparasite responses relative to those expected under more natural conditions [22].

### *Trait-mediated indirect effects*

Predators have long been known to induce indirect effects on non-prey species via effects on their prey, both by reducing prey density (density-mediated indirect effects; DMIE) and by modifying the traits of their prey in ways

that alter interactions with non-prey species (trait-mediated indirect effects; TMIE) [25,26]. For example, predators can induce changes in prey behavior or space use that affect competitive interactions [25]. Recent evidence from predator–prey ecology indicates that TMIEs can have equal or greater impacts on communities than DMIEs [25,27]. Although parasites can also induce indirect effects by influencing host traits [9,28], such interactions have received less attention than predator-induced TMIEs.

Predators and parasites can also be the recipients of TMIEs when biotic or abiotic factors influence traits of species with which they interact [25,29]. For example, a superior competitor can disrupt refuge use by prey, making them easier for predators to locate and attack [25], and recent evidence suggests that TMIEs of pesticides (increased host susceptibility) on trematode infection rates in tadpoles are stronger than DMIEs of the pesticides (reduced parasite survival) [30,31]. Decreased resources can also lead to suppression of host immune responses with subsequent effects on parasite infection rates [32], but such effects are seldom referred to as TMIEs and are essentially absent from the TMIE literature. Both parasites and predators appear more often to be considered as drivers than recipients of TMIEs. Whether this apparent trend is caused by a research bias toward studying top-down TMIEs and natural enemies tending to occur toward the top of interaction webs [33] or whether there is a true predominance of top-down TMIEs in natural communities is an open question.

Predators can also induce TMIEs on parasites and vice versa, and these effects could have important implications for disease emergence and parasite regulation. Species coexistence patterns, parasite populations and disease emergence can be profoundly affected by parasite-driven changes in hosts that elevate predation on infected prey [7,34]. Here, the parasite is the driver and the predator is the recipient of the TMIE. However, the reverse scenario has been largely ignored [9], despite evidence that predators cause prey stress responses, which can induce immune suppression [32]. Such TMIEs could counteract or reinforce DMIEs of selective predation on infected hosts, altering probabilities of coexistence and disease emergence [7].

In addition to individual-level effects, there are numerous examples of TMIEs being transmitted to population and community levels in predator–prey ecology [17,25], but examples in the parasite–host literature remain rare. This rarity could be due to a bias arising from the assumption that antiparasite responses are weak relative to antipredator responses, based on parasitic infections usually being less immediately fatal than predation [35]. However, infections can be detrimental to hosts and more frequent than predation events, so that investment in defenses against parasites should not necessarily be lower than against predators. Hence, parasites might commonly both drive and be the recipients of TMIEs, with profound effects on biodiversity and ecosystem function [36].

### *Enemy-mediated facilitation: associational resistance and the dilution effect*

Indirect positive effects between organisms sometimes outweigh direct negative effects in communities, leading to net facilitation between community members that would otherwise be antagonists [37]. Predators can induce net increases in prey populations indirectly, by selectively attacking other predators (intraguild predation [38]) or superior competitors of prey (keystone predation [39]). Similar positive indirect effects on hosts have also been recognized for parasites, such as the whirling disease agent in fish which facilitates invasion of its host brown trout *Salmo trutta* by infecting and killing off trout competitors with minimal pathogenicity to the trout [40].

Prey can also facilitate one another. For instance, a resistant prey species can benefit another prey species via negative effects on a shared predator, if the costs of attacking the resistant prey outweigh the benefits of consuming it (horizontal facilitation; Figure 2biii). If costs and benefits are balanced, the indirect effect on the susceptible prey or host is neutral, creating an indirect amensalism (Figure 2bii). Hence, there should be a gradient from apparent competition to indirect amensalism to enemy-mediated facilitation, a continuum which depends upon the efficacy of the antienemy defenses of the resistant host species. Such unidirectional facilitation has been recognized for decades in the predation literature in studies of associational resistance [41], apparent facilitation [42] and of Batesian mimicry [43], which can be thought of as a type of associational resistance in which the 'association' is the close resemblance of a susceptible prey species to a resistant (often toxic) species.

A recent parasite ecology concept analogous to associational resistance is the dilution effect [44]. As with associational resistance, the dilution effect occurs when one host species facilitates another by exerting a net negative effect on a shared parasite. Dilution effects are potentially common [45] and might occur most often in parasite–host systems with vectors or frequency-dependent transmission [46,47]. Examples are the negative effect of alternative mammalian hosts on Lyme disease transmission in mice [44] and reduced trematode infection in toad tadpoles due to parasite-resistant tree frog tadpoles [48].

Parallels between the dilution effect and associational resistance can guide future hypothesis development and tests in parasite–host ecology. For example, aposematism and Batesian mimicry are common in predator–prey ecology [43] but have not been considered in parasite–host systems. Assuming that parasites can directly or indirectly (e.g. by manipulating vector behavior) choose which hosts they infect, a resistant (diluting) host species should distinguish itself from susceptible hosts (aposematism). Conversely, susceptible hosts should mimic resistant hosts so as to avoid infection. The implicit assumption of parasite choice is most plausible for parasites that actively search for hosts (e.g. trematodes and bacteriophages [49]) and for parasites that exert considerable control over the transmission process by manipulating host behavior [28]. Hence, the prevalence of aposematism and mimicry might increase with the degree of active parasite transmission or host manipulation. Moreover, parasites typically have poorly developed vision and thus are unlikely to locate hosts using visual signals as in many predator–prey systems (Figure 3). Hence, Batesian mimicry in response to parasites might be common but unobserved due to para-

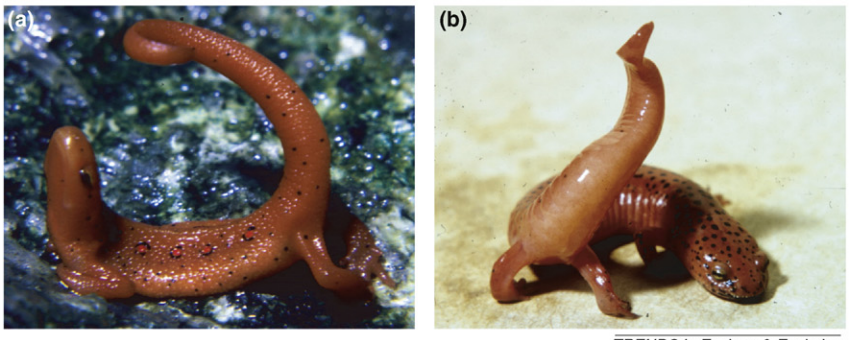


**Figure 3**. Batesian mimicry of **(a)** the toxic red-spotted newt (*Notophthalmus viridescens*) by **(b)** the nontoxic red salamander (*Pseudotriton ruber*). The similar red coloration and postural displays of *P. ruber* fool predators into mistaking them for the toxic *N. viridescens*, making predators avoid both species [71]. Susceptible hosts might also evolve mechanisms to mimic parasite-resistant alternate hosts if parasites actively avoid infecting resistant hosts, causing parasites to avoid the mimic as well. Such mimicry is likely to involve chemical rather than visual signals, however, because parasites typically have poorly developed vision. Photos were provided by Edmund D. Brodie, Jr. of Utah State University.

sites using subtle sensory mechanisms, such as olfaction, to choose hosts.

Natural enemies might also mediate bidirectional positive effects between two or more prey or host species. Bidirectional enemy-mediated facilitation, known as 'apparent mutualism,' can be mediated by a single natural enemy species if natural enemy density is completely or partially (i.e. through temporal delay) independent of prey or host density, if the natural enemy is cannibalistic, or if natural enemies switch between prey or host types depending upon which is more abundant and profitable [50–52]. Multiple resistant prey or hosts could also facilitate each other via negative effects on a shared natural enemy, as in cases of Müllerian mimicry (multiple toxic prey items mimicking each other [53]).

Finally, apparent mutualism might arise when the prey or host species share multiple enemies but are resistant to different enemies (Figure 2biii). This can be envisioned as a type of niche partitioning driven by variation in antienemy defenses, with different species complementing each other by specializing on resistance to different natural enemies. This outcome is more likely if different defense mechanisms are needed to defend against different natural enemies, and if these defenses trade off with each other, as is likely to be true if one natural enemy is a predator and the other a parasite [54,55].

Most species have multiple natural enemies and share enemies with several other species [56], making apparent mutualism a potentially important mechanism of coexistence in natural communities. As with unidirectional facilitation, there should be a continuum from apparent competition to enemy-mediated facilitation for bidirectional interactions (Figure 2b). Despite the potential importance of enemy-mediated apparent mutualism (e.g. birds that form mixed-species flocks for mutual protection against predators [57]), we know of no empirical studies seeking such an interaction in parasite–host systems. Indeed, the frequency of apparent mutualisms among prey or hosts in natural communities is essentially unknown but might be quite high, especially as many of these interactions might be partially or fully masked by simultaneous counteracting interactions among prey or hosts (e.g. resource competition).

Neutral and asymmetric positive interactions are common in interaction webs and can influence community stability [58,59]. Enemy-mediated facilitation is likely to be asymmetric owing to differential resistance of prey or hosts, so that the special cases of bidirectional neutral interactions or balanced apparent mutualism (Figure 2bii,iii) are probably rare. Hence, enemy-mediated facilitation might also be an important factor in the maintenance of community stability and thus biodiversity. Asymmetric facilitation might also influence the order in which species invade communities (community assembly) because susceptible species might be unable to establish without the presence of enemy-resistant species. Conversely, invasive hosts might sometimes benefit native hosts when parasites attempt to infect often-resistant invasive hosts [60].

The frequency and strength of enemy-mediated facilitation in communities might be influenced by resource availability (Figure 2c). Resource limitation could limit the strength and types of antienemy defenses available to prey. Assuming that such defenses are costly and that different defense mechanisms are used to combat different natural enemies, more resources should be required to defend against multiple enemies than against one. Thus, when resources are limited, species might be forced to specialize on defending against particular natural enemies (e.g. investing in antipredator defenses to the detriment of immune defenses). This type of specialization could be facilitated by associating with species that have complementary defenses (e.g. another species invested in immune defenses instead of antipredator defenses), leading to stronger reliance on apparent mutualisms. Consequently, we postulate that resource limitation could promote greater reliance on enemy-mediated facilitation with species that have complementary defenses (Figure 2c). These responses could occur rapidly in response to changing resource abundance if antienemy responses are plastic, allowing individual animals to shift from general to specialized defense on short timescales. This hypothesis predicts that complementarity of prey or host defenses should influence species distributions and coexistence. The proposal that resource limitation (a biotic stressor) increases reliance on facilitative interactions

complements theoretical and empirical studies showing that abiotic stress can sometimes intensify facilitative interactions in communities [61].

### Turning the analogy around: parasites inform predator–prey ecology

Despite being a younger field, parasite–host ecology can offer insights into predator–prey ecology. First, parasite ecologists often focus on controlling disease outbreaks by reducing susceptible host population density to a level below which parasites cannot persist [62]. Resource managers can use this threshold host density as a guide to cull or vaccinate hosts. There have been many successful applications of this theory to controlling and preventing disease outbreaks [63,64], despite evidence that thresholds are often difficult to identify in natural systems [62].

Although the concept of threshold prey density is nothing new to the predator–prey literature [51], we are unaware of this concept being used for managing predatory pests and invasive species. Managers protecting prey populations usually focus on killing predators by applying pesticides or introducing 'biocontrol' organisms [65] rather than by managing prey. However, managing or manipulating susceptible prey might be more feasible in some situations and possibly with fewer negative ramifications, especially when all predators cannot be eliminated directly. By identifying threshold densities of susceptible prey, managers can determine how many resistant individuals are required in a population to bring unwanted predation under control. Such a strategy could allow use of resistant crop varieties without necessitating complete replacement of susceptible crops. Such intercropping could alleviate problems with monocultures of genetically modified organisms, such as reduced genetic diversity, tradeoffs between predator resistance and productivity, and consumer wariness [66].

Parasite ecologists have also proposed taking advantage of host heterogeneities in parasite transmission rates to improve parasite control [67]. By treating or removing only individuals or functional groups with especially high rates of infection, thereby contributing disproportionately to disease transmission ('superspreaders'), one can control disease outbreaks without causing unnecessary damage to the host population [67]. This concept could also inform predator control if sufficient within-population variation in prey antipredator defenses can be identified. For example, especially bold or aggressive prey individuals can be more susceptible to predation [68] and could be targeted for treatment or removal. Caution must be used in applying this strategy, however, to avoid diminution of genetic diversity and removal of individuals that contribute disproportionately to prey population persistence.

Parasite ecologists have also developed and advanced the new field of phylodynamics, which unifies the study of ecological and evolutionary parasite dynamics [69]. This field was spurred by the fact that parasite evolution is frequently rapid enough to matter at the ecological timescale of the host. Although predators typically have long generation times relative to their prey (Box 2), evolutionary changes in predator and prey populations can sometimes operate on ecological timescales [70]. Phylogenetic methods developed to elucidate the evolutionary history and past population dynamics of infectious diseases [69] might prove useful for identifying and studying the dynamics of such predator–prey systems.

### Conclusions and synthesis

Parasitism is not a specialized form of predation. Rather, parasites and predators are overlapping categories of natural enemies, with fundamental differences arising from the close physical relationship of parasites with their hosts and the consumption of host tissue by predators (Box 2). Thus far, the parasite-as-predator analogy has served both fields well (Box 1). Many concepts from predator–prey ecology, such as hypotheses describing prey responses to

**Box 3. Outstanding questions**

**Using predator–prey ecology to guide hypotheses in parasite ecology**

(i) Do hosts respond to predictable cycles of infection risk, or to temporal variation in this risk, in ways consistent with predator–prey theories, such as the risk allocation hypothesis?
(ii) Do inducible defenses stabilize parasite–host dynamics, as antipredator responses stabilize predator–prey interactions?
(iii) Is the dilution effect for parasites functionally similar to associational resistance for predators? How common is dilution in parasite–host systems, and what other mechanisms drive facilitative interactions between hosts?
(iv) Do parasites induce mimicry in their hosts, and how does mimicry affect parasite–host dynamics?
(v) How important are anthropogenic changes in the traits of parasites and hosts for disease emergence? How significant are parasite-induced trait-mediated effects to communities?

**Using parasite–host ecology to inform predator–prey ecology**

(i) Are concepts from parasite ecology, such as threshold host density, useful for controlling predatory pests?
(ii) Can prey phenotypes that disproportionately contribute to the growth of predatory pest populations be targeted for removal to improve pest control?
(iii) How important are evolutionary dynamics to predator–prey interactions on ecological timescales? Can concepts and techniques of phylodynamics developed by parasite ecologists be applied to predator–prey systems, despite prey having the evolutionary advantage rather than vice versa?

**Ways the two fields can work together to advance natural enemy ecology**

(i) Are host responses to parasites as strong or costly as prey responses are to predators? What is the relative importance of parasite-mediated interactions versus predator-mediated interactions for community structure, function and stability?
(ii) How common is enemy-mediated facilitation in natural communities? Is apparent mutualism important to species coexistence and community stability and, if so, under what conditions? What are the primary mechanisms that generate facilitative interactions? Is complementarity of prey and host defenses against diverse natural enemies a major driver of facilitation? How does facilitation influence the progression of community assembly?
(iii) How important are trait-mediated indirect effects of natural enemies relative to direct effects and density-mediated indirect effects? How do they influence community structure and stability? What traits are influenced by predators and parasites? Are trait-mediated effects predominantly top-down effects, and how do trait-mediated effects on predators and parasites influence their dynamics?

temporal variation in predation risk, how predators mediate indirect effects on other community members, and enemy-mediated facilitation, seem naturally extendable to parasite–host systems (Box 3). Well-developed concepts in the parasite ecology literature, such as threshold host density, host heterogeneities and phylodynamics, can be applied to predator–prey systems, with potential applications for managing pests and invasive species (Box 3). Most importantly, predator–prey and parasite–host investigators working hand in hand would aid further development of these concepts, setting the stage for long-standing collaborations between these fields.

Despite fundamental differences between parasitism and predation, these two interaction types appear functionally equivalent in most cases. Theoretical and empirical developments for predator–prey ecology are likely to mirror results in the parasite–host literature, and vice versa. Because parasitism and predation are different interaction types, however, the analogy should not be used for all aspects of natural enemy ecology (Box 2). Ecologists must continue to refine and assess the analogy by comparing theoretical and empirical developments in the two fields. It would benefit both fields to improve communication by developing a more unified field of natural enemy ecology, combining insights from both predator–prey and parasite–host ecology.

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