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REVIEWS

Review

Indirect Effects Explain the Role of Parasites in Ecosystems

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Parasites are increasingly recognized as integral members of ecological communities, but their ecological effects remain less clear. Here, I propose that, to uncover the unique role of parasites, we must understand their indirect effects, which differ in important ways from those caused by predators. Similar to predators, parasites can cause density-mediated indirect effects (DMIEs) through their consumptive effects, and trait-mediated indirect effects (TMIEs) through their nonconsumptive effects; however, because they can consume a host without killing it, parasites can also trigger TMIEs through their consumptive effects. I consider the relative importance of each parasite-induced indirect interaction type and demonstrate their population-, community-, and ecosystem-level consequences. This paper contributes to recent efforts to unite predator–prey and parasite–host theory under a general consumer–resource framework.

The Framework

Bighorn sheep populations in British Columbia are dwindling, and a unique type of species interaction could be to blame. The parasitic mite, *Psoroptes ovis*, causes lesions that block the ear canal of sheep and impair their hearing [1]. In turn, hearing loss could make infected sheep more vulnerable to cougar predation [2,3]. Thus, an indirect interaction between the mite and the cougar is mediated by a trait change (in this case, physiology) of their shared victim, the sheep (A.T. Ford, personal communication, 2017). However, this interaction is fundamentally different from trait-mediated indirect interactions initiated by predators. Predator-induced **TMIEs** (see Glossary) arise from **nonconsumptive effects**; that is, the trait change is a defensive strategy meant to reduce the likelihood of consumption. By contrast, the above example represents a **consumptive TMIE**: the mite consumes (i.e., infects) the sheep and simultaneously changes the traits of the latter, thereby altering its interaction with the cougar. Such interactions arise because parasites are not immediately lethal to their hosts, and they can propagate downward to resources, upward to other consumers, or sideways through nontrophic interactions.

Despite recognition that parasites often alter host traits and cause TMIEs [4–6], the distinction between consumptive and nonconsumptive TMIEs has been overlooked (but see [7,8] for a discussion of consumptive TMIEs induced by herbivores). Here, I consider indirect effects of parasites within the framework established for predator–prey interactions (Figure 1). I discuss the relative frequency and strength of each indirect interaction type and highlight their potential effects on populations, communities, and ecosystems. Finally, I examine the potential for parasites to affect biodiversity via their indirect effects.

Parasites as Consumers

As defined by Lafferty and Kuris [9], predators (except **micropredators**; see later) kill and consume multiple victims during a life-history stage. By contrast, infectious agents attack and consume a single victim during a life-history stage and do so without immediately killing it. For example, **parasitoids** kill their host as a required part of their development, but they consume it gradually. **Parasitic castrators** also imperil host fitness, but, unlike parasitoids, they do so without killing their host. **Microparasites** and **macroparasites** can be, but are not necessarily, lethal to their hosts. Microparasites multiply in or on their host, but macroparasites do not. **Trophically transmitted parasites** do not kill their host outright but require the host to be eaten by a predator for successful transmission. In addition to these five types of infectious agent, micropredators (e.g., mosquitoes, koalas, or leafhoppers) can also consume a victim without killing it. Similar to predators, micropredators attack more than one victim during a life-history stage and are not infectious, but, unlike predators, they only take a small

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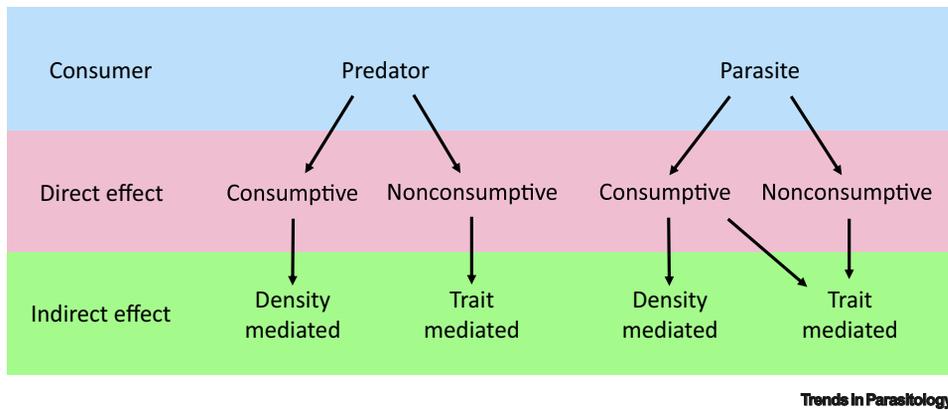
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Figure 1. Direct and Indirect Effects of Predators and Parasites.
Predators can trigger density-mediated indirect effects (DMIEs) through their consumptive effects and trait-mediated indirect effects (TMIEs) through their nonconsumptive effects. However, because parasites are not immediately lethal to their victims, in addition to these two types of indirect effect, they can also cause TMIEs through their consumptive effects.

meal and, hence, do not kill their victim. Following Lafferty and Kuris [9], I consider distinctions between these consumer types to be functional rather than taxonomic, and a consumer species may fall into more than one category, depending on how it interacts with its resources. Hence, predators are immediately lethal to their victims, but parasites are not.

Direct Effects of Parasites

Following the convention established for predators, I consider parasites to have **consumptive effects** if they extract energy from their victims, or nonconsumptive effects if they do not. For example, trematodes can cause trait changes in their tadpole hosts both before and after infection. Tadpoles might increase their activity level to avoid becoming infected (a nonconsumptive effect [10]), and they might grow extra limbs after infection (a consumptive effect [11]). Although the amount of energy parasites extract from their hosts varies widely and can depend on infection intensity or load, any trait change that occurs after infection is consumptive in nature because parasites, by definition, extract energy from their hosts [12].

Similar to predators, parasites can induce victims to adopt defensive strategies that reduce the likelihood of consumption [13]. For example, healthy lobsters avoid sheltering with virus-infected conspecifics [14]; termites isolate corpses from the rest of the colony [15]; and sheep avoid foraging on vegetation contaminated with feces [16]. Such trait changes constitute nonconsumptive (or risk) effects, analogous to those triggered by predators, and they benefit the victim at the expense of the consumer. When infective stages are detectable, behavioral changes could be driven by fear, but, when they are less detectable, parasite avoidance might be motivated by disgust [17]. The mechanisms behind such trait changes are not fully understood, but, as for predator-induced trait changes, stress-induced hormonal responses are probably involved [18].

Similar to predators, parasites can also consume (or infect) their victims, but unlike predators (except micropredators; see Box 1), they can do so nonlethally. Therefore, parasites are capable of altering victim traits (behavior, development, morphology, physiology, or life history) through their consumptive effects. Due to the intimate and durable nature of the parasite–host interaction [12], these trait changes occur through a more diverse set of mechanisms than those induced by nonconsumptive effects. Specifically, parasites modify host behavior via neural, endocrine, neuromodulatory, and immunomodulatory pathways [19], and can modify other traits through mechanisms ranging from energy drain to mechanical disruption. For example, *Ribeiroia ondatrae*, a trematode parasite, causes

Glossary

Consumptive effect: a negative effect of one organism on another due to energy extraction. Consumptive effects can be lethal or nonlethal, encompassing predation and infection.

Consumptive trait-mediated indirect interaction: an indirect interaction in which one species (the initiator) consumes another species (the transmitter) nonlethally and alters its phenotype (i.e., trait), thereby changing its per capita effects on a third species (the receiver).

Density-mediated indirect effect (DMIE): an indirect interaction in which one species alters the density of another species, thereby changing its effects on a third species.

Macroparasite: an infectious agent that does not multiply within its host and does not necessarily kill it.

Microparasite: an infectious agent that multiplies within its host and does not necessarily kill it.

Micropredator: a free-living predator that only consumes part of its prey and does not kill it.

Nonconsumptive effect: a nonlethal effect of a consumer on its victim due to perceived predation or infection risk; does not involve energy extraction.

Parasitic castrator: an infectious agent that eliminates host fitness without killing its host.

Parasitoid: an infectious agent that kills its host as a required part of its development.

Trait-mediated indirect effect (TMIE): an indirect interaction in which one species (the initiator) alters the phenotype (i.e., trait) of another species (the transmitter), thereby changing its per capita effects on a third species (the receiver).

Trophically transmitted parasite: an infectious agent in a prey host that is transmitted to the next host in its life cycle via predation.

Box 1. Micropredators Can Cause Consumptive TMIEs

Similar to parasites, micropredators can consume a victim without killing it and, therefore, can trigger consumptive TMIEs. Carnivorous micropredators sometimes consume appendages of their victims (a consumptive effect), and loss of these structures can alter how victims interact with other species [59]. For instance, limb loss (which is often induced by predation) decreases feeding rates of the multiarmed sea star, *Heliaster helianthus* [60]. Although a few examples of carnivorous micropredator-induced TMIEs exist, the potential to cause TMIEs through their consumptive effects is best described for herbivorous micropredators [7,8]. For instance, the caterpillar *Pieris brassicae* develops faster and reaches larger body mass on plants previously attacked by aphids [61]. This effect is probably mediated by interference in signal transduction (a physiological trait change), because phloem-sucking aphids attenuate the phytohormones needed to defend against leaf-chewing herbivores, such as caterpillars [62]. The frequency and strength of micropredator-induced versus parasite-induced consumptive TMIEs remain open questions.

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missing, extra, or deformed limbs (a morphological trait change) in its amphibian hosts by perturbing cells involved in limb bud formation [11]. Trait changes associated with consumptive effects can benefit the parasite, the host, both, or neither. For example, behavioral changes induced by trophically transmitted parasites increase the likelihood of parasite transmission, often resulting in host death (e.g., [19]). Other trait changes associated with consumptive effects constitute host adaptations to minimize damage caused by parasites (e.g., [20]), or by-products of infection that benefit neither party (e.g., [21]). Thus, unlike predators, parasites can modify victim traits through their consumptive effects.

Indirect Effects of Parasites

Indirect effects occur when one species interacts with a second species, thereby altering its interaction with a third species [22]. For example, if consumers are lethal, they can alter victim density, thereby triggering DMIEs on species with which victims interact. If consumers are not immediately lethal, they can alter victim traits, thereby triggering TMIEs on species with which victims interact. In their classic paper, Werner and Peacor [23] defined trait-mediated indirect interactions as interactions in which one species (the initiator) alters the phenotype (i.e., trait) of another species (the transmitter), thereby changing its per capita effect on a third species (the receiver). They posited that 'ecological communities are replete with TMIEs' and called for studies examining species interactions and effects 'due to agents other than predators'. Next, I describe three types of indirect effect initiated by parasites. Given that consumptive DMIEs and nonconsumptive TMIEs are well documented in the predator–prey literature, I discuss these interaction types only briefly, concentrating instead on consumptive TMIEs.

Consumptive Density-Mediated Indirect Effects

Similar to predators, parasites can kill their victims, thereby triggering DMIEs. For example, rinderpest virus was eradicated from the Serengeti during the 1960s, prompting the detection of a density-mediated trophic cascade. Wildebeest populations that were formerly kept in check by the deadly virus irrupted, thereby increasing grazing pressure on grasses (Figure 2, Key Figure). Thus, when it was present, the pathogen facilitated grasses by killing herbivores, a consumptive DMIE. Direct effects of parasites on host density can also propagate upwards to other consumers (e.g., [23]), or sideways through nontrophic interactions (e.g., [24]) (Figure 2). If the receiver of a consumptive DMIE is a resource of the host, then it benefits from the interaction, but if the receiver is a consumer of the host, then it can benefit or be harmed. Hence, when they are lethal to their hosts, parasites can cause DMIEs through their consumptive effects, and these DMIEs are similar to those caused by predators.

Nonconsumptive Trait-Mediated Indirect Effects

Similar to predators, parasites can cause victims to adopt defensive strategies that reduce the risk of consumption, suggesting that the ecology of fear applies beyond predation. Such nonconsumptive

Key Figure

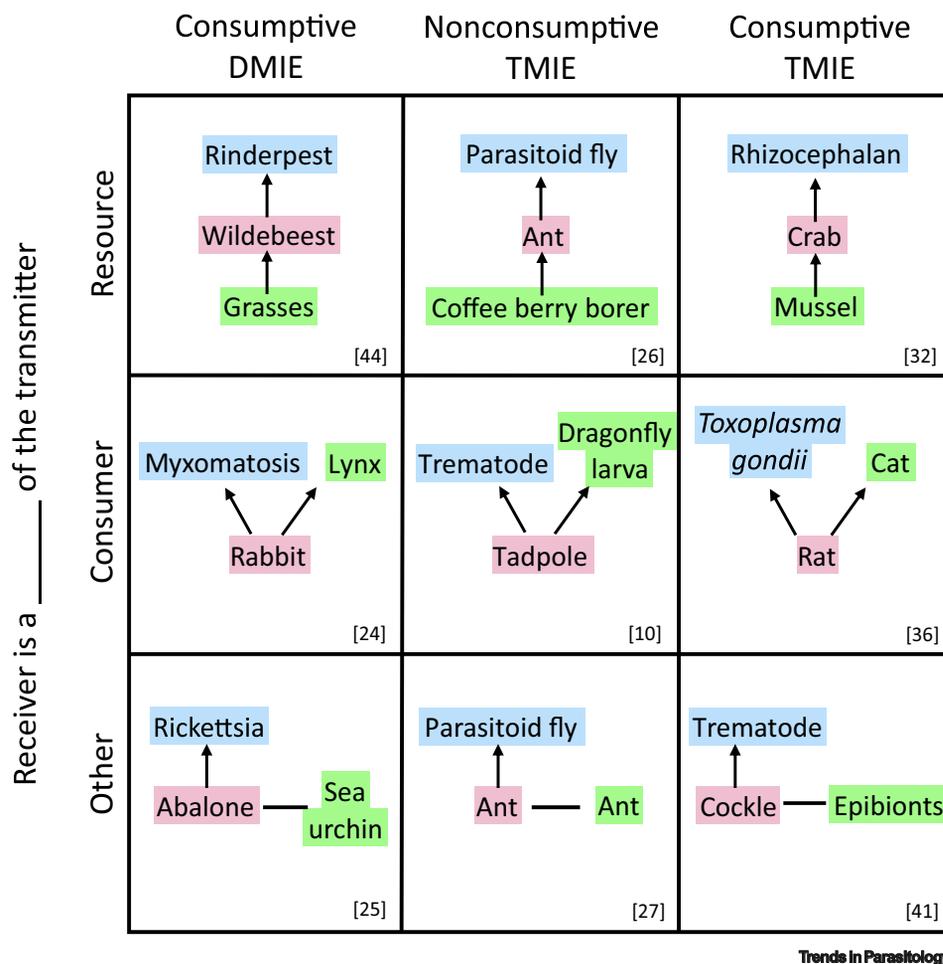## Examples of Parasite-Induced Indirect Effect Types

Figure 2. Examples of consumptive density-mediated indirect effects (DMIEs), nonconsumptive trait-mediated indirect effects (TMIEs), and consumptive TMIEs. Initiators (parasites) are highlighted in blue, transmitters (hosts) in pink, and receivers in green. The receiver is a resource of the transmitter (corresponding to Figure 3A in the main text), a consumer of the transmitter (corresponding to Figure 3B the main text), or a species that interacts with the transmitter nontrophically (corresponding to Figure 3C the main text). (See [10,24–27,32,36,41,44].)

direct effects can trigger TMIEs on other species. For example, in the presence of parasitoid flies, *Azteca* ants modify their behavior to avoid infection, thereby benefitting their prey, the coffee berry borer [26]. Thus, an indirect interaction between the parasitoid and the coffee berry borer is mediated through the trait (i.e., behavior) of the ant (Figure 2). Similar to consumptive effects, nonconsumptive effects of parasites on hosts can also propagate upwards to other consumers (e.g., [26]), or sideways through nontrophic interactions (e.g., [27]) (Figure 2). If the receiver of a nonconsumptive TMIE is a resource of the host, then it usually benefits from the interaction, although exceptions exist (e.g., [28]). If the receiver is a consumer of the host, then it can benefit or be harmed, depending on whether the adaptive responses of the hosts to the two consumers are similar to, or different from, one another

[23]. Hence, parasites can trigger TMIEs through their nonconsumptive effects, and these TMIEs are similar to those caused by predators.

Consumptive Trait-Mediated Indirect Effects

Unlike predators (except micropredators; see Box 1), parasites can consume a victim without killing it. This raises the potential for parasites to cause consumptive TMIEs (Figure 1), indirect interactions in which one species (the initiator) consumes another species (the transmitter) nonlethally and alters its phenotype (i.e., trait), thereby changing its per capita effect on a third species (the receiver). Although parasites are known to trigger TMIEs through their consumptive effects (Table 1), consumptive TMIEs have not been properly distinguished from nonconsumptive TMIEs; neither have they been considered within the theoretical framework of indirect interactions. Similar to other types of indirect effect, consumptive TMIEs qualify as a type of higher-order interaction (HOI [29]), and can arise through three interaction motifs (Figure 3), which I describe here.

The three-species food chain (Figure 3A), or trophic cascade, was first conceived with predators in mind [30]. However, parasites can also trigger trophic cascades, and a significant proportion of those cascades involve consumptive TMIEs [31]. For example, infection with a rhizocephalan barnacle reduces the activity level of a mud crab, ultimately benefitting the resource of the crab, a mussel [32] (Figure 2). Other parasite-induced trophic cascades involving consumptive TMIEs come at a cost to the receiver (Table 1). Hence, parasites can benefit or harm the resource of their host through consumptive TMIEs.

The shared resource web (Figure 3B) embodies classic exploitative competition, and several types of consumptive TMIE fit into this motif. Trait changes associated with infection can increase host vulnerability to predation (Table 1). For example, predators selectively prey on red grouse that are infected by a cecal nematode, *Trichostrongylus tenuis* [33]. The mechanism behind this effect is unknown, but it could be mediated through a physiological trait change; birds with heavy worm burdens emit more scent and, therefore, are more vulnerable to mammalian predators. Through such consumptive TMIEs, parasites can facilitate predators, but these interactions are usually maladaptive for the parasite and the host, because their fitness is eliminated upon predation. However, at the population level, this interaction could be adaptive for the host; by targeting infected individuals and removing them from the population, predators could help maintain healthy herds [34].

Whereas many parasites die when their host is eaten, others have adapted to survive predation and infect the predator [35]. Such trophically transmitted parasites often manipulate host traits, thereby increasing the likelihood of predation. For example, by altering the aversion of its hosts to predator odors, *Toxoplasma gondii* increases the likelihood that infected rodents will be eaten by cats [36] (Figure 2). Other trophically transmitted parasites incapacitate (a physiological trait change), alter microhabitat choice by (a behavioral trait change), or modify the appearance of (a morphological trait change) their hosts, thereby increasing the likelihood of transmission (Table 1). Given that trophically transmitted parasites infect both the prey and its predator, these consumptive TMIEs benefit the initiator and the receiver at the expense of the transmitter, and could be considered as a special case of intraguild predation.

Whereas some parasites alter host vulnerability to predators, others alter host vulnerability to other parasites. Given that parasites form long-lasting (durable) relationships with their hosts [12], most hosts are infected by multiple species [37], and co-infecting parasites can facilitate one another through consumptive TMIEs (Table 1). For example, by modulating the immune system of the host, gastrointestinal nematodes facilitate bovine TB in African buffalo [38]. Conversely, co-infecting parasites can inhibit one another. For instance, parasitic cyst nematodes reduce the biomass and flower production of a root hemiparasitic plant with which they share a host plant [39]. These examples demonstrate that co-infecting parasites can interact with one another through consumptive TMIEs. However, classifying interactions between co-infecting parasites as consumptive TMIEs requires

Consumer strategy	Initiator	Transmitter	Receiver	Effect on receiver	Refs
Three-species food chains (see Figure 3A)					
Parasitic castrator	Larval trematode (*Cryptocotyle lingua*)	Snail (*Littorina littorea*)	Macroalgae	Positive	[73]
	Larval trematode (*Trichobilharzia physellae*)	Snail (*Physa actua*)	Algae	Negative	[74]
	Larval trematodes	Snail (*Hydrobia ulvae*)	Algae	Negative	[75]
	Rhizocephalan (*Loxothylacus panopaei*)	Mud crab (*Eurypanopeus depressus*)	Mussel (*Brachidontes exustus*)	Positive	[32]
Parasitoid	Tachinid fly (*Drino rhoeo*)	Carolina sphinx moth (*Manduca sexta*)	Sacred datura (*Datura wrightii*)	Positive	[76]
	Wasp (*Zele deceptor*)	Autumnal moth (*Epirrita autumnata*)	Mountain birch (*Betula pubescens* ssp. *czerepanovii*)	Positive	[77]
	Wasp (*Apanteles obliquae*)	Jute hairy caterpillar (*Spilosoma obliqua*)	Jute (*Corchorus* spp.)	Positive	[78]
	Wasp (*Cotesia glomerata*)	Cabbage butterfly (*Pieris brassicae*)	Black mustard (*Brassica nigra*)	Positive	[79]
	Wasp (*Pteromalus albipennis*)	Tephritid flies (*Tephritis femoralis*)	Thistle (*Saussurea nigrescens*)	Negative	[80]
	Wasps	African cotton leafworm (*Spodoptera littoralis*)	Maize (*Zea mays*)	Positive	[81]
	Wasp (*Cotesia rubecula*)	Small cabbage white butterfly (*Pieris rapae*)	Thale cress (*Arabidopsis thaliana*)	Positive	[82]
	Wasp (*Diplazon laetatorius*)	Marmalade hoverfly (*Episyrphus balteatus*)	Pea aphid (*Acyrthosiphon pisum*)	Positive	[83]
	Wasp (*Microplitis tristis*)	Lynchis moth (*Hadena bicruris*)	White campion (*Silene latifolia*)	Positive	[84]
	Wasp (*Cotesia plutellae*)	Diamondback moth (*Plutella xylostella*)	Cabbage (*Brassica oleracea*)	Negative	[85]
Microparasite	Microsporidian (*Thelohania contejeani*)	White clawed crayfish (*Austropotamobius pallipes*)	Amphipod (*Gammarus pulex*)	Positive	[86]
Three-species shared-resource webs (see Figure 3B)					
Trophically transmitted parasite	Larval trematode (metacercariae) (*Euhaplorchis californiensis*)	Killifish (*Fundulus parvipinnis*)	Birds	Positive	[20]
	Larval acanthocephalans	Amphipods	Ducks	Positive	[87]
	Toxoplasma gondii	Rat (*Rattus norvegicus domestica*)	Cat (*Felis catus*)	Positive	[36]
Parasitic castrator	Larval trematode	Snail (*Physa integra*)	Fish, crayfish	Positive	[88]

**Table 1. Representative Examples of Parasite-Induced Consumptive Trait-Mediated Indirect Effects Based on Food Web Motifs**

Consumer strategy	Initiator	Transmitter	Receiver	Effect on receiver	Refs
Macroparasite	Microsporidian (*Glugea anomala*)	Stickleback (*Gasterosteus aculeatus*)	Cichlid fish (*Tilapia mariae*)	Negative	[89]
	Gastrointestinal nematodes	Buffalo (*Syncerus caffer*)	TB (*Mycobacterium bovis*)	Positive	[38]
	Nematode (*Heligmosomoides polygyrus*)	Wood mouse (*Apodemus sylvaticus*)	Tapeworm (*Catenotaenia pusilla*)	Positive	[90]
	Gastrointestinal nematodes	Wood mouse (*Apodemus sylvaticus*)	Protozoan (*Eimeria* sp.)	Negative	[91]
	Nematode (*Heterodera trifolii*)	Red clover (*Trifolium pretense*)	Yellow rattle (*Rhinathus serotinus*)	Negative	[39]
	Nematode (*Trichostrongylus tenuis*)	Red grouse (*Lagopus lagopus scoticus*)	Various predators	Positive	[33]
Parasitic castrator	Rhizocephalan (*Loxothylacus panopaei*)	Mud crab (*Eurypanopeus depressus*)	Blue crab (*Callinectes sapidus*)	Positive	[92]
Parasitoid	Nematomorph (*Paragordius tricuspidatus*)	Cricket	Various predators	Positive	[63]
	Chytrid fungus (*Polycaryum laeve*)	*Daphnia pulicaria*	Various fish	Positive	[93]
	Phorid fly (*Pseudacteoin* spp.)	Ant (*Azteca sericeasur*)	Rove beetle (*Myrmedonota xipe*)	Positive	[94]
Microparasite	Yeast (*Candida humicola*)	Tadpole (*Rana aurora*)	Newt (*Taricha granulosa*)	Positive	[95]
	Fungus (*Verticillium dabliae*)	Cotton (*Gossypium hirsutum*)	Spider mite (*Tetranychus urticae*)	Negative	[96]
	Fungus (*Marssonina betulae*)	Birch (*Betula pendula*)	Aphid (*Euceraphis betulae*)	Positive	[97]
Nontrophic indirect effects (see Figure 3C)					
Trophically transmitted parasite	Larval trematode (*Curtuteria australis*)	Cockle (*Austrovenus stutchbury*)	Epibionts	Positive/ negative	[41]
Macroparasite	Gall wasp (*Andricus quercuscalifornicus*)	Oak (*Quercus lobata*)	Jumping spiders (Salticidae)	Positive	[68]
	Mistletoe (*Phoradendron juniperinum*)	Juniper (*Juniperus monosperma*)	Mycorrhizae	Negative	[98]
Microparasite	Microsporidian (*Nosema bombi*)	Bumble bee (*Bombus* spp.)	Various plants	Negative	[99]
	Malaria (*Plasmodium azurophilum*)	Lizard (*Anolis gingivinus*)	Lizard (*Anolis watsii*)	Positive	[100]

Table 1. Continued

knowledge of the mechanism, because, in addition to host-mediated interactions, co-infecting parasites can also interact with one another directly (e.g., [40]).

Thus far, I have discussed consumptive TMIEs in which receivers are either resources (Figure 3A) or consumers (Figure 3B) of transmitters, but receivers can also interact with transmitters through nontrophic interactions, such as interference competition or mutualism (Figure 3C). For example, in New Zealand mudflats, cockle shells represent the only hard surface on which epibionts, including limpets and anemones, can establish. By altering the ability of cockles to bury themselves in mud, trematodes facilitate limpet and suppress anemone populations [41] (Figure 2). Thus, the receivers of consumptive TMIEs need not be resources or consumers of transmitters; they can also interact with transmitters through nontrophic interactions (Table 1).
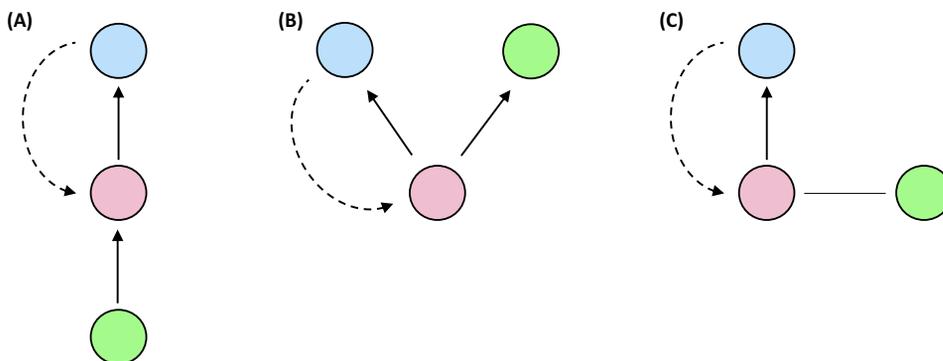
Trends in Parasitology

Figure 3. Food Web Motifs Depicting Three Types of Trait-Mediated Indirect Effect (TMIE).
(A) Three-species food chains, (B) three-species shared-resource webs, and (C) nontrophic indirect effects. Blue circles represent a parasite that initiates a TMIE (the 'initiator'), pink circles represent its host, which responds phenotypically (the 'transmitter'), and green circles represent the species that experiences the indirect effect (the 'receiver'). Solid arrows point in the direction of energy flow, and the solid line indicates a nontrophic interaction. Dashed lines indicate an effect on a trait (behavior, physiology, morphology, development, or life history) of the species to which the arrow points. Adapted from [23].

### Relative Importance of Indirect Interaction Types

As detailed earlier, parasites can trigger three (as opposed to two) types of indirect effect through their consumptive and nonconsumptive direct effects. The relative importance of each indirect interaction type probably depends on their frequency and strength. Considering only the three-species food chain motif (Figure 3A), 60% of reported parasite-induced trophic cascades involve consumptive DMIEs, while 45% involve consumptive TMIEs, and only 6% involve nonconsumptive TMIEs [31]. Although no study has compared the relative strength of all three indirect interaction types, this would be possible to measure using the common currency of the effect on the receiver, as has been done for predator-induced indirect effects (e.g., [42]). For example, if the receiver is a resource of the host (Figure 3A), then the strength of each indirect interaction type could be measured by quantifying the biomass of this resource through time in the presence or absence of an infectious agent. I predict that highly lethal parasites (e.g., parasitoids) will elicit strong avoidance responses from hosts, thereby strengthening nonconsumptive TMIEs relative to consumptive TMIEs and DMIEs. However, the relative strength of consumptive TMIEs should increase with decreasing parasite lethality, because hosts will neither engage in potentially costly avoidance responses (thereby triggering nonconsumptive TMIEs) nor die (thereby triggering DMIEs) when faced with an insubstantial threat. Furthermore, I expect the magnitude of the effect on the receiver to depend on the temporal scale over which it is measured; nonconsumptive TMIEs should dominate before the host becomes infected, consumptive TMIEs should operate after infection but before death, and DMIEs should occur once the host succumbs to infection. Opposing effects at different timescales would also be possible.

Effects at Higher Levels of Ecological Organization

In addition to their effects on individual receivers, parasites can also have population-, community-, and ecosystem-level consequences via their indirect effects. For example, nematode infection increases snowshoe hare vulnerability to predation, causing hare populations to cycle, and these cycles are stronger than those caused by parasite-induced reduction in hare fecundity [43]. At the community level, one indirect effect can trigger another, forming so-called 'indirect interaction webs' [7,8] (Box 2), which can affect community structure and function, including biodiversity (Box 3). Parasite-induced indirect effects might even affect ecosystem function. For example, before it was eradicated

Box 2. Parasite-Induced Indirect Interaction Webs

Indirect effects can reverberate at the community level if the receiver of one indirect effect initiates another. Indirect interaction webs were conceived with herbivore–plant interactions in mind [7,8], but the theory should apply similarly to parasite–host interactions. For example, crickets infected by horsehair worms seek and jump into water, increasing their vulnerability to fish predators [63], a consumptive TMIE. However, the indirect effects do not end here. When the diet of fish is supplemented by crickets, they consume fewer benthic invertebrates, which, in turn, increases predation pressure on algae [64] (Figure I). Thus, the parasite triggers an indirect effect that propagates through the community. By linking multiple indirect effects (including nontrophic indirect links), indirect interaction webs depict community networks more realistically than do traditional food webs [7]. Understanding linkages between indirect interactions can provide valuable insights into the mechanisms by which parasites shape community structure and function.
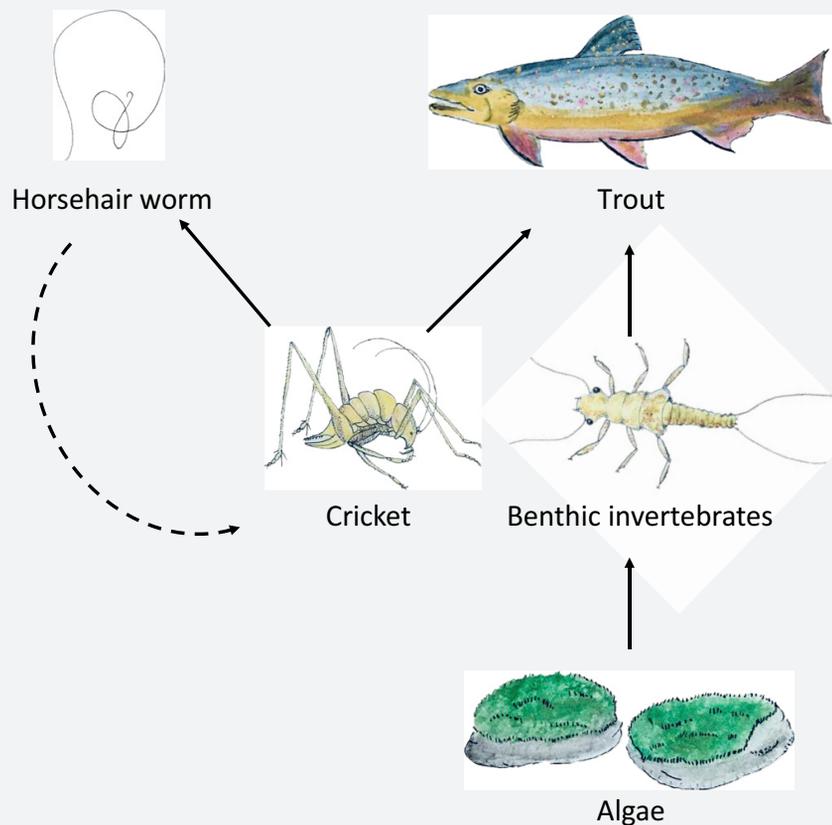
Trends in Parasitology

Figure I. A Parasite-Induced Indirect Interaction Web.

Horsehair worms manipulate the behavior of crickets (dashed arrow), causing them to seek and jump into water. In turn, trout eat more crickets and fewer benthic invertebrates, thereby increasing predation pressure on algae. Adapted from [64]. Artwork: Savannah Cooper.

Box 3. Parasites Influence Biodiversity via Indirect Effects

When parasites alter the traits of their hosts through consumptive and nonconsumptive effects, they create diverse phenotypes within host populations. Indeed, infected and uninfected hosts might effectively act as two different species, and parasites that cause intensity-dependent pathology could create a continuous distribution of phenotypes within a host population. By increasing trait diversity within host populations [65], parasites expand ecological niches available to species that interact with their hosts. For instance, the trematode *Euhaplorchis californiensis* alters the behavior of its killifish host, increasing the likelihood that it will be eaten by the final host of the parasite, a bird [20], rendering this a consumptive TMIE. By making infected fish easier to capture, the parasite provides a subsidy to birds [66]. However, although some parasites appear to promote coexistence by facilitating species with which hosts interact, others might hinder coexistence [65] by negatively affecting receiver species (see Table 1 in the main text). A recent theoretical study showed that parasites are most likely to promote coexistence at intermediate intensities [67], where the trait diversity of hosts is maximized.

Although experimental evidence is limited, at least one study demonstrates that parasites can promote diversity via their indirect effects. The California gall wasp (a macroparasite; see Table 1 in the main text) is an ecosystem engineer: it creates oak apple galls on valley oak trees. Galls, which can persist for many years after wasps vacate them, are colonized by predatory jumping spiders. Thus, an indirect interaction between the wasps and the spiders is mediated by a morphological trait change of the oak tree, constituting a consumptive TMIE. In turn, jumping spiders suppress dominant herbivorous arthropods on oak trees, thereby increasing beta diversity [68]. Thus, although free-living species diversity is known to promote parasite diversity [69], parasites might also promote free-living species diversity via their indirect effects. This could explain why parasites are positively associated with biodiversity [4,70–72], despite negative effects on hosts.

in the Serengeti, rinderpest facilitated grasses via a DMIE. This, in turn, promoted fire and suppressed trees, altering carbon cycling in this iconic ecosystem [44].

How might the ecosystem-level effects of parasites compare to those of predators? Given that parasites are not immediately lethal to their victims, both their consumptive and nonconsumptive effects could be weaker than those of predators [31]. Nevertheless, infectious processes are recognized to shape ecosystem structure and function, possibly to the same extent as predation [45,46]. How is this possible? Peacor and Werner [47] suggested that nonconsumptive effects of predators contribute strongly to their net effects because nonconsumptive effects can influence the entire prey population, whereas consumptive effects only influence a few individuals. I posit that, because parasites can alter host traits through both consumptive and nonconsumptive effects, this explanation might apply doubly. Even if the effects of individual parasites on hosts are weaker than those of predators, their net effects could be as strong or stronger because they are more abundant than predators [46], often affect nearly every individual in a host population [12], and can do so through both consumptive and nonconsumptive effects.

Concluding Remarks

Over the past two decades, calls to include parasites in food webs (e.g., [48]) have been heeded (e.g., [49–53]), increasing our understanding of the role of parasites in ecological communities (e.g., [54,55]). Nevertheless, incorporating parasites into food webs will not reveal their full role in ecosystems because food webs do not account for trait changes. Specifically, if a host reacts to the presence of a parasite by altering its phenotype, this trait change can alter the per capita effect of the host on other species, even in the absence of an effect on host density. TMIEs were conceptualized to account for this context dependence [23] and have been instrumental in revealing the role of predators in ecosystems (e.g., [56]). Here, I demonstrate that applying a framework established to study predator–prey interactions to parasite–host interactions can help reveal the role of parasites in ecosystems. Although all consumer types can trigger DMIEs through their consumptive effects and TMIEs through their nonconsumptive effects, parasites can have their cake and eat it too: they can simultaneously consume a host and alter its traits, raising the potential for consumptive TMIEs. In turn, all types of parasite-induced indirect effect can reverberate at higher levels of ecological organization. Similar

Outstanding Questions

Infectious agents trigger three (as opposed to two) types of indirect interaction. What is the relative frequency and strength of different indirect interaction types? Does this depend on parasite lethality or temporal scale?

How do the frequency and strength of indirect effects induced by parasites compare to those induced by predators?

How do the frequency and strength of consumptive TMIEs induced by parasites compare to those induced by micropredators?

Are parasite-induced indirect effects as important as parasite-induced direct effects in shaping ecosystem structure and function?

Most consumer-induced higher-order interactions described in the literature involve nontrophic interactions between initiators and transmitters, whereas consumptive TMIEs involve trophic interactions between initiators and transmitters. How does this affect predictions about biodiversity, stability, and other ecosystem properties?

Are parasites more likely to facilitate or prevent coexistence via their indirect effects, and does this depend on parasite prevalence?

How common and how important are parasite-induced indirect interaction webs in ecological communities?

What other novel insights can be gained by applying predator–prey theory to parasite–host dynamics?

to predator-induced indirect effects, parasite-induced indirect effects may be as important, if not more important, than their direct effects in shaping ecosystem structure and function [22].

Future studies should concentrate on quantifying the importance (frequency and strength) of different parasite-induced indirect interaction types (see Outstanding Questions). Testing the relative strength of indirect interaction types using a single initiator, transmitter, and receiver species would be more powerful than comparing strengths of these effects across studies (and species), because effect strength should vary with parasite lethality. However, a meta-analytical approach would allow comparison across parasite types. Finally, we need to investigate the relative importance of each indirect interaction type in affecting ecosystem structure and function. Most consumer-induced higher-order interactions described in the literature (i.e., nonconsumptive TMIEs) involve nontrophic interactions between initiators and transmitters, whereas consumptive TMIEs involve trophic (albeit nonlethal) interactions between initiators and transmitters. How this should alter predictions about biodiversity, stability, and other ecosystem properties remains unexplored. Moving forward, I recommend that researchers treat consumptive and nonconsumptive TMIEs as distinct (but related) phenomena, rather than risking confusion by lumping them together. By considering parasites within the theoretical framework established for predator–prey dynamics, this paper advances recent efforts (e.g., [11,15,31,57,58]) to unite predator–prey and parasite–host theory under a general consumer–resource framework.

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