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REVIEWS

Review

Infectious Agents Trigger Trophic Cascades

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Most demonstrated trophic cascades originate with predators, but infectious agents can also cause top-down indirect effects in ecosystems. Here we synthesize the literature on trophic cascades initiated by infectious agents including parasitoids, pathogens, parasitic castrators, macroparasites, and trophically transmitted parasites. Like predators, infectious agents can cause density-mediated and trait-mediated indirect effects through their direct consumptive and nonconsumptive effects respectively. Unlike most predators, however, infectious agents are not fully and immediately lethal to their victims, so their consumptive effects can also trigger trait-mediated indirect effects. We find that the frequency of trophic cascades reported for different consumer types scales with consumer lethality. Furthermore, we emphasize the value of uniting predator–prey and parasite–host theory under a general consumer–resource framework.

Trends

Trophic cascades originate with consumers (predators or infectious agents).

The frequency of trophic cascades reported for different consumer types scales with consumer lethality.

Unlike most predators, infectious agents can consume (i.e., infect) a victim without killing it. This raises the potential for infectious agents to cause consumptive trait-mediated indirect effects, which can be positive or negative.

Infectious agents seem less likely than predators to cause consumptive density-mediated indirect effects and nonconsumptive trait-mediated indirect effects, but are uniquely capable of causing consumptive trait-mediated indirect effects.

Unification of predator–prey and parasite–host theory under a general consumer–resource framework will benefit both fields.

Infectious Agents Can Trigger Trophic Cascades

What does a sea otter have in common with a bacterial pathogen? Superficially, these organisms could hardly be more different. However, they are united in their role as top-down forces in ecosystems: both control sea urchin populations, allowing kelp to flourish [1,2]. Like predators, infectious agents are typically placed one level above their victims in food webs [3], an ideal vantage point from which to drive top-down direct and indirect effects, including **trophic cascades** (see Glossary). Unlike most predators, however, infectious agents do not necessarily kill their victims, which could diminish their ability to cause trophic cascades through changes in host density. Nevertheless, many infectious agents do kill their hosts, and others could trigger trophic cascades through changes in host behavior [4]. The prolonged (durable) and physically intimate relationship between infectious agents and their hosts [5] makes this especially likely. Here we review empirical studies on trophic cascades triggered by infectious agents including **parasitoids**, **pathogens**,**parasitic castrators**, **macroparasites**, and **trophically transmitted parasites** examine the potential for infectious agents to cause direct (consumptive and nonconsumptive) and indirect (density-mediated and trait-mediated) effects, and discuss unique challenges and opportunities that arise in the effort to detect infectious-agent-induced cascades.

Literature Survey

Following Ripple *et al*. [6], we conducted a literature survey using the Web of Science for articles published between 1986 and 2015 containing the terms 'trophic cascade(s)' and parasit*, infect*, or pathogen in the title, abstract, or keywords. Our search resulted in 173 articles. Like Ripple *et al*. [6], we found an exponential rise in use of the term 'trophic cascade(s)' in conjunction with the term 'parasit*', 'infect*', or 'pathogen' (Figure 1), indicating that detection of trophic cascades involving infectious agents has paralleled recognition of trophic cascades involving predators (Figure 2A), albeit with around a tenth as many infectious-agent-induced cascades reported over the same time period. We examined these articles and included them

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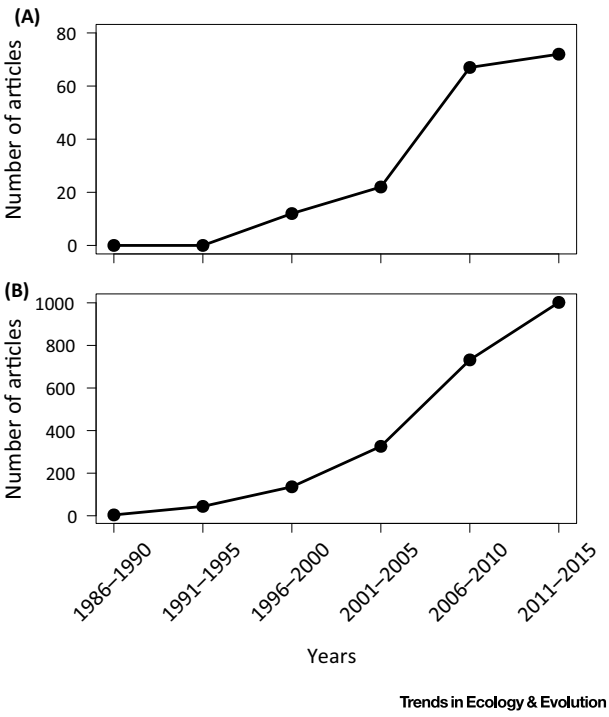

Figure 1. Trends in the Number of Articles Published on Infectious Agent-Induced Trophic Cascades and All Trophic Cascades. Number of articles published over time (A) using the word 'trophic cascade(s)' in conjunction with the word 'parasit*', 'infect*', or 'pathogen', and (B) using the word 'trophic cascade(s)' (data from Ripple *et al*. [6]). Note the order of magnitude difference in the *y*-axes. The trend through time in detection of infectious-agent-induced trophic cascades has paralleled the trend for all trophic cascades, including the steep rise following the turn of the century.

in Table 1 if they demonstrated a trophic cascade triggered by an infectious agent. To gather additional examples, we expanded our survey by (i) searching for articles containing the term 'tritrophic' in conjunction with the term 'parasit*', 'infect*', or 'pathogen' in the title, abstract, or keywords, (ii) consulting known works (e.g., [7]) that have examined the potential for infectious agents to induce trophic cascades, and (iii) consulting disease ecologists. Our search yielded 47 infectious-agent induced cascades including 30 caused by parasitoids, 13 caused by pathogens, four caused by parasitic castrators, zero caused by macroparasites, and zero caused by trophically transmitted parasites (Table 1). While most infectious agents caused positive indirect effects, 13% (6/47) caused negative indirect effects (Table 1; Box 1). Although most infectious agent-induced cascades occurred in terrestrial ecosystems, several occurred in freshwater and marine ecosystems (Table 1). We note that only around a third of the studies cited in Table 1 use the term 'trophic cascade(s)' in the title, abstract, or keywords, and we encourage more general use of this term when top-down indirect effects of infectious agents are detected.

Most demonstrated trophic cascades caused by infectious agents involve parasitoids, organisms that kill their host as a normal and required part of their development [8]. For example, parasitoid wasps attack beetles that feed on legume seeds. Parasitized beetles consume seeds less extensively than do uninfected conspecifics, with consequences for germination success [9] (Figure 2B). This demonstrates that direct negative effects of parasitoids on hosts can benefit plants, which is the basis for many biological control programs (Box 2). In fact, when attacked by herbivores, some plants produce volatile chemicals that attract parasitoids [10], strongly suggesting that parasitoids can indirectly protect plants through their top-down effects.

In contrast to parasitoids, pathogens (e.g., viruses) multiply within their host and do not necessarily kill it [8]. Though less often reported than parasitoid-induced cascades (Table 1), several pathogen-induced cascades have been demonstrated. For example, the eradication of

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.
Density-mediated indirect effect (DMIE): an indirect effect of a consumer on its victim's resource mediated by a change in victim population density.
Knock-on effect: indirect effects of consumers that spin off from the main interaction chain [6].
Macroparasite: a typical parasite, that is, an infectious agent that does not multiply within its host and does not necessarily kill it, for example, an adult trematode in a bird [8].
Micropredator: a free-living predator that only consumes part of its prey and does not kill it, for example, a mosquito [8].
Nonconsumptive effect: a nonlethal effect of a consumer on its victim due to perceived risk of predation or infection.
Parasitic castrator: an infectious agent that eliminates host fitness, typically without killing its host. Because parasitic castrators are not lethal, changes in host density are necessarily delayed by one generation, for example, a larval trematode in a snail [8].
Parasitoid: an infectious agent that requires the death of its host as a necessary part of its development, for example, a wasp that develops and pupates within a caterpillar [8].
Pathogen: a microparasite, an infectious agent that multiplies within its host and does not necessarily kill it, for example, rinderpest, a viral pathogen of ungulates [8].
Trait-mediated indirect effect (TMIE): an indirect effect of a consumer on its victim's resource mediated by a change in victim traits (e.g., behavior, physiology, etc.).
Trophically transmitted parasite: an infectious agent in a prey host that is transmitted to the next host in its life cycle via predation, for example, a larval trematode in a fish, which is transmitted to a bird when its host fish is consumed [8].
Trophic cascade: an indirect species interaction that originates with a consumer (predator or infectious agent) and spreads downward through the food web.

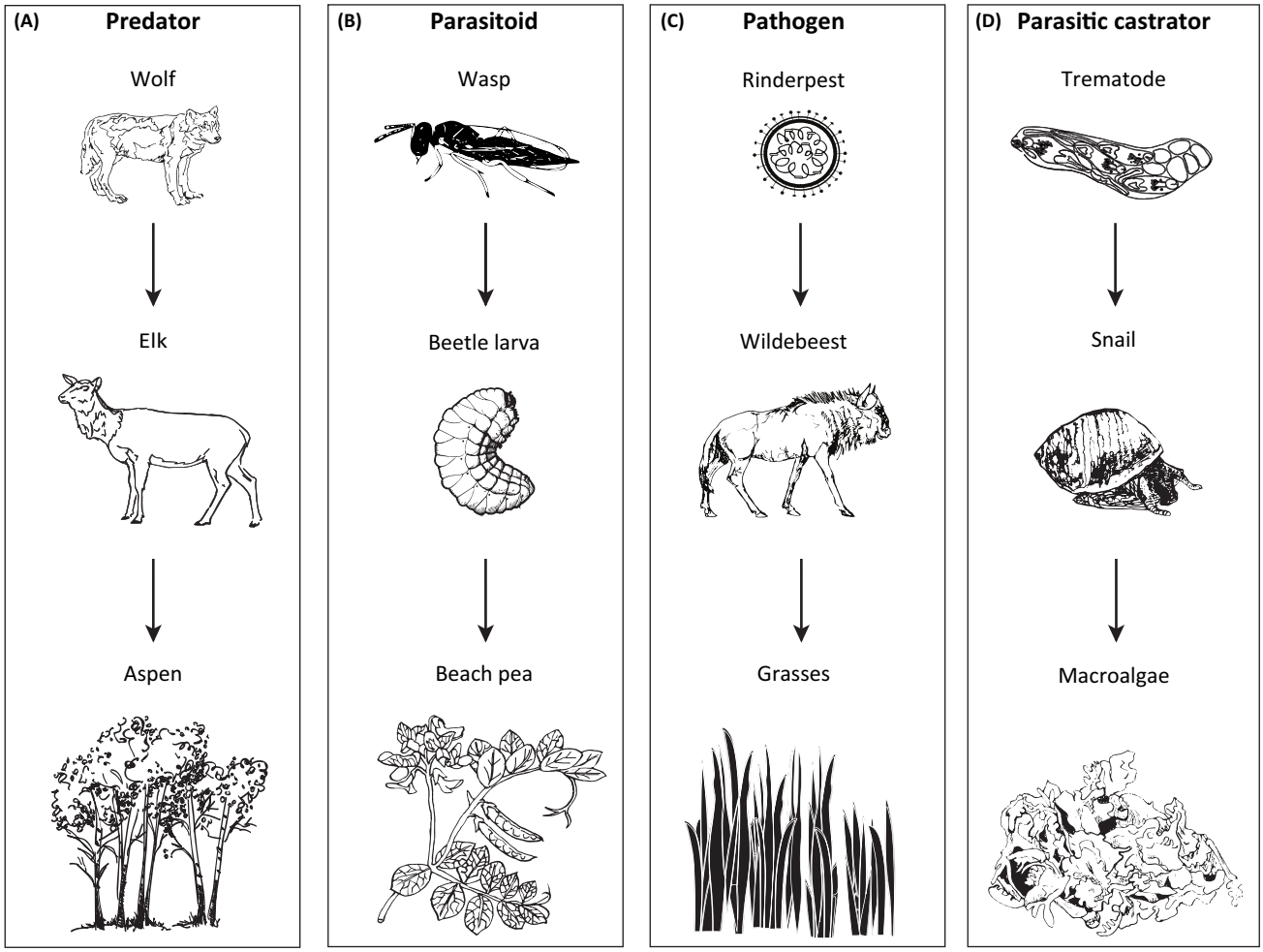

Figure 2. Trophic Cascades Caused by Different Types of Consumers. (A) Classic predator-induced cascade, (B) parasitoid-induced cascade, (C) pathogen-induced cascade, (D) parasitic-castrator-induced cascade. Our literature search yielded no examples of macroparasite-induced cascades, and trophically transmitted parasites cannot cause cascades.

rinderpest from the Serengeti in the 1960s prompted the detection of a pathogen-induced trophic cascade (Figure 2C). Once held in check by the disease, grazer populations increased, with consequences for plant community composition [11]. Like rinderpest, most pathogens that have triggered trophic cascades are lethal to their hosts. However, the variable lethality of pathogens (which is related to reproduction within the host) could explain why pathogen-induced trophic cascades are less often reported than those triggered by parasitoids.

Like parasitoids, parasitic castrators eliminate host fitness, but unlike parasitoids, they typically do so without killing their host [8]. Resource consumption by infected hosts could be affected immediately, but changes in host density are necessarily delayed by one generation. Though less often reported than parasitoid- and pathogen-induced cascades, parasitic-castrator-induced cascades have been demonstrated (Table 1). For example, in a field experiment, digenetic trematode infection reduced grazing by an intertidal snail, increasing ephemeral macroalgal cover [12] (Figure 2D). This demonstrates that nonlethal infectious agents can also trigger trophic cascades, but parasitic-castrator-induced cascades are infrequently reported, perhaps because castrators are typically not lethal to their hosts.

Table 1. Infectious Agent-Induced Trophic Cascades

Infectious agent type	Infectious agent	Host	Resource	Effect on resource	Consumptive/ nonconsumptive	DMIE[a]/ TMIE[b]	Ecosystem	Refs
Parasitoid	Entomopathogenic nematode	Ghost moth	Bush lupine	Positive	Consumptive	DMIE	Terrestrial	[48]
Parasitoid	Entomopathogenic nematode	Black vine weevil	Azalea	Positive	Consumptive	DMIE	Terrestrial	[49]
Parasitoid	Mite	Weevil	Yellow starthistle	Positive	Consumptive	DMIE	Terrestrial	[50]
Parasitoid	Phorid fly	Ant	Fall armyworm	Positive	Nonconsumptive	TMIE	Terrestrial	[21]
Parasitoid	Phorid fly	Ant	Coffee berry borer	Positive	Nonconsumptive	TMIE	Terrestrial	[51]
Parasitoid	Tachinid fly	Carolina sphinx moth	Sacred datura	Positive	Consumptive	TMIE	Terrestrial	[52]
Parasitoid	Wasp	Jute hairy caterpillar	Jute	Positive	Consumptive	TMIE	Terrestrial	[53]
Parasitoid	Wasp	Small cabbage white butterfly	Cabbage	Positive Negative	Consumptive	DMIE TMIE	Terrestrial	[54,55]
Parasitoid	Wasps	Foxglove aphid	Pepper	Negative	Consumptive Nonconsumptive	DMIE TMIE	Terrestrial	[56]
Parasitoid	Wasp	Strawberry leaf beetle	Meadowsweet	Positive	Consumptive	DMIE	Terrestrial	[57]
Parasitoid	Wasps	Wheat stem sawfly	Wheat	Positive	Consumptive	DMIE TMIE	Terrestrial	[58,59]
Parasitoid	Wasps	Moth	Wild petunia	Positive	Consumptive	DMIE TMIE	Terrestrial	[60,61]
Parasitoid	Wasps	Unidentified moth	Wild petunia	Positive	Consumptive	DMIE	Terrestrial	[62]
Parasitoid	Wasp	Arrowhead scale	Satsuma mandarin	Positive	Consumptive	DMIE	Terrestrial	[63]
Parasitoid	Wasp	Sugarcane borer moth	Artificial diet	Positive	Consumptive	TMIE	Terrestrial	[64]
Parasitoid	Wasp	Cabbage butterfly	Black mustard	Positive	Consumptive	TMIE	Terrestrial	[65]
Parasitoid	Wasps	African cotton leafworm	Maize	Positive	Consumptive	TMIE	Terrestrial	[66]
Parasitoid	Wasp	Diamondback moth	Cabbage	Negative	Consumptive	TMIE	Terrestrial	[67]
Parasitoid	Wasp	Small cabbage white butterfly	Thale cress	Positive	Consumptive	TMIE	Terrestrial	[68]
Parasitoid	Wasp	Russian wheat aphid	Grains	Positive	Consumptive	DMIE	Terrestrial	[69,70]
Parasitoid	Wasp	Yucca moth	Yucca	Positive	Consumptive	DMIE	Terrestrial	[71]
Parasitoid	Wasp	Marmalade hoverfly	Pea aphid	Positive	Consumptive	TMIE	Terrestrial	[72]
Parasitoid	Wasp	Gall wasp	Compass plant	Positive	Consumptive	DMIE	Terrestrial	[73]
Parasitoid	Wasp	Lychnis moth	White campion	Positive	Consumptive	TMIE	Terrestrial	[74]
Parasitoid	Wasps	Tobacco budworm	Artificial diet	Positive	Consumptive	TMIE	Terrestrial	[75]
Parasitoid	Wasps	Tephritid flies	Thistle	Negative	Consumptive	TMIE	Terrestrial	[76]
Parasitoid	Wasps	Beetle	Beach pea	Positive	Consumptive	DMIE	Terrestrial	[9]
Parasitoid	Wasp	Autumnal moth	Mountain birch	Positive	Consumptive	TMIE	Terrestrial	[77]
Parasitoid	Various wasps	Weevil	Spiny madwort	Positive	Consumptive	DMIE	Terrestrial	[78]
Parasitoid	Various	Soybean aphid	Soybean	Positive	Consumptive	DMIE TMIE	Terrestrial	[79]
Pathogen[c]	Mite	Red fox	Various	Positive	Consumptive	DMIE	Terrestrial	[38]

Table 1. (continued)

Infectious agent type	Infectious agent	Host	Resource	Effect on resource	Consumptive/ nonconsumptive	DMIE[a]/ TMIE[b]	Ecosystem	Refs
Pathogen	Anthrax	Impala	Acacia	Positive	Consumptive	DMIE	Terrestrial	[80]
Pathogen	Unidentified bacteria	Purple sea urchin	Kelp	Positive	Consumptive	DMIE	Marine	[2]
Pathogen	Chytrid fungus	Amphibians	Algae	Positive	Consumptive	DMIE	Freshwater	[20]
Pathogen	Microsporidian	Caddisfly	Periphyton	Positive	Consumptive	DMIE	Freshwater	[81]
Pathogen	Microsporidian	White clawed crayfish	Amphipod	Positive	Consumptive	TMIE	Freshwater	[82]
Pathogen	Amoeba	Green sea urchin	Kelp	Positive	Consumptive	DMIE	Marine	[83]
Pathogen	Unidentified	Black-spined sea urchin	Fleshy macroalgae	Positive	Consumptive	DMIE	Marine	[84]
Pathogen	Unidentified densovirus	Sunflower star	Green sea urchin	Positive	Consumptive	DMIE	Marine	[85]
Pathogen	Canine parvovirus	Wolf	Moose	Positive	Consumptive	DMIE	Terrestrial	[86]
Pathogen	Myxoma virus	Rabbit	Various	Positive	Consumptive	DMIE	Terrestrial	[87–89]
Pathogen	Plague	Prairie dog	Various	Positive	Consumptive	DMIE	Terrestrial	[90]
Pathogen	Rinderpest virus	Ungulates	Grasses	Positive	Consumptive	DMIE	Terrestrial	[11,91,92]
Parasitic castrator	Trematode	Snail	Ephemeral macroalgae	Positive	Consumptive	TMIE	Marine	[12]
Parasitic castrator	Trematodes	Snail	Benthic algae	Negative	Consumptive	TMIE	Marine	[93]
Parasitic castrator	Trematode	Snail	Algae	Negative	Consumptive	TMIE	Freshwater	[94]
Parasitic castrator	Rhizocephalan barnacle	Crab	Mussel	Positive	Consumptive	TMIE	Marine	[95]

[a]Density-mediated indirect effect.
[b]Trait-mediated indirect effect.
[c]*Sarcoptes scabiei*, the mite that causes sarcoptic mange, is a pathogen because it reproduces on its host.

Box 1. Positive or Negative Indirect Effects

The green world hypothesis states that in three-level food chains, predators reduce the abundance of herbivores, allowing plants to flourish [96]. Extending this logic to systems with different numbers of trophic levels leads to the prediction that top consumers benefit plants in food chains with odd numbers of trophic levels and harm plants in food chains with even numbers of trophic levels [97]. However, despite the fact that all trophic cascades listed in Table 1 have three levels, 13% (6/47) of studies detected indirect negative effects (as opposed to indirect positive effects) of infectious agents on the host's resource. What accounts for this perplexing pattern? As described in Box 3, infected hosts might consume more (instead of less) than their uninfected counterparts. Furthermore, because relationships between hosts and infectious agents are durable [5], the direction and magnitude of the effect on the host's resource could depend on the time scale over which it is observed. For instance, some parasitoids do not immediately arrest host development. Instead, the infected host continues to consume resources while the parasitoid develops, sometimes at an increased rate compared to uninfected conspecifics. Although parasitoids are eventually lethal to their hosts, delayed lethality allows infected herbivores to effect significant damage to plants before they succumb. For example, Xi *et al*. [76] found that a koinobiont wasp parasitoid stimulated growth and seed consumption by infected fly larvae compared to uninfected larvae. Thus, the parasitoid indirectly suppressed seed production through a consumptive trait-mediated indirect effect. This experiment took place within the larval duration of the study species, but positive effects of the parasitoid on the plant might be observed if parasitoid, host, and plant populations were tracked over several generations. Though relatively uncommon overall (Table 1), such negative indirect effects are more likely to be caused by infectious agents that eliminate host fitness (i.e., parasitoids and parasitic castrators), because host adaptive responses are impossible. Furthermore, such negative indirect effects are impossible for predators, because they result from consumptive trait-mediated indirect effects.

Like pathogens, macroparasites (e.g., adult trematodes) do not necessarily kill their host, but unlike pathogens, macroparasites do not multiply within their host [8]. Macroparasites generally aggregate among hosts, so that few host individuals harbor many parasites, while most individuals have low infection loads [13]. Because pathology is intensity-dependent [8], this aggregated distribution limits macroparasite-induced mortality [14], perhaps diminishing their potential to trigger trophic cascades. Indeed, our literature search revealed no examples of macroparasite-induced cascades.

Unlike other infectious agents, trophically transmitted parasites (e.g., trematode metacercariae) do not kill their host outright, but require the host to be eaten for successful transmission [8]. Therefore, the effects of trophically transmitted parasites are likely to spread upward through food webs (from host to predator), not downward. Bottom-up indirect effects, while entirely plausible, would not qualify as a trophic cascade according to Ripple *et al*.'s [6] definition. Because trophically transmitted parasites typically extract minimal energy from their hosts, direct pathological effects could be limited. However, it has been suggested (but not yet demonstrated) that trophically transmitted parasites could alter the strength and/or detectability of predator-induced trophic cascades by making prey more vulnerable to predation [15,16].

To summarize, most trophic cascades are triggered by predators (used throughout this paper to mean free-living secondary consumers), which are fully and immediately lethal to their victims (except **micropredators**; see later). Parasitoids, which are also fully (though not immediately) lethal, cause the majority (30) of reported infectious-agent induced cascades. Pathogens, which have intermediate lethality, cause 13 reported cascades, and parasitic castrators, which are typically not lethal, cause four reported cascades. No reported cascades are triggered by macroparasites, possibly because parasite aggregation limits their effects on hosts. Trophically transmitted parasites cannot trigger cascades, though they might alter predator-induced cascades. Finally, micropredators (e.g., mosquitoes) are not infectious agents, but have low lethality nonetheless, because they only consume part of their victim [8]. Although they could cause trophic cascades, we know of no examples of micropredator-induced cascades from the literature. Therefore, we arrive at the novel finding that the frequency of trophic cascades reported for different consumer types scales with consumer lethality.

Box 2. Use of Infectious Agents in Biological Control Programs

Infectious agents are often used in biological control [98]. For example, the small cabbage white butterfly, *Pieris rapae*, which is native to Europe, Asia, and North Africa, was accidentally introduced to North America in the 1860s. Its larva causes significant damage to cabbage and other mustard family crops. To control this pest, the parasitoid wasps *Cotesia glomerata* and *Cotesia rubecula* were introduced in 1884 and 1960–1992 respectively. These wasps, and *C. rubecula* in particular, effectively control populations of the small cabbage white butterfly, with current infection rates averaging up to 75% in some areas [99]. The resulting trophic cascade [54,55] has increased agricultural production in New England [100].

Another example of the use of an infectious agent in a biological control program involves myxoma virus, a virulent poxvirus of rabbits which is transmitted directly and via biting arthropods. This virus was introduced to Australia in the 1950s to control populations of the invasive European rabbit, *Oryctolagus cuniculus* [101]. Initially, the biological control program was successful, causing fatality rates up to 99.8% [102] and triggering a trophic cascade [89]. However, in time, host-pathogen coevolution reduced lethality, and rabbit populations recovered [101]. This example demonstrates that natural selection can thwart biological control efforts [103], especially if the infectious agent and host have rapid generation times and if the infectious agent is not necessarily lethal (e.g., a pathogen).

Biological control programs must take into account the coevolutionary history of the infectious agent and its host [98]. A lack of coevolutionary history (due to infectious agent, host, or both being non-native species) could strengthen trophic cascades, because naïve hosts might be highly susceptible. For example, the myxoma virus was first observed in Uruguay, and its dramatic effect on populations of European rabbits is attributed to lack of coevolutionary history [101]. However, lack of coevolutionary history could also prevent successful infection of hosts by infectious agents due to incompatibility [5]. For this reason, infectious agents that share coevolutionary history with the target host are usually selected for biological control programs. For example, the parasitoid wasps *C. glomerata* and *C. rubecula* are native to the same region as their host, *P. rapae*, and were selected as biological control agents because they are highly specialized, and therefore less likely to affect nontarget species.

Updated Definition

Ripple *et al*. [6] defined trophic cascades as 'indirect species interactions that originate with predators and spread downward through food webs.' Given recent efforts to unite predator–prey and parasite–host theory under a general consumer–resource framework (e.g., [17–19]), we generalize this definition to include trophic cascades that originate with infectious agents. Hence, trophic cascades are indirect species interactions that originate with consumers (predators or infectious agents) and spread downward through food webs.

Direct and Indirect Effects of Infectious Agents

Like predators, infectious agents can consume (i.e., infect) their victims with lethal consequences. This direct **consumptive effect** on the host can, in turn, trigger a **density-mediated indirect effect (DMIE)** on the host's resource (Figure 3). For example, chytridiomycosis, a disease caused by a fungal pathogen, decimated amphibian populations in the Neotropics following its outbreak in the 1990s. Where chytrid extirpated tadpoles from stream habitats, chlorophyll *a* and ash-free dry mass increased, and algal community composition shifted drastically [20]. However, infectious agents might be less likely than predators to cause density-mediated indirect effects because they are not always lethal to their hosts. Nevertheless, many infectious agents do kill their hosts, and 60% (28/47) of reported infectious agent-induced cascades involve such consumptive density-mediated indirect effects (Table 1).

Like predators, infectious agents can cause their victims to alter their behavior to reduce the likelihood of being consumed. This direct **nonconsumptive effect** on the host can, in turn, trigger a **trait-mediated indirect effect (TMIE)** on the host's resource if hosts modify behavior to reduce infection risk (Figure 3). For example, phorid fly parasitoids do not need to infect their predatory ant hosts to have an effect – their presence is sufficient to reduce the likelihood that ants will attack, carry away, and force herbivores off plants [21]. This example demonstrates that the ecology of fear applies to parasitism in addition to predation [17]. However, because infectious agents, in contrast to most predators, are not fully and

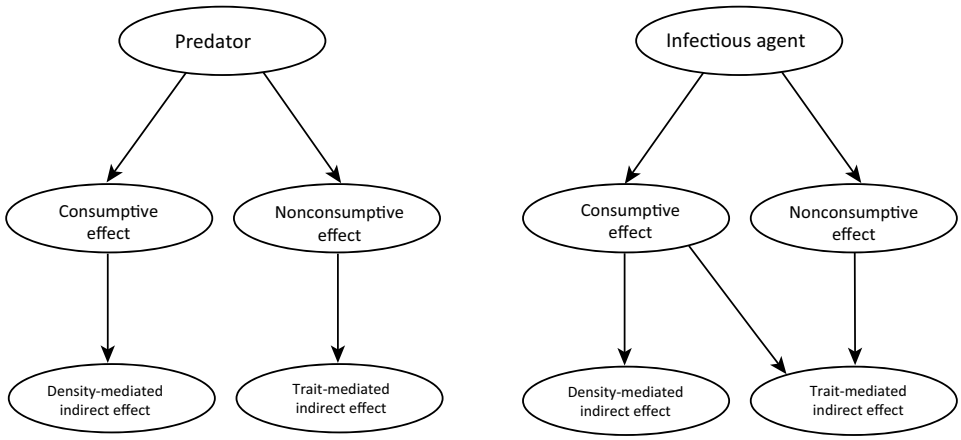

Figure 3. Direct and Indirect Effects of Predators and Infectious Agents. Because predators (except micropredators) are fully and immediately lethal to their prey, they can cause consumptive density-mediated indirect effects or nonconsumptive trait-mediated indirect effects. In addition to causing consumptive density-mediated indirect effects and nonconsumptive trait-mediated indirect effects, infectious agents can also cause consumptive trait-mediated indirect effects. This possibility results from the prolonged (durable) and intimate relationship between an infectious agent and its host.

immediately lethal, victims might perceive them as the lesser of two evils [22]. This could weaken their nonconsumptive effects relative to those of predators. Indeed, only 6% (3/47) of reported infectious agent-induced cascades involve such nonconsumptive trait-mediated indirect effects (Table 1; but see Box 3). Therefore, while the effects of intimidation might

Box 3. The Consumptive–Nonconsumptive Dichotomy Is Actually a Continuum

Paralleling the framework used for predators and their prey, we consider all cases where infectious agents infect hosts to be consumptive effects, and all cases where infectious agents induce fear to be nonconsumptive effects (Table 1). However, behavioral modification of infected hosts, which is accomplished through neural, endocrine, neuromodulatory, and immunomodulatory pathways [4], can occur either as a byproduct of altered energy requirements, or as an adaptation to increase parasite transmission [104]. The former is clearly a consumptive effect, but the latter could be considered a nonconsumptive effect if the parasite extracts minimal energy from its host. In this way, an infectious agent that has successfully infected its host could cause a nonconsumptive trait-mediated indirect effect. For example, the larval acanthocephalan parasite *Acanthocephalus tahlequahensis* alters the behavior of its isopod host, causing it to consume less detritus [105]. In this case, behavioral modification is likely an adaptation to increase parasite transmission to the final host, a fish, because infected isopods contain lower levels of serotonin and dopamine than uninfected conspecifics and do not avoid fish predators [106,107]. This could be considered a nonconsumptive trait-mediated indirect effect, because the mechanism by which the parasite alters host behavior does not deplete host energy reserves.

In contrast, if infection modifies host behavior by altering energy requirements, this should be considered a consumptive trait-mediated indirect effect. Infected hosts might consume more than do uninfected conspecifics (Box 1) if they compensate for energy extracted by the parasite for its own growth and reproduction, or because mounting an immune response is energetically costly. Alternatively, infected hosts might consume less than uninfected conspecifics due to the anorexic response [108]. Hence, the effects of altered energy requirements can benefit the infectious agent, the host, both, or neither.

The mechanisms that underlie parasite-induced behavioral modification have received little study [27], and we suggest that the extent to which infectious agents deplete host energy reserves should actually be represented as a continuum rather than a dichotomy. Hence, it would be impossible to determine the mechanisms behind all trait-mediated indirect effects listed in Table 1 and to categorize each as a consumptive or nonconsumptive effect on this basis. We therefore classify all examples in which infectious agents successfully infect hosts as consumptive effects, and caution that some could be considered nonconsumptive effects if parasites modify host behavior without altering host energetic requirements. We consider this to be a valid approach, because, by definition, all parasites extract at least a small amount of energy from their host.

overshadow those of direct consumption for predators [23], our results indicate that this does not hold for infectious agents. Furthermore, infectious agents seem less likely than predators to trigger trophic cascades by inducing fear.

In contrast to most predators (except micropredators), infectious agents can consume a victim without killing it, and even when infectious agents are lethal, significant time lags typically occur between infection and death. This raises the (previously unconsidered) potential for infectious agents to trigger trophic cascades through consumptive trait-mediated indirect effects, an impossibility for most predators (Figure 3). For example, as described above, *Cryptocotyle lingua*, a trematode parasite, infects snails (a consumptive effect), triggering a trophic cascade by reducing snail activity (a trait-mediated indirect effect) [12]. Indeed, 45% (21/47) of reported infectious agent-induced cascades involve such consumptive trait-mediated indirect effects (Table 1). Therefore, while both predators and infectious agents can trigger density-mediated and trait-mediated indirect effects, only infectious agents can do both through their consumptive effects. Furthermore, although infectious agents are probably less likely than predators to trigger trophic cascades through nonconsumptive effects on their hosts (but see Box 3), they could be more likely than predators to trigger trait-mediated indirect effects, because they can do so through both consumptive and nonconsumptive effects.

In addition to their direct and indirect top-down effects, infectious agents, like predators, can cause **knock-on effects**, which are indirect effects that spin off from the main interaction chain [6]. For example, after rinderpest was eradicated from the Serengeti, wildebeest populations irrupted and reduced grass biomass (Figure 2C). In turn, fire frequency decreased and tree cover increased (knock-on effects), thereby shifting the ecosystem from a net source to a net sink for carbon [11]. This suggests that, similar to predators, infectious agents might play a keystone role in ecosystems [24]. In fact, infectious agents are increasingly recognized to be capable of profoundly affecting ecosystem structure and function [7,25,26].

Reciprocal Effects between Predators and Infectious Agents

Many parasites manipulate host behavior, often through sophisticated mechanisms, to increase their own fitness [27]. This could be considered as a special class of consumptive trait-mediated indirect effect. Because behavioral manipulation often increases the likelihood that the infected host will be consumed, it could affect predator-induced trophic cascades by altering host vulnerability. For example, nematomorph parasites (horsehair worms) alter the behavior of their orthopteran (cricket and grasshopper) hosts, causing them to enter aquatic habitats where the parasite emerges as a free-living adult. Infected orthopterans comprise 60% of the annual energy intake of an endangered Japanese trout population [28]. When infected orthopterans are available, trout consume fewer benthic invertebrates, ultimately decreasing benthic algae biomass and increasing leaf breakdown rate [29]. Thus, by increasing the likelihood that their host will be consumed by a predator, parasites that manipulate host behavior have strong potential to alter predator-induced trophic cascades.

If parasites can affect predator-induced trophic cascades, is the opposite scenario also possible? The extirpation and extinction of top predators from ecosystems, a global-scale problem, has released prey species from predation pressure [30]. The resulting increase in prey populations can promote disease, particularly if disease transmission requires contact between infected and susceptible individuals. Below a certain threshold host density, epidemics cannot occur because contact rate falls below the death or cure rate of infected hosts [31]. However, if host populations increase due to release from predation pressure, then transmission rate could increase [32,33], thereby promoting epidemics (but see [34]). Here, an infectious agent can act as the missing predator, controlling prey populations and potentially causing indirect effects on lower trophic levels. For example, near the Channel Islands National Park in California, lobsters

have historically controlled sea urchin populations [35]. Where urchin predators are fished, however, urchin populations increase and overgraze algae, providing evidence of a predator-induced trophic cascade [2]. Starting in 1992 when an urchin-specific bacterial disease entered the region, dense urchin populations were more likely to experience epidemics [2]. However, the bacterial disease did not reduce urchin populations to the same extent that predators did, and so did not fully replace predators in the trophic cascade. Hence, as top predators continue to be lost from ecosystems [30], predator extirpation could trigger an increase in infectious agent-induced cascades, but infectious agents might not control prey populations as effectively as predators once did.

Detecting Infectious Agent-Induced Trophic Cascades

Parasitism is the most common consumer strategy on Earth [36], but most demonstrated trophic cascades are triggered by predators, not infectious agents. How can we explain this paradox? Sampling bias might explain why infectious agent-induced cascades are less commonly reported than predator-induced cascades. Due to their size and free-living lifestyle, predators are more conspicuous than parasites, which increases the detectability of predator-induced trophic cascades. Additionally, as the sea otter–bacterial pathogen example demonstrates, predators are far more charismatic than infectious agents (to most people), and interest and research funding might correlate with consumer charisma.

Beyond sampling bias, methodological considerations could reduce the detectability of infectious agent-induced cascades relative to predator-induced cascades. The extirpation and/or reintroduction of top predators to ecosystems has created 'natural experiments', which have allowed for the detection of many trophic cascades [37]. Analogous to predator reintroduction events, epidemics could present opportunities to detect infectious agent-induced trophic cascades. For example, during the late 1970's and 1980s, an epidemic of sarcoptic mange, caused by a burrowing mite (*Sarcoptes scabiei canis*), reduced red fox (*Vulpes vulpes*) population densities in Scandinavia [38]. In turn, prey populations increased markedly. However, using natural experiments to detect infectious agent-induced trophic cascades presents a major challenge. Because predator reintroduction is generally controlled by scientists or managers, community attributes can be sampled before and after the event. In contrast, disease epidemics are rarely planned, generally not predictable (but see [20]), and are usually only detected once they are underway. As a result, opportunities to sample community attributes before the event will be limited, which hinders efforts to detect trophic cascades.

A second method used to detect trophic cascades is to exclude predators from study plots and then compare community attributes between experimental and control plots (e.g., [39]). This approach can also be used to detect trophic cascades caused by infectious agents, but because infectious agents are smaller than their hosts (and often orders of magnitude so [8]), experimentally excluding them is challenging. Nevertheless, experimental exclusion of infectious agents has led to the detection of several trophic cascades. For example, Wood *et al*. [12] installed cages in the rocky intertidal zone, which they populated with infected or uninfected snails. While experimental exclusion of infectious agents is possible, it represents a significant challenge to the detection of infectious agent-induced trophic cascades.

Lastly, consumer–victim compatibility and specialization (which are related to coevolutionary history; Box 2) could explain why infectious-agent-induced cascades are less commonly reported than predator-induced cascades. A consumer's ability to trigger a trophic cascade might depend on the degree to which it specializes on its victims [40] (but see [41]). Specialist consumers target one victim species, and might have strong effects on its density. Nevertheless, trophic cascades triggered by specialists could be difficult to detect if functionally redundant nonvictim species step in to control the victim's resource. In contrast, generalist

consumers spread their impact over many victim species, which might share resources with one another. Consequently, the consumer's effect on a given victim species might be weak (or not detectable), but the effect on the victim's resource could be easier to detect because the consumer targets the entire guild, leaving fewer functionally redundant species available to fill the empty niche. Because parasites form intimate relationships with their hosts and must contend with host immune responses [5], they tend to be more specialized than predators. Differential specialization could strengthen the effects of infectious agents relative to those of predators, while simultaneously making infectious-agent-induced cascades more difficult to detect. Indeed, in a meta-analysis contrasting indirect effects of carnivores on plant fitness, predators caused ten times more indirect effects than parasitoids, but parasitoids caused stronger indirect effects than predators [42]. Together, these factors could explain the relative paucity of infectious-agent-induced trophic cascades reported in the literature.

Other factors could increase the detectability of infectious-agent-induced trophic cascades relative to predator-induced trophic cascades in some systems. First, because most infectious agents are smaller than most predators (at least relative to their victims) [8], the spatial and temporal scales over which their effects can be detected are often smaller. For example, detecting a trophic cascade originating with wolves requires sampling over large spatial and temporal scales [43], whereas detecting a trophic cascade originating with trematodes can be accomplished in small cages over the course of a few weeks [12]. Second, whereas manipulating the presence of infectious agents through traditional exclosure methods might prove difficult, many parasites can be excluded pharmacologically, for example, through the experimental application of antihelminthic medications (e.g., [44]). Lastly, whereas the experimental manipulation of top predators is typically fraught with ethical concerns, parasite elimination is usually considered culturally acceptable. These advantages could increase the detectability of infectious-agent-induced trophic cascades relative to predator-induced trophic cascades, but whether they can overcome the challenges presented above remains an open question.

Concluding Remarks and Future Directions

The potential for infectious agents to cause trophic cascades has been recognized for nearly half a century. Nevertheless, until now, trophic cascade theory has failed to consider similarities and differences between predator-induced and infectious-agent-induced cascades. Here we do so, arriving at novel insights that broaden trophic cascade theory. We find that, like predators, infectious agents can trigger trophic cascades. Unlike most predators, however, infectious agents are not fully and immediately lethal to their victims. This could make infectious agents less likely than predators to cause density-mediated indirect effects, and also less likely to cause nonconsumptive (fear-based) effects. However, because infectious agents form durable and intimate relationships with their hosts, they seem more likely than predators to cause trait-mediated indirect effects. Hence, our paper demonstrates that the disjunct fields of predator–prey ecology and parasitology have much to offer one another.

Although infectious agents are usually considered only in light of their direct negative effects on hosts, like predators, they can also shape ecosystem structure and function through their indirect effects [7,25,26]. The potential for infectious agents to benefit their host's resource through trophic cascades is recognized and commonly applied in agricultural settings (Box 2, but see Box 1), but is typically ignored by natural resource managers. Analogous to historical predator extermination efforts, current management efforts generally attempt to eradicate infectious agents to control wildlife disease. Many predator elimination efforts were abandoned following a shift in public perception of predators [45]. We suggest that going forward, managers should consider adopting the same attitude towards naturally-occurring infectious agents; like predators, they are integral members of ecological communities whose influence can extend far beyond their direct negative effects on victims. Infectious agents might even be

Outstanding Questions

Infectious agent-induced trophic cascades are less commonly reported than predator-induced cascades. Is this due to sampling bias or inherent differences between predators and infectious agents? Are infectious-agent-induced trophic cascades common enough to merit consideration in models of energy flow through food webs?

How does the strength of infectious-agent-induced trophic cascades compare to that of predator-induced trophic cascades? Does coevolutionary history shape the strength of trophic cascades?

Can macroparasites and micropredators cause trophic cascades? Can trophically transmitted parasites alter the strength and/or detectability of predator-induced trophic cascades?

Should trophic cascade theory consider consumptive/nonconsumptive effects as a continuum rather than a dichotomy?

Can natural selection thwart biological control efforts? How might coevolutionary history between infectious agent and host affect this?

Will the continued loss of top predators from ecosystems prompt an increase in detection of infectious-agent-induced trophic cascades? Will infectious agents control host populations as effectively as predators formerly controlled prey populations?

Should management efforts to control naturally-occurring wildlife diseases be abandoned? Should conservation efforts focus on preserving rather than eradicating infectious agents?

worthy of conservation attention [46], as their specialized nature makes them particularly vulnerable [47]. We hope that our paper will further efforts to unite predator–prey and parasite–host theory under a general consumer–resource framework [17–19], and inspire the search for more infectious agent-induced trophic cascades in varied ecosystems.

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