Review

When parasites become prey: ecological and epidemiological significance of eating parasites

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Recent efforts to include parasites in food webs have drawn attention to a previously ignored facet of foraging ecology: parasites commonly function as prey within ecosystems. Because of the high productivity of parasites, their unique nutritional composition and their pathogenicity in hosts, their consumption affects both food-web topology and disease risk in humans and wildlife. Here, we evaluate the ecological, evolutionary and epidemiological significance of feeding on parasites, including concomitant predation, grooming, predation on free-living stages and intraguild predation. Combining empirical data and theoretical models, we show that consumption of parasites is neither rare nor accidental, and that it can sharply affect parasite transmission and food web properties. Broader consideration of predation on parasites will enhance our understanding of disease control, food web structure and energy transfer, and the evolution of complex life cycles.

Introduction

The idea of eating prey triggers revulsion in most people. But if you have ever appreciated a glass of dessert wine, you might have unwittingly consumed (and likely enjoyed) 'noble rot,' a parasitic fungus (*Botrytis cinerea*) used to desiccate grapes and concentrate sugar. Nor is the consumption of parasites always inadvertent. Worldwide, parasites are prized as delicacies, including botfly larvae, lice, mites, tapeworms, flukes and parasitic crustaceans [1]. In Wisconsin, giant liver flukes (*Fascioloides magna*), which achieve a length of 8 cm inside deer, are sautéed in butter and served as 'liver butterflies.'

Beyond human cuisine, growing evidence suggests that consumption of parasites is neither rare nor strictly accidental. Lafferty *et al.* [2], for example, estimated that 44% of links in an estuarine food web involved predation on parasites. For some multi-host parasites, predation is an important transmission pathway, allowing infections to spread from prey to predator (trophic transmission). However, ingestion by the 'wrong' predator also occurs, leading to parasite death and digestion. Vertebrate and invertebrate predators actively consume parasite free-living stages, including eggs, miracidia, cercariae, zoospores, nematode larvae and trophozoites [5]. Considering the tremendous reproductive output of parasites [3], their consumption likely represents a far more important trophic linkage than previously recognized; parasites have the potential to transfer substantial biomass and energy up the food chain.

Despite recent calls for enhanced integration of research on parasite transmission, foraging ecology and community structure [6–8], the ecological significance of parasite ingestion has rarely been considered, particularly when consumption does not lead to transmission. Here, we

Glossary

Biological control: the intentional introduction of predators, parasites, or pathogens to reduce the population of a 'pest' species
Cleaning symbioses: the removal of ectoparasites, diseased or injured tissue, or other particles by a cleaning organism from another cooperating organism
Complex life cycle: transmission among different host species that are used for stages of parasite development or reproduction
Concomitant predation: form of predation in which predators consume parasites alongside or within their primary diet items
Connectance: calculated as the ratio of the number of observed links over the number of potential links within a food web
Dilution effect: a reduction in parasite transmission resulting from increases in community diversity
Free-living stages: the infective stages of parasites that exist outside the body of a host
Grooming: behavior of an organism towards itself or other species to clean body surfaces of debris and remove ectoparasites
Hyperparasitism: the infection of parasites by other parasites
Intraguild predation: the consumption of species potential competitors based on similar resource use
Parasite: an organism that causes harm to another organism with which it lives in intimate contact
Parasitoid: organisms whose larval stages live as parasites and typically kill their hosts after completing development
Predator: an organism that consumes other organisms as food
Transmission: infective stages that are transmitted to a new host through predation
Trophic transmission: infective stages of parasites are ingested by the host

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evaluate the phenomenon of predation on parasites in natural food webs, including the forms in which it occurs and its ecological, evolutionary and epidemiological significance. Predation on parasites has implications for understanding not only food-web dynamics, but also the transmission of human and wildlife pathogens and the control of agricultural pests. We consider the importance of parasite consumption for: (i) enhancing our understanding of food web topologies, resource dynamics and energy transfer; (ii) identifying the biotic factors that control parasite transmission and disease; and (iii) assessing the epidemiological importance of anthropogenically-altered communities of predators and parasites, particularly those undergoing biological invasions and extirpations.

Forms of predation on parasites

Predation on parasites can assume a variety of forms within food webs. In the sections that follow, we define each form of predation on parasites in detail and examine their occurrence in natural communities.

Concomitant predation

The most common way for parasites to become prey is through concomitant predation, which occurs when an infected host is eaten by a predator. Considering that most prey individuals are infected by many parasites, this type of parasite consumption probably contributes significantly to parasite mortality (see online supplemental material). When parasites represent a substantial fraction of host biomass, such as trematodes in snails, ascarid worms in mammal intestines, parasitoids in aphids, or larval tapeworms in stickleback fish, the nutritional contribution of parasite tissue for a predator could be substantial (Figure 1a). Some complex life cycle parasites induce changes in host characteristics, such as physical appearance, stamina or behavior, that increase the likelihood of host consumption by downstream hosts [9–12]. However, parasite manipulation might also result in ingestion by 'unsuitable' predators, in which case parasites are digested along with prey [13–14]. Even ectoparasites feeding on hosts can consume a considerable number of parasites alongside host tissue [15–16].

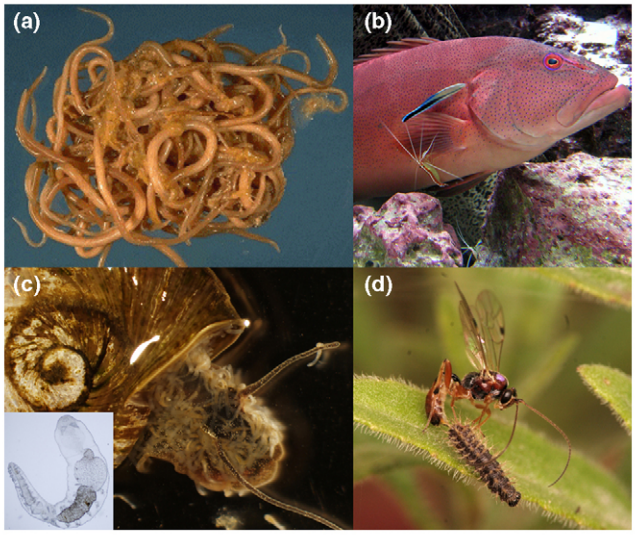

Figure 1. Forms of predation on parasites. **(a)** Concomitant predation: aggregation of nematode worms (*Ascaris* sp.) from the intestines of vertebrates can occupy substantial biomass and are frequently consumed alongside their hosts; **(b)** Grooming: cleaner wrasse (*Labroides dimidiatus*) and cleaner shrimp (*Lysamata* sp.) removing ectoparasites from a coral trout (copyright A. Grutter; reproduced with permission); **(c)** Predation on free-living parasite stages: oligochaete worms (*Chaetogaster* sp., see lower left inset) commensal with freshwater snails (*Helisoma* sp.) feed on trematode miracidia and cercariae (copyright P. Johnson and S. Orlofske); **(d)** Hyperparasitism: the hyperparasitoid *Mesochorus stigmanticus* probing in an *Melitaea cinxia* caterpillar for parasitoid larvae to infect (copyright S. van Nouhuys; reproduced with permission).

Grooming behavior

The most easily observable form of predation on parasites involves grooming. Self- and intraspecific grooming are widespread among mammals such as ungulates and primates [17–19] and among birds [20]. Although intraspecific grooming has often been co-opted for social functions such as alliance formation, particularly in primates [19], it remains an effective method of ectoparasite control [21]. Interspecific grooming can also be an important form of predation on parasites, involving the consumption of ectoparasites by small 'cleaning' species as part of a co-evolved mutualistic association with a larger host species (Figure 1b). Cleaning symbioses are particularly well documented in marine ecosystems, where over 130 species of fish and crustaceans act as cleaners ([22]; Figure 2a). While the majority of these are facultative, the best-studied cleaners are fish belonging to the genus *Labroides*, which specialize on ectoparasites ([22–23]; Figure 1b). Field estimates suggest that *L. dimidiatus* can eat two-thirds of all gnathiid isopods infecting reef fishes [23–25], significantly reducing the impact of these parasites on their hosts. Cleaning also occurs in terrestrial systems [see online supplemental material]. For example, oxpeckers consume large numbers of ticks from African ungulates, reducing the impact of ectoparasites on herds while also exerting selective pressure on tick populations [26].

Consumption of parasite free-living stages

A less conspicuous form of predation on parasites involves active or passive foraging on free-living stages of parasites. Free-living infectious stages occur in the life cycles of helminths (e.g. eggs, miracidia and cercariae in trematodes, or eggs and juveniles in nematodes), fungi (e.g. zoospores), protists (e.g. some trophozoites) and certain ectoparasites. These stages are vulnerable to predation by numerous organisms ([5]; Figure 1c). For example, earthworms and dung beetles frequently ingest parasite eggs and larvae while feeding on the feces of other organisms, sometimes leading to reduced parasite transmission [27–29]. Similarly, domestic pigs consume feces and destroy the larval stages of nematodes infective to cattle, while domestic dogs perform the same role in human societies affected by ascarid nematodes [30]. Fungi that consume nematode larvae can be used in biological control against nematode larvae ([31]; Figure 2b).

In aquatic systems, predators ranging from oligochaetes and insect larvae to carnivorous plants all prey on trematode miracidia and cercariae, leading to reductions in transmission ([3,32–35]; Figure 2c). Likewise, chytrid zoospores fall prey to zooplankton and can even enhance

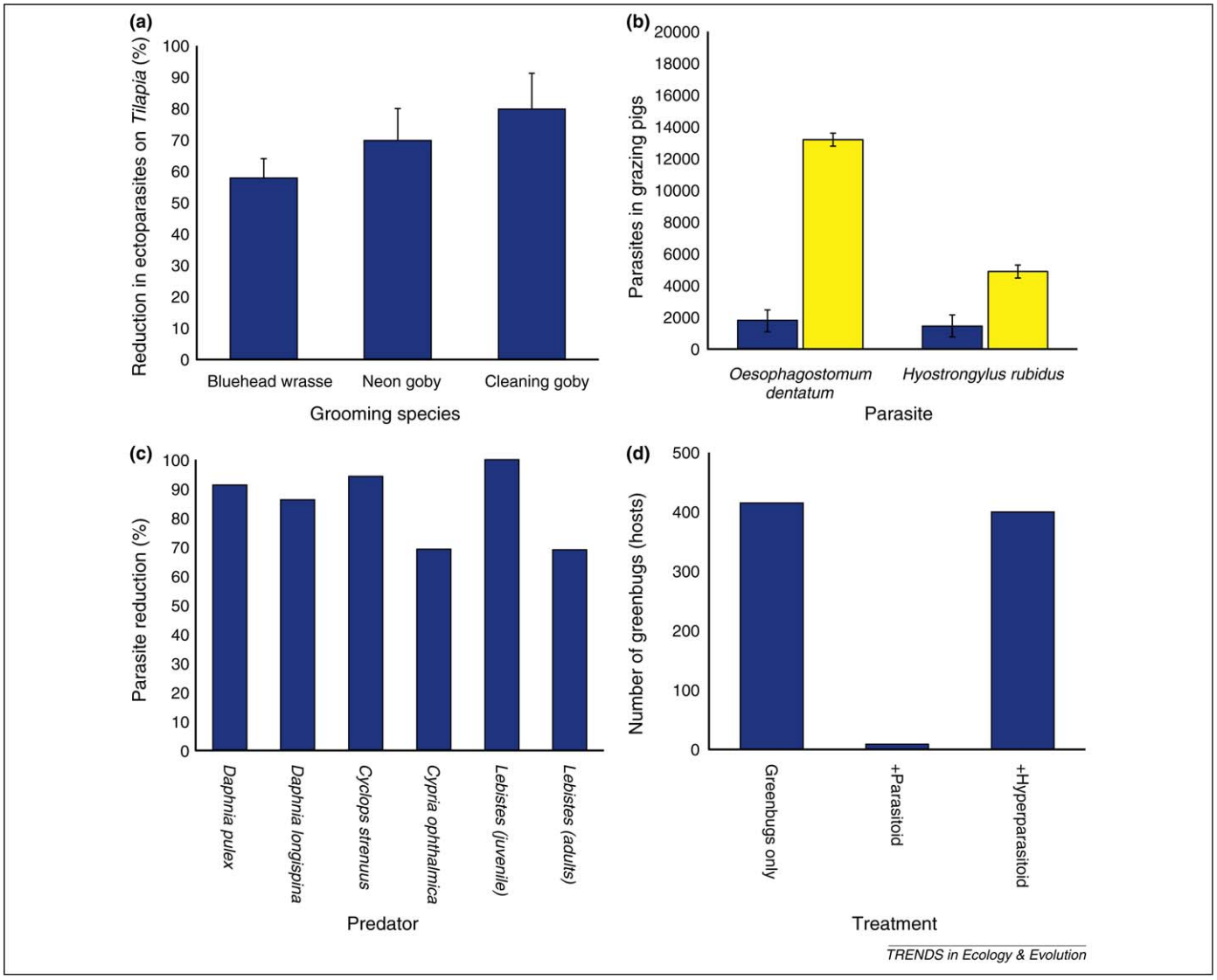

Figure 2. Effects of predation on parasite infection and transmission. **(a)** Percentage reduction in monogenean ectoparasites on *Tilapia* fish owing to predation by cleaner fishes [88]; **(b)** Pigs (blue bars) grazing on pastures previously dosed with a nematophagous fungus (*Duddingtonia flagrans*) exhibited lower burdens of nematodes (*Oesophagostomum dentatum* and *Hyostrongylus rubidus*) than pigs (yellow bars) on untreated pastures [89] owing to consumption of the free-living nematode stages by fungi; **(c)** Consumption of free-living cercariae by aquatic predators, including zooplankton (*Daphnia* and *Cyclops* spp.), ostracods (*Cypria ophthalmica*), and guppies (*Lebistes* sp.). Depicted is the percentage reduction in mouse infections by radioactively labeled *Schistosoma mansoni* cercariae relative to no-predator treatments [90]; **(d)** Effects of parasitoids (*Lysiphlebus testaceipes*) and hyperparasitoids (*Charips* sp.) on the total number of greenbugs (*Schizaphis graminum*) in greenhouses [91].

predator growth [36]. In terrestrial systems, engorged ticks and ectoparasites are easy and nutritious prey for rodents, birds, lizards, ants and spiders ([37–38]; Box 1). In marine environments, anemones and filter-feeding molluscs prey upon trematode cercariae and metacercariae [39–41]. These examples (along with those in the online supplemental material) probably reflect only a small fraction of the naturally-occurring spectrum of predation on parasites.

Parasites preying on other parasites

Parasites also act as predators or parasites of other parasites, perhaps more commonly than we might imagine. For instance, microsporidians frequently infect trematodes, increasing mortality and decreasing transmission [42]. Some parasites specialize in attacking other parasites, particularly among the parasitoid Hymenoptera, some of which require a host previously infected by another parasitoid [43]. Hyperparasitoids, or parasitoids that feed on other parasitoids, occur in 17 families of hymenopterans along with some Coleoptera and Diptera ([44]; Figure 1d). A community of 25 meadow aphid species, for example, was attacked by 18 species of primary parasitoids, which were in turn attacked by 25 species of hyperparasitoids [45]. The presence of hyperparasitoids can even induce trophic cascades, releasing hosts from mortality induced by parasitoids (Figure 2d).

Intra-guild predation is another common form of parasite antagonism, typically when host resources are limited, as for many parasitoids and parasitic castrators [46]. Parasitoids will actively consume other parasitoids within a given host; a necessary strategy when only one parasite can successfully emerge [47]. Inside snail hosts, some trematodes, which are parasitic castrators, consume the larval stages of other parasites, such that multi-parasite infections are uncommon and often transient [46]. Similarly, immature and adult ticks frequently feed on other ticks, including conspecifics [48].

Box 1. Desert Islands in the Gulf of California

Small desert islands in the Midriff area of the Gulf of California support surprisingly high productivities of small predators, including spiders, scorpions and lizards. Consumers on these islands rely mainly on allocthonous input, functionally connecting marine and terrestrial food webs [92–93]. Seabirds, which nest on these islands to avoid predators, contribute significant resources via fish scraps, carcasses and their parasites [92]. This input supports high densities of spiders, scorpions and lizards compared to bird-free islands [93–94]. Indeed, spiders, scorpions and lizards were shown to eat ectoparasites alongside other seabird nest-dwelling arthropods [93–94]. For example, 98% of insects collected from seabird islands were ectoparasitic bobitos (*Paraleucopsis mexicana*), which also constituted 98% of prey items in spider webs on islands with seabird colonies [92,94]. Densities of spiders, ants, scorpions and lizards that feed on seabird parasites and scavengers were 1–2 orders of magnitude higher on seabird islands compared to islands without colonies [92]. Densities of spiders were 4–5 times greater on islands with nesting colonies than those without, and 12 times higher within the colonies compared to away [94]. Lizards were 4 times more numerous on islands with seabirds than those without and 21 times more abundant within the colonies than away from them [93]. On islands with seabird colonies, ectoparasites help convert marine-derived seabird tissue into terrestrial biomass. The allochthonous input derived from seabirds also increases the complexity of the island food web by channeling energy through different pathways (including parasites) and contributing prey biomass for higher-level predators (Figure I).

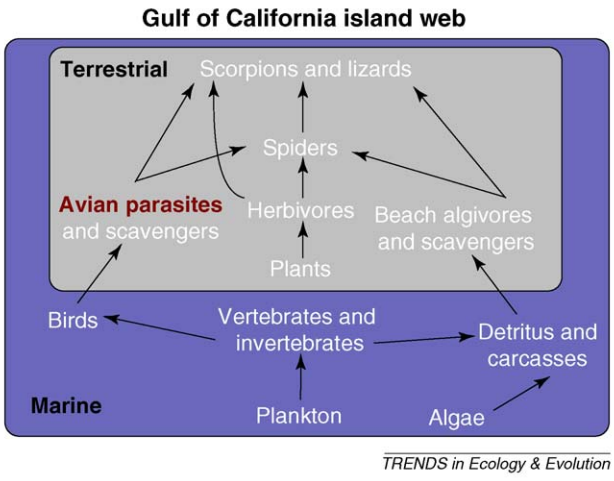

Figure I. Representative food web on small islands in the Gulf of California with seabird colonies. Seabird ectoparasites are important components of the diets of intermediate level predators and top predators on these islands. Furthermore, they contribute to prey biomass for the top predators. Redrawn from [94].

Ecological significance of parasite predation

The ecological and evolutionary significance of predation on parasites events remains an open question. What do parasites contribute to predator diet quantity or quality, and how does incorporation of predation on parasites affect our understanding of food webs and parasite evolution? Can predation control parasite transmission and reduce disease risk in natural environments? These are important questions for future research programs, which we explore below.

Food quantity and quality

Recent food-web studies have clearly established that predation on parasites is common, comprising between 36 and 44% of observed trophic links [2,49]. Nevertheless, the significance of parasites in the diet and growth of free-living organisms remains poorly understood. For select groups of specialists, parasites can comprise the majority of ingested material. Grutter [24] estimated that a cleaner wrasse consumes >1200 ectoparasitic gnathiid isopods per day, representing 99.7% of its diet. Similarly, commensalistic oligochaetes (e.g. *Chaetogaster* spp.) can derive substantial dietary benefits by consuming the trematode cercariae and miracidia attempting to infect their freshwater snail hosts ([50]; Figure 1c).

Whereas concomitant predation on parasites in prey will often have only small energetic benefits for a predator, foraging on free-living stages of parasites could provide significant energetic and nutritional resources. At the ecosystem-scale, parasites generate tremendous numbers of infective stages (e.g. [4]), most of which are unsuccessful in finding hosts and should, therefore, contribute to the diet of opportunistic predators and decomposers. Most estuarine fishes readily consume trematode cercariae in the laboratory, and the annual production of cercariae (estimated at 5 g m^{-2}) at Carpinteria Salt Marsh could support ~2–3% of the total energetic demands of the fish community [34]. However, the general energetic contributions of parasites to predator communities remain unknown for most systems.

Parasites also alter the susceptibility of infected prey to predation, significantly affecting the foraging success and growth of predators. Examples of parasites that alter intermediate host behavior, morphology or conspicuousness, and thereby enhance predation by downstream hosts, are common [9–12]. Although these infections can incur added costs to predators in the form of new infections, the increase in foraging success will often outweigh any increase in pathology [51–52]. Killifish infected with the trematode *Euhaplorchis californiensis* were up to 30 times more vulnerable to bird predation [53], strongly suggesting that the resulting increase in bird foraging success dwarfed any added costs to the birds associated with infection acquired from consuming fish. Furthermore, not all trophically transmitted parasites successfully infect their final host, with perhaps most being digested by non-host predators. These predators probably benefit from consuming altered and infected prey, leading to an increase in prey acquisition with little or no physiological cost [13]. For example, nematomorph parasites drive crickets into streams where the helpless (yet nutritious) insects are readily eaten by non-host predators such as trout [54], which are not infected by the worms.

The relative value of parasites as prey depends on their energy content. Free-living infective stages are typically rich in glycogen and lipids while devoid of difficult to digest shells or exoskeletons [55–56]. The tissue of tapeworms, for instance, includes 20 to 50% glycogen reserves and >30% lipid content, making it extremely nutritious. Parasites can also affect the energetic content of their hosts. Brine shrimp infected by tapeworms exhibited nearly 2x the concentration of triglycerides relative to uninfected hosts [57]. In other cases, however, parasites can reduce the quality of hosts as a prey resource [47]. Because they accumulate heavy metals, some intestinal helminths are

a source of contaminants to predators [58]. Similarly, chytridiomycete infections reduce the nutrient and fatty acid content of parasitized *Daphnia* [59]. Considering the importance of *Daphnia* for juvenile fish growth and the prevalence of infected *Daphnia* in fish stomachs (up to 100%; [11]), these parasite-induced changes could inhibit fish growth and nutrition.

Predation on parasites and food-web topology

Parasites are often omitted from food-web analyses because they are cryptic, difficult to measure, and assumed to occur in low biomass [60]. However, adding parasites to food webs can alter topological properties of the network such as linkage density, food chain length, connectance and nestedness [49,60–61]. In the Carpinteria Salt Marsh web [62], for instance, there are 615 parasite-host links and 910 predator-parasite links relative to 505 'conventional' predator–prey links. Thus, food webs that exclude parasites underestimate a large portion of biodiversity, omit many links and miscalculate the true topological structure of a network [60].

Predator–parasite links close a tight triangular loop between a predator, prey and parasite. This loop is a consequence of intimacy and results in a form of nesting (the parasite feeds on a subset of the predator's diet) that can increase estimates of nestedness in a network [2]. This looping would be even tighter if the predation involves self-grooming. In this case, the predator would ingest biomass ultimately derived from itself. Whether this aspect of nestedness has the same implications for food-web dynamics as other types of nesting is, as yet, unexplored and we advocate calculating nestedness with and without predator-parasite links [2].

Proper incorporation of parasites into food webs requires additional sub-webs, including parasite–host, parasite–parasite and predator–parasite [2]. Emerging evidence indicates that parasite-related links differ from those involving strictly free-living species alone. For example, in a subarctic lake food web the predator–parasite sub-web had twice the connectance of the overall web because many predators consumed each parasite [49]. Integration of parasites therefore has the potential to alter our understanding of food web structure and theory, including food web properties such as complexity, food chain length, predator-prey body size relationships and energy transfer [60]. The resulting changes have applied importance for predicting food web dynamics and their stability in response to perturbation [61].

Regulation of parasite transmission

In each of its forms, predation on parasites can significantly reduce transmission (Figure 2). Predation on free-living stages, for example, can substantially reduce the infection and resultant pathology in down-stream hosts ([Figure 2b; [3,5]). Active predators such as shrimps and crabs and passive filter feeders like bivalves can inhibit infections in target hosts by 40–91% in experiments [5]. The myxosporean parasite (*Myxobolus cerebralis*) depends on *Tubifex* worms as intermediate hosts, but resistant lineages of the worm will consume and digest parasite myxospores, possibly lowering the incidence of salmonid whirling disease [63]. On pastures, nematophagous fungi

Box 2. Transmission consequences of predation on parasites

The dynamic consequences of predation on parasites can be examined using the classic framework for the dynamics of parasitic helminthes [96]. Initially we consider the three-equation version of the model that explicitly considers the free-living infective stages, W, definitive hosts, H, and adult parasites, P.

$$\frac{dH}{dt} = (b-d)H - \alpha P \quad (1)$$

$$\frac{dW}{dt} = \lambda P - \delta W - \beta WH \quad (2)$$

$$\frac{dP}{dt} = \beta WH - (d+\mu+\alpha)P - \alpha\frac{P}{H}\frac{k+1}{k} \quad (3)$$

where, b and d are the birth and death rates of the host, α is the parasite induced host death rate, λ is the birth rate of free-living parasite infective stages, $1/\delta$ is the life expectancy of free-living infective stages, β is the rate at which these infect the host, $1/\mu$ is the life expectancy of adult parasites (which might be reduced by host grooming), and k is the aggregation parameter of the negative binomial distribution. Predation can directly affect five of these parameters: d, α, δ, β and μ; each of these terms could also include dynamic terms for the abundance of different predators in a food web. Here, we treat the variables as constants. The framework allows us to identify four classes of predation on parasites:

1) ***Concomitant predation*** occurs when parasites are ingested alongside a prey host. This will always increase the host death rate, d, and it will provide a nutritional supplement to the predator. This type of predation always reduces the parasite's fitness.
2) ***Trophic transmission*** occurs when the predator ingesting the host can support the parasite. This form of predation impacts host death rate, d, which will be enhanced if the parasite increases the host's susceptibility to predation, α [46]. The predatory host receives a nutritional benefit from digesting the infective host, but the subsequent establishment and development of the parasite in this host can lead to nutritional and fitness costs. Parasite fitness will be increased if it subsequently reproduces in the predator or is transmitted from it to the next host in the life cycle.
3) ***Oral transmission*** occurs when hosts ingest free-living infective stages and become infected at a rate, β. This increases the fitness of the parasite (if it establishes and reproduces). The host might receive a nutritional benefit but also pays a reduction in fitness if the parasites establish.
4) ***Predation on free-living stages of parasites*** will increase their mortality rate, δ, and reduce infections in downstream hosts. Sometimes susceptible hosts can also act as predators. The relative importance of this form of predation depends on the life expectancy of the free-living stages and on host density, which can be readily seen by assuming the dynamics of equation 2 are fast relative to those of the other two equations. The density of free-living infective stages can then be set to equilibrium W^* such that $dW^*/dt = 0$

$$W^* = \frac{\lambda PH}{\beta(H_0+H)} \ldots\ldots\ldots\ldots\ldots\ldots \text{with..} H_0 = \delta/\beta \quad (4)$$

Anderson and May [96] substitute this into 3 to give the more frequently used two-species host-parasite equation; here it is worth noting that $H/(H_0+H)$ is the proportion of free-living larvae that successfully invade a host. Predation on free-living stages always increases δ, and reduces the proportion of larvae infecting the next host.

5) ***Grooming*** is an extreme example of predation that actively reduces parasite abundance once the parasite has attached to the host; it occurs when either the host or a mutualist removes parasite stages, thereby increasing the magnitude of adult parasite mortality, μ.

reduce infective nematode abundances and their transmission to mammals ([31,64–65]; Figure 2b). Predation on vectors, including mosquitoes, ticks, leeches and sand flies that are infected with viruses, bacteria and protists could also decrease pathogen transmission through concomitant predation. With respect to ectoparasites, animals prevented from grooming incur much higher infections than those allowed to groom ([66–67]; Figure 2a). Impala prevented from grooming had 20 times more ticks than impala allowed to groom naturally [68]. Intraguild predation among trematodes caused a 16% reduction in trematode individuals found in the marine snail *Cerithidea californica* [69], and such effects are common in other systems [46].

The widespread consumption of parasites by predators clearly demonstrates that parasite transmission does not occur in an ecological vacuum, but is instead part of a much larger network of interactions. Models of parasite population dynamics (which usually integrate the survival of free-living stages as static death rates) would gain from incorporating the role of predator–parasite interactions (Box 2). The examples presented here (along with those in the online supplemental material) illustrate that predation on parasites is an important mechanism of the 'dilution effect,' a hypothesis linking community diversity and parasite transmission, ultimately affecting disease prevalence and severity. Whereas previous work on the dilution effect has focused on the role of alternate hosts, especially in vector-borne systems [7,35,70], we argue for a broader incorporation of interactions across trophic levels, including both inter- and intraguild predation (Box 3).

Box 3. Future research questions surrounding parasites as prey

Evolutionary considerations

- Is predation on parasites an important (and continuing) force in the evolution of complex life cycles?
- How often do parasites induce changes in the anti-predator behaviors or morphology of their hosts to reduce concomitant predation?
- Can predators alter the strength of selection imposed by virulent pathogens, for example by decreasing the density of infected hosts, which may be proportional to selection intensity?

Parasite transmission and disease

- When are predators likely to have a significant effect on parasite transmission and disease levels?
- Is predation a widespread mechanism of the dilution effect? For what types of predator-parasite combinations and at what respective densities?
- How does the loss or addition of predators to communities (e.g. invasions and extinctions) affect parasite transmission? When can the addition or maintenance of predators be used for parasite or pest control?
- Can predator-induced changes in infection lead to trophic cascades in either the abundance of parasites or of their hosts?
- How do we extend the concept of the dilution effect to a variety of trophic levels, including intra- and interguild predators?

Food web structure and dynamics

- What fraction of free-living parasite stages becomes prey for opportunistic predators and/or decomposers? How does this affect predator growth and ecosystem energy flows?
- Do parasites alter the nutritional value of infected prey?
- How does the presence of parasite-modified prey alter the foraging success of predators? How do such changes influence community diversity and abundance within an ecosystem?
- What are the net energetic consequences of consuming parasitized prey for predators?
- How does incorporation of parasites as prey into food webs alter their dynamics, structure, and theoretical development? Do predator–parasite links increase the stability and robustness of food webs?

Evolutionary considerations

High levels of predation likely exert strong selection pressures on parasites. But the nature of this selection, and the evolutionary direction it favors, depends on whether the parasite can survive predation and exploit the predator to improve its own transmission. If direct predation on a parasite is inevitably fatal, selection will favor strategies that primarily benefit the parasites, leading to different life history trade offs. Consumption of infected animals by non-host predators might select for parasite-induced anti-predator behavior [71]. For example, the nematode *Phasmarhabditis hermaphrodita* kills its slug host underground where there are fewer predators and scavengers, thereby allowing adequate time for the nematode to complete its development inside the host cadaver [72]. Indeed, evidence suggests that amphipods infected with acanthocephalans can avoid non-host predators more effectively than uninfected conspecifics [71,73]. Ectoparasites of coral reef fishes subjected to predation from cleaner wrasses show attachment site preferences and body coloration suggestive of past selection for crypsis [25,74]. In contrast, if infective stages can resist digestion, and if survival within the predator allows the subsequent completion of the parasite's life cycle, selection could favor predation as a transmission route. This is one possible path for the evolution of trophic transmission, by downward incorporation of new hosts in the life cycle. Mathematical models indicate that if parasite propagules are frequently ingested by a predator, and if that predator is itself a regular prey of the parasite's definitive host, then selection might favor addition of the predator as an intermediate host [75–76]. In these situations, infective stages would evolve adaptations to enhance their risk of predation. This is beautifully illustrated by the cercariae of trematodes that attach to each other by their tails to form large clusters, thereby mimicking food items [34,77]. In a third of the predator–parasite links in the Carpinteria Salt Marsh Web, parasites within prey are able to avoid digestion and exploit the predator as a host [62]. The 'ghost of predation past' can therefore still be seen in the life cycles of trophically transmitted parasites, in which parasites that might have once been victims of predation now exploit their former predators as hosts (Box 3).

Invasions and extinctions

The role of predation on parasites is of particular importance in light of ongoing changes in natural communities (e.g. extirpations and invasions). Invasions and extinctions of predators could indirectly alter parasite transmission through at least three pathways (Figure 3). First, invaders can prey directly on parasites or their free-living stages [78]. For example, non-native Pacific oysters and American slipper limpets consume cercariae of native trematode

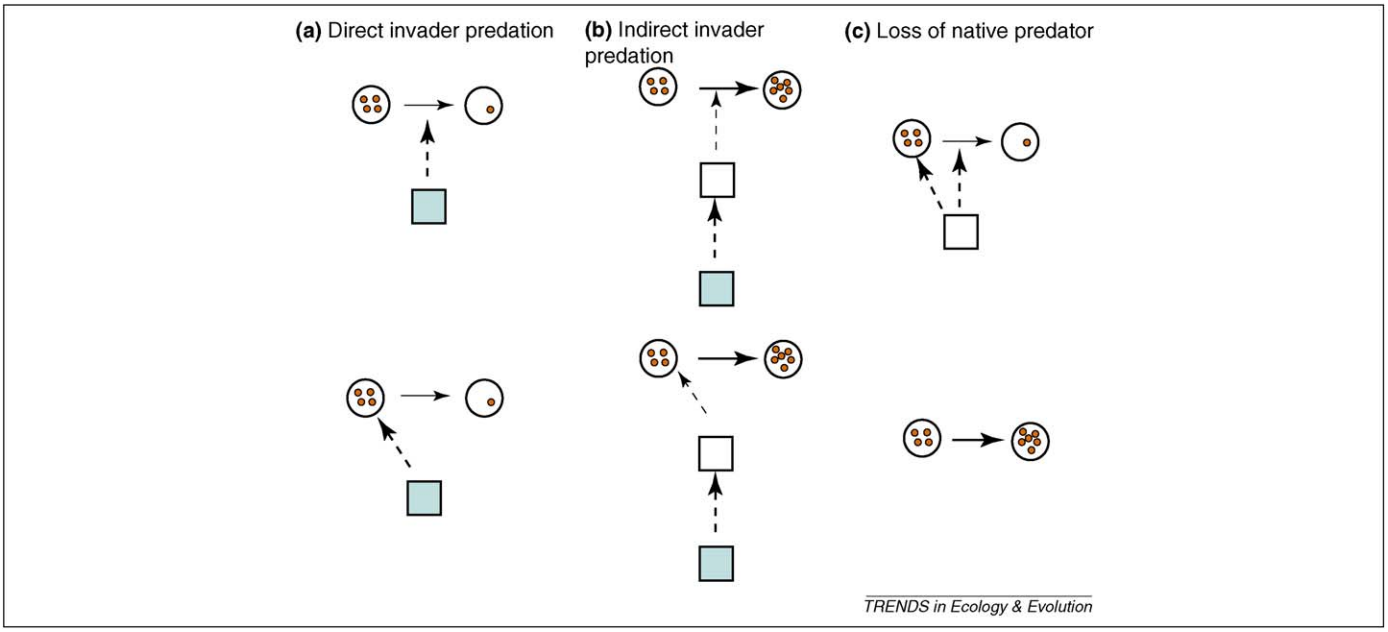

Figure 3. Effects of species invasions and biodiversity loss on parasite transmission. Varying infection intensities of parasites (small orange circles) in hosts (large circles) are symbolised with the number of circles. Transmission is symbolised by arrows and predation is symbolised by dashed arrows, with the size of the arrows indicating interaction strength. **(a)** Invaders (blue squares) can directly prey upon parasite free-living stages, thus reducing infections, or upon parasites in their hosts (concomitant predation). **(b)** Invaders (blue squares) can consume native predators (white squares) which affect parasites via concomitant predation or predation on free-living stages, thus indirectly releasing parasites from predation pressure. **(c)** Similarly, the loss of native predators (white square) can lead to an increase in infections by relieving parasites from concomitant predation or predation on free-living stages.

parasites but are unsuitable hosts, leading to reduced infections in native mussel hosts [40]. Second, invaders could consume native predators, indirectly releasing parasites from predation pressure and increasing transmission to native hosts. Although we are not aware of specific examples, this seems a likely scenario given that invaders often reduce the abundance of native species [79–80]. Third, loss of a native predator on parasites could increase infections in other species. Ostfeld and Holt [81] argued that the loss of mesopredators, many of which feed on rodents, increases the risk of rodent-borne zoonotic diseases in humans [82]. Similarly, the loss of top predators could increase disease in prey, particularly if infected prey are easier to catch [83]. These latter scenarios serve to link the phenomenon of predation on parasites with the epidemiological concept of the dilution effect, in which reductions in biodiversity can amplify disease risk [7,35,70,84] (Box 3).

Conclusions

While distasteful from a human perspective, predation on parasites is widespread in nature and assumes a variety of forms, including consumption with the host (concomitant predation), on the host (grooming), outside the host (predation on free-living stages), or within the host (intraguild predation). The recent inclusion of parasites into select food webs has led to the startling realization that predation on parasites is one of the most common linkage pathways, and its incorporation affects patterns of biodiversity, linkage density and connectance, with implications for changing interaction networks and network stability. The significance of such observations is only beginning to be appreciated; more studies need to address whether parasites make significant contributions to predators' nutrition. In some cases this is possible but often parasites will make up only a small portion of predator diets. Some parasites, however, increase predator foraging success or provide unique nutritional contributions to predator diets, suggesting that biomass alone is insufficient to evaluate the significance of parasites to energy transfer. In a food web context, predation on parasites is likely to consist of many weak links (i.e. weak energy flows from parasites to predators compared to those from 'real' prey to predators) with the majority occurring at low trophic levels. In theory, a high proportion of weak interactions, or a high variance in interaction strength, can reinforce a network's stability [85–87]. Thus an important next step in food web research will be to measure the impact that the previously overlooked links associated with predation on parasites have on dynamic food web properties like stability and resilience. This will require first and foremost that more studies integrate parasites (and especially predation on parasites) into highly resolved food webs.

Research on parasite consumption provides an immediate and direct opportunity for linking the fields of foraging ecology and food web dynamics with epidemiology and disease ecology, including recent interest surrounding the link between community structure and parasite transmission [7–8,82,84]. The examples provided here illustrate that predation on parasites can strongly influence parasite transmission and patterns of disease pathology in both humans and wildlife. In some cases, such predation can help to effectively control diseases and pests with medical, veterinary or agricultural importance, highlighting the applied importance of predation on parasites. From a theoretical perspective, predation on parasites helps to unite two emerging concepts in disease ecology: the roles of biodiversity generally (e.g. the 'dilution effect') and of

predators specifically in controlling disease [81–83]. Understanding the relative importance of predation in controlling infections as a function of pathogen type or life stage, predator abundance, and host abundance should be priorities for future investigations. Broad efforts are needed to join experimental and theoretical approaches into a community ecology framework of disease that examines disease emergence and transmission within an ecological matrix of species interactions, including modules for host–parasite, vector–parasite, and predator–prey interactions [8,82,95]. Ambient diversity can play a crucial role for diseases caused by macropasites with complex life cycles [35] and predation on parasites might be an important mechanism by which community diversity reduces disease risk [7,35]. Changes in natural predator communities (e.g. biodiversity losses and biological invasions) indirectly affect infectious diseases, underscoring how host–parasite systems are embedded within complex and dynamic ecological communities while highlighting the urgency of additional research to address the mechanisms and implications of these changes (Box 3).

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.tree.2010.01.005.

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